Illicit street drugs are readily available throughout the world. For many reasons, their use is associated with a multitude of complications which require surgical treatment. Although, there are hundreds of different illicit drugs, they can be broadly classified into ‘uppers’ and ‘downers.’ Within a broad array of ‘uppers,’ cocaine is the stereotypic example with the other ‘uppers’ having similar physiologic effects. Heroin is the classic ‘downer’ with other ‘downers’ having physiologic effects which mirror heroin. There are several combinations of these broad groups including many designer drugs. The most common combinations, however, would be the mixture of an ‘upper’ or a ‘downer’ with alcohol.

The use of illicit street drugs presents a well known cadre of social, legal, and economic challenges. The prohibition of these agents drives the commercial aspects of trade into the underground part of our society, thus, facilitating an illicit drug industry which has grown to gigantic proportions.

Illicit street drugs, Cocaína, Heroína, Drogas callejeras, Complicaciones quirúrgicas de drogas recreativas.
States which has current expenditures exceeding many billions of dollars, and has created a situation where illicit street drugs are available everywhere.\(^2,6\) This underground industry has led to the ‘war on drugs’ which, in the United States includes a ‘drug czar’ in charge of an enormous policing industry designed to eradicate these illicit activities.\(^3\) Despite the many forces directed against this industry, little emphasis is placed upon the medical and surgical challenges created by illicit drug use. This article focuses on the surgical complications of both ‘uppers’ and ‘downers’ using cocaine as the stereotypic ‘downer’ and cocaine as the stereotypic ‘upper.’

**HEROIN USE**

Heroin is a commercial product of opium which has probably been available from the time of prehistoric man. Opium can be obtained from poppies by a process of harvesting and preparation requiring no distillation.\(^1\) Although, there are many genera of the poppy, opium is obtained from *Papaver somniferum*; the species name implies that this is the sleep-inducing poppy in the Linnaeus botanical classification (Fig. 1). Only this species contains the alkaloid morphine. The raw opium is boiled and sieved to remove impurities, re-boiled, and then allowed to sun dry so that the ‘prepared’ opium is much purer than the raw opium.

**Drug Abscesses**

The infectious complications related to heroin use reflect the multiple dilutions that are used to dilute the ‘pure’ opium for usage. These dilutions made with water and other particular substances often are not accomplished in a sterile manner so that the final injectate contains bacteria and foreign bodies. When the injection is made into the subcutaneous tissue, the bacteria rapidly lead to a subcutaneous abscess and cellulitis. During a 12-month period at the Detroit Receiving Hospital (DRH), the surgical services drained a total of 421 abscesses over a 12-month span; this included all types of abscesses including postoperative complications. Drug injection related abscesses accounted for 2/3 of these abscesses with the common location being in the extremities especially the groin; 10% of these patients had abscesses at multiple sites due to multiple prior injections.\(^4\) The most common organism was *Staphylococcus aureus*, which was often resistant to methicillin. Most abscesses had multiple organisms.\(^4\)

**Subcutaneous Spreading Infection**

When the subcutaneous infection goes untreated in an impaired patient, it may rapidly spread to involve the whole extremity requiring extensive excisional debridement (Fig. 2). This type of spreading infection involves the skin and subcutaneous fat but seldom the underlying
muscles, nerves, and vessels; the extensive debridement should be limited to skin and subcutaneous tissues (Fig. 3)\textsuperscript{7} Eventually, these wounds will heal; when extensive loss of skin was necessitated because of the extent of infection, split-thickness skin graft can be applied and most function preserved.

When the extensive spreading subcutaneous infection extends beyond the shoulder or beyond the hip an emergency forequarter or hindquarter amputation may be necessary in order to preserve life.\textsuperscript{4,7} Simple incision and drainage of the extremity will not prevent the continued rapidly spreading infection over the torso by which time the overwhelming infectious insult becomes lethal. A chest wall compartment syndrome with impaired ventilation may contribute to the ultimate demise.\textsuperscript{7}

**MAINLINING**

When the addict wishes to have a quicker euphoria or ‘high,’ a direct intravenous injection is used. Early in the course of the patient’s addiction, this can be readily achieved by way of the superficial forearm veins and the superficial arm veins. Eventually, these veins become sclerotic due to the irritating injection of the impure ‘mixed jive’ so that they will no longer accommodate a direct injection and the patient must use larger veins, particularly, the common femoral vein; this process is known as ‘mainlining.’ These larger veins become thrombosed with the result that the patients purposely inject the drugs into the subcutaneous tissues (skin popping) which facilitates spreading subcutaneous abscesses, extensive lymphangitis, and cellulitis. This interferes with lymphatic return.

**Venous Stasis**

With continued use of more proximal and larger veins, these veins also become thrombosed. The combination of thrombosed large veins plus lymphangitis associated with repeated cellulitis associated with subcutaneous injections causes impaired venous return and all of its sequelae. Some of the worst postphlebitic legs with large venous stasis ulcers are seen in long-term users. Following modest debridement of necrotic tissue, these patients are candidates for long-term treatment with an Unna boot, changed weekly, until complete healing occurs; they then will require life-long compression with support hose supplied by either a Jobst pressure hose at 40 torr or long-term daily use of ace wraps. Extensive soft tissue debridement with ligation of the perforating leg veins will not succeed in these patients.

**THE ‘PINKY’**

After long-term mainlining, the scar tissue around the involved area, more commonly at the elbow or groin, impairs a direct venous hit so that the long-term user often gets a ‘pinky’ which is recognized by the bright red blood return typically seen with an intra-arterial aspiration. When the user is already under the influence of a previous recent injection, the ‘pinky’ may not be recognized and a direct intra-arterial injection is made. The various compounds including talc which have been used to cut the ‘mixed jive’ heroin then embolize into the peripheral arterial tree causing a sudden and intense burning. This is due to small vessel embolization with the intense burning typically occurring in the hands or the feet. When the injection is made at the elbow, the intense burning will be in the distribution of either the radial artery or the ulnar artery. This burning is followed by ischemia to the involved part. Sometimes the ischemia is extensive so that the patient will lose digits. In other situations, the ischemia will lead to skin necrosis typical of what is seen in a patient with stage 3 frostbite. The intense pain caused by this ischemia may last for weeks and months. When long-term symptoms persist, a significant relief or decrease in the amount of pain can be achieved by sympathectomy using the retroperitoneal approach for the lumbar chain or the transaxillary approach for dorsal chain.

**Infected Pseudoaneurysm**

More commonly, the errant intra-arterial injection associated with the ‘pinky’ leads to a periarterial hematoma which becomes infected because of the contaminated injectate. This leads to an intra-arterial communication

![Fig. 4: The cellulitis and abscess cavity surrounding the aneurysmal abscess may 'camouflage' the underlying arterial communication](image-url)
through the injection site to the surrounding periarterial abscess resulting in what is commonly called a ‘mycotic aneurysm.’ The aneurysmal abscess was first described by Koch in 1851 in a patient who had endocarditis due to prior rheumatic fever and endocarditis resulting in bacterial embolization to the superior mesenteric artery.8 The bacteria embolize into the vasa vasorum and the abscess forms in the medial portion of the artery which, in the patient described by Koch, ruptured causing her immediate death. Several years later, Osler in 18859 described ‘mycotic aneurysm’ in his Gulstonian Lecture on Malignant Endocarditis. Osler thought that the white wall of the infected aneurysm reminded him of mycoses, thus, the origin of the term Mycotic Aneurysm. The application of this term to the drug addict has become common although, technically, the drug addict has an ‘infected pseudoaneurysm.’

This entity is common among long-term users. During a 22-month interval from July 1980 through March 1982, the surgical services at the Detroit Receiving Hospital (DRH) excised 52 ‘mycotic aneurysms’ in 50 patients. Almost all gave a history of a ‘pinky’ associated with burning of the hand or foot at the time of injection (Fig. 4). The swelling around the aneurysmal abscess represents cellulitis so that, in many patients, the transmitted pulsation is not appreciated by the examiner as an infected pseudoaneurysm. Most of these abscesses occurred in the lower extremity which is the more common site for injection in mainliners.10

When the unsuspecting surgeon takes a patient with obvious cellulitis and suspected abscess to the operating room for incision and drainage, there will be a bright red gush of blood announcing to the surgeon that this is a mycotic aneurysm. The primary surgeon must use direct digital pressure with his/her finger while a second surgeon obtains proximal and distal control around the surgeon’s finger prior to directly exposing the ruptured mycotic aneurysm. Peripheral extremity aneurysmal abscesses are best treated by excision with the proximal and distal ligation done close to the aneurysm.10 Occasionally, the injection is made proximal to theinguinal ligament resulting in an aneurysmal abscess or mycotic aneurysm of the external iliac artery (Figs 4 and 5). When this occurs, proximal exposure is best obtained by angling the incision laterally parallel and superior to the inguinal ligament to expose the superficial iliac artery in its retroperitoneal location. Likewise, the injectate may be significantly below the inguinal ligament into the superficial femoral artery distal to the bifurcation of the profunda femoral artery. The common femoral artery should be protected when obtaining proximal and distal control.

**Treatment of Mycotic Aneurysms**

Since, the infected pseudoaneurysm is in the midst of cellulitis and abscess, surgical reconstruction of the involved artery in its normal anatomic plane is doomed to failure. The vast majority of these patients, therefore, are treated with excision being sure that the proximal and distal ligation site are located immediately proximal and immediately distal to the aneurysmal abscess; this helps preserve collateral flow.

**Post-Ligation Ischemia**

The aneurysmal abscess that comprises the common femoral, the superficial femoral, and the profunda femoral arteries and is most likely to lead to post-excision ischemia.10 Successful hemostasis requires ligation of all three vessels (Fig. 6). When the patient has diffuse circumferential cellulitis from the hip to the knee, there is no safe rearterialization procedure that can be performed and the patient is doomed to an above knee amputation. When the patient has the area of cellulitis limited to the femoral triangle and complains of constant pain after ligation and there is evidence of ischemia, immediate bypass can be performed utilizing the obturator foramen to bypass the Dacron graft from the external iliac artery to the popliteal artery; none of these patients has a patent saphenous vein.11 A size 8 Dacron graft is preferred. In the author’s experience, all patients who continued with drug usage eventually had thrombosis of the graft within 3 years. Long-term patency can be anticipated if the patient quits drugs. Mycotic aneurysms of the upper extremity rarely lead to ischemia following proximal and distal ligation with excision (Fig. 7). Recidivism is common and many patients have required more than one infected aneurysmectomy. Occasionally, a patient will present with an infected pseudoaneurysm involving both the artery and the vein. This is more likely to occur in the groin and results in an arterial venous fistula (Fig. 8). This can be identified on the arteriography by the presence of a double shadow representing the rapid movement of dye from the artery into the vein (Fig. 8). When this occurs, resection of both the arterial aneurysm with proximal and distal ligation in addition to venous resection with appropriate hemostasis is essential (Fig. 8).

**Cervical Mycotic Aneurysms**

Occasionally, the long-term user will rely completely upon the jugular venous system to obtain vascular access. Indeed, more than one patient has avoided the groin for fear of having complications requiring an amputation without considering the potential for a stroke. Over the years, repeated cervical injections causes fibrosis and
Fig. 5: When the injection is inadvertently made proximal to the inguinal ligament the aneurysmal abscess will occur in the external iliac artery; proximal control is best made by making a ‘hockey stick’ incision parallel and superior to the inguinal ligament in the retroperitoneal plane.

Fig. 6: The pseudocapsule in this patient with an aneurysmal abscess involving the common femoral artery, superficial femoral artery, and profunda artery was extensive; postoperative ischemia necessitated an obturator bypass graft.

Fig. 7: Excision of the aneurysmal abscess with ligation of the proximal and distal artery precludes disruption of collateral flow minimizing the likelihood of postexcision ischemia.

Fig. 8: The arteriogram on the left shows the double shadow with venous filling from the arterial venous fistula. The operative findings on the right show the artery ligated proximal and distal to the fistula as the vein is being freed; note the clot between artery and vein.

Fig. 9: Infected pseudoaneurysms of the carotid artery always have extensive surrounding cellulitis precluding interposition graft replacement.

Fig. 10: The immediate post injection arteriogram on the left shows no flow through the excised left carotid artery but, on the right, one can see excellent backflow later during the arterial injection.
thrombosis of the jugular veins resulting in an intraarterial injection into the carotid artery with the associated perivascular abscess and extensive surrounding cellulitis (Fig. 9). Vascular reconstruction following excision of an infected carotid artery aneurysm is impossible due to the surrounding cellulitis. Fortunately, most of these patients thrive after carotid aneurysmectomy because of excellent retrograde filling from the contralateral side (Fig. 10).

**Thoracic Mycotic Aneurysms**

Long-term users with thromboses of many named veins, often resort to streetwise professional phlebotomists who are skilled at ‘hitting the pocket’ or accessing the subclavian veins. Their incidence of pneumothorax is far less than that experienced by junior surgical residents. Repeated pocket hits is associated with ‘pinkies’ from the innominate, left common carotid, or subclavian artery. The sequelae of aneurysmal abscess is common. A median sternotomy gives the best access for all three vessels including the proximal left subclavian artery. Excision with proximal and distal ligation is the only therapeutic option (Fig. 11).

**Refractory Endocarditis**

A common complication of injecting contaminated mixed jive into the venous system is infection of the heart valves. Treatment requires long-term antibiotic support which may be successful in those who give up their drug usage. Frequently, however, recidivism occurs so that the patient becomes re-infected with resistant organisms which cannot be eradicated with antimicrobials and the patient is a candidate for valvulectomy.12 Valvular replacement in these patients for refractory endocarditis may be successful if the patient no longer uses so that the replacement valves do not become infected. When the replacement valves become infected with resistant bacteria, the patient is doomed for death.

One of the causes for reinfection of a replaced valve or infection of another valve is recontamination from an intrasplenic abscess. The splenic abscess is a result of the embolization from the heart valves. Faced with the problem of recurrent valvular infection in patients with recidivism, Arbulu was the first to do valvulectomy of the pulmonary valve without replacement. Incidentally, the pulmonary valve is a commonly involved valve. Using this technique, he observed a 62% 25 year survival. Prevention of this complication can occur by performing splenectomy at the time of, or shortly following, valvular replacement.13

**COCAINEx USE**

Cocaine has a rich history that dates back to antiquity.2 The cocoa leaves were partially chewed as a local anesthetic in patients undergoing trephination for intracranial hematomas. The Inca empire was founded in the early 11th century in Cuzco, Peru; the Inca ‘Queen’ was known as Mama Cuca, a reference to the cocoa leaf.14 The consumption of the cocoa leaf was limited as part of the ‘divine’ right of the Inca to high-ranking officials, priests, and heroic soldiers as a reward or token of appreciation. Food supplies to the mountainous city of Cuzco were provided by a ‘human’ express of runners carrying fresh food and messages to the Incas. There were stores of cocoa leaf along the route from the ocean to the mountain tops in order to aide each runner with the accumulated average distances being about 150 miles per day. A ‘cocada’ was defined as a measure of distance that a runner could go without tiring under the influence of cocaine. Following the conquering of Peru by Pizarro, the Indian slaves working in the silver mines were allowed to use cocaine in order to fight fatigue and hunger, thereby working longer and producing more.2,15 The Spanish warlords imposed a 5% tax on the cocoa crop in order that the Church personnel could receive ‘their share’.2,15

Chemically, cocaine is benzoylmethylecgonine which consists of the ester benzoic acid and the nitrogen-containing base ecgonine; ecgonine, in turn, is a tropine derivative and is the parent compound of atropine and scopolamine. Once ingested, cocaine reaches his highest concentrations in the brain, spleen, kidney, and lungs.2 Clinically significant serum levels are reached within seconds after ingestion and maintain a half-life of approximately 30 to 90 minutes. Nasal ingestion by crack cocaine causes intense vasoconstriction which decreases the nasal uptake of cocaine, thus, prolonging the ephoritic effect for 1 or 2 hours. Over 80% of the cocaine is metabolized to 1) ecgonine methyl esters by rapid enzymatic hydrolysis in the plasma and by liver esterases; 2) benzoylcgonine by spontaneous nonenzymatic hydrolysis; and 3) norcocaine by liver N-demethylation. A small portion remains unmetabolized and is excreted into the urine 3 to 6 hours after ingestion.2,15 The primary physiologic effect of cocaine is to block the reuptake of catecholamines by the presynaptic sympathetic nerve fibers resulting in catecholamine accumulation in the synaptic cleft and increased cell receptor stimulation.

**Cardiac Effects**

Cocaine causes a decrease in the rate of depolarization and amplitude of the action potential within the myocyte.
This may slow the conduction rate of the action potential leading to cardiac arrhythmias and sudden death. Cardiac arrhythmia and death can occur within 3 minutes of cocaine exposure; emergency treatment requires rapid securing of the airway with close cardiac monitoring and cardiopulmonary resuscitation if needed. Simultaneous beta blockade may help control the excessive acute sympathomimetic effects; barbiturates should be added if convulsions occur.

**ACUTE STROKES**

Within the mesolimbic and mesocortical areas of the brain, cocaine blocks the dopamine uptake pump and impairs dopamine reuptake into the presynaptic neurons; this results in dopamine accumulation in the synaptic cleft and sustains stimulation of the dopaminergic receptors. These actions cause an intense euphoria but this is associated with tachyphylaxis so that larger and larger doses of cocaine are needed to produce the same euphoric effect. The intense vasoconstriction associated with these pharmacologic effects may lead to acute stroke, typically, of the ischemic variety. When the vascular compromise occurs in the hypothalamus or the pituitary gland the patient may develop diabetes insipidus. Seizures are a common result of the small vascular infarcts and the intense vasoconstriction within the brain.

**Duodenal Ulcer Disease**

The central nervous system stimulation by cocaine which binds to the sigma and muscarinic (M #1 and possibly M #2) receptors may lead to an anticholinergic effect by way of muscarinic receptor blockade. This, in turn, results in decreased gastric motility and the potential for subsequent ulceration due to prolonged acid exposure. The clinical sequence of ulcer formation following cocaine exposure, however, suggests that the acute ulceration is brought about by intense vasoconstriction with resultant focal ischemia in the proximal duodenum. The patient who presents with a perforated duodenal ulcer gives a history that the intense pain began within an hour or two after taking the cocaine. The pain then persists until the generalized peritonitis develops which represents the time of the perforation and leakage of air and bile into the peritoneal cavity. There are no histologic studies confirming this sequence of events related to perforated duodenal ulcers after cocaine ingestion.

**Small Bowel Perforation**

The intense vasoconstrictive effects of cocaine on the gastrointestinal tract are more commonly seen with the small intestine. The patient typically presents to the emergency department with severe abdominal pain and tenderness which can be traced back to the time shortly after the cocaine was taken. Often there is a delay of some hours before the actual presentation by which time the patient obviously has localized peritonitis. Pneumoperitoneum in this setting is rare. Often, when exploratory laparotomy is performed, the patient will be found to have localized inflammation in one or more parts of the small intestine with one of the adjacent loops of small intestine serving as a patch by covering the source of the inflammation. When the loops of bowel are carefully separated, the source of inflammation will be a small, almost pinpoint, perforation of the antimesenteric surface of the small bowel which had been covered the loop of intestine that is not ischemic. Resection and histologic examination of this segment of small bowel will demonstrate full-thickness necrosis with small intramural thrombosis surrounded by a liquefactive necrosis leading up to the point of small perforation (Fig. 12). Postoperatively, the patients are advised to avoid further use of cocaine.

When there is a distinct history of cocaine ingestion occurring prior to the onset of severe, constant pain followed by peritoneal guarding, a clinical diagnosis of cocaine induced ischemia with microperforation can be made and a decision to treat the patient nonoperatively can be implemented. This approach has evolved after a number of laparotomies done for localized minute small intestinal perforations for which an adjacent normal small bowel loop has served as a patch. When a nonoperative regimen is followed, one has to reassess frequently each 12 hours with the expectation that bowel function will return after 3 or 4 days, at which time diet can be initiated.

Although the nonoperative approach may be successful initially, the patient may develop the symptoms of bowel obstruction down the road due to the adhesions created by the localized perforation. This may present as a paradox in a patient who has the signs and symptoms of small bowel obstruction but does not have a hernia and has never undergone a previous laparotomy (Fig. 13). Often, it is not possible to get a history of cocaine use in the preoperative period. When operation is done in these patients, the typical finding will be adhesions of small bowel stuck to other loops of small bowel without any adhesions to the remaining viscera within the peritoneal cavity. Resection of the obstructed segment will usually identify that there is a prior area of intestinal necrosis caused by the initial exposure to cocaine (Fig. 14). The microscopic examination will demonstrate the focal area of necrosis with a surrounding inflammatory response (Fig. 15). Once the patient is educated about
the importance of this finding, the history of cocaine can usually be obtained. Most patients who use cocaine for its euphoric effect will rapidly quit using it and find some other substance.

**COLON PERFORATION**

Cocaine ingestion leading to localized colon perforation is less common but can occur in any part of the colon. When it occurs in the sigmoid colon, the presentation is similar to somebody who has acute diverticulitis with localized left lower quadrant tenderness and guarding associated with leukocytosis and fever. Unlike most sigmoid diverticulitis, the patient does not improve as rapidly as one would expect and may often require laparotomy. During exploration, one typically finds a loop of small bowel which has attached itself to the sigmoid colon. When the small bowel is gently mobilized away from
the colon, a small area of perforation will be identified. Unlike sigmoid diverticulitis, the inflammation will not extend down between the two layers of the mesentery toward the origins of the mesentery since the perforation is located on the antimesenteric border. This site of the perforation leads one to suspect a vascular phenomenon. This is confirmed on histologic examination. The history of cocaine use is easier to obtain once the intraoperative findings are identified and explained to the patient. Since the mesentery is not thickened and inflamed, it is safer to do a resection with primary anastomosis in this setting than it is with perforated sigmoid diverticulitis. This is also true for the other segments of the colon where there is a localized perforation from cocaine ingestion.

**SPLENIC INJURY**

Cocaine induced infarction of the spleen with subsequent hemorrhage has also been reported. Such patients may present with vague but persistent left upper quadrant pain which will become more severe if there is a subsequent rupture of the intrasplenic hematoma. The imaging studies may be similar to that seen with blunt rupture to the spleen. When operation is performed, the finding of a focal rupture without large parenchymal tear leads to the suspicion that this may be a cocaine induced bleed.

**COCAINE ‘BODY PACKERS’**

The surgeon may be consulted to see a patient who is functioning as a body packer who swallows packets of cocaine along with a constipating agent in order to transport the cocaine across a border. Once they get through Customs, the use of laxatives or enemas will facilitate the cocaine packets to be excreted. Each of these packets contains 3 to 7 gms of cocaine which is a fatal dose if the packet ruptures and the cocaine is absorbed. When seeing such a patient nonoperative therapy includes hydration and cathartics. When a patient shows evidence of increased sympathetic tone, rupture should be suspected and an emergency operation performed.

**RENAL COMPLICATIONS**

Cocaine produces intense renal vasoconstriction which can lead to segmental renal thrombosis. The thrombosis is caused by an imbalance between thromboxane and prostacyclin synthesis in the damaged renal artery endothelial cells. Cocaine induces macrophage interleukin-6 production and subsequent musangial cell proliferation which causes focal glomerular sclerosis. This may be the cause of renal failure which is made worse by associated muscle ischemia and rhabdomyolysis. Cocaine induced rhabdomyolysis of the leg muscles in association with intramedullary rodding of the ipsilateral femur may be mistaken for direct blunt injury of the leg rather than rhabdomyolysis due to the cocaine. These patients should be suspected of having a compartment syndrome which is caused not by the external blunt force but rather by the cocaine induced muscle death.

The cocaine induced vasoconstriction of the intramuscular arteries often results in muscular ischemia with myofibrillar degeneration and acute rhabdomyolysis. The addition of rhabdomyolysis to the ischemia renal parenchymal injury increases the likelihood that the patient will have rhabdomyolysis induced renal failure requiring subsequent dialysis. When patients present with muscle pain and tenderness, rhabdomyolysis should be anticipated and followed for hyperkalemia, hyperphosphatemia, and hyperuricemia. Typically, the high levels of creatinine kinase will exceed 100,000 units/L. These renal complications can be ameliorated by generous hydration but may require hemodialysis.

**LARGE VESSEL INJURY**

Cocaine also may be associated with thrombosis of large vessels including the renal arteries and the abdominal aorta. When named intraabdominal arteries become occluded, the mechanism of injury is thought to be related to stasis in the vasa vasorum which, in turn, leads to cessation of blood flow to the intima. This is associated with intimal damage and platelet aggregation. This, in turn, stimulates platelet adherence as the coagulation cascade begins. The specific therapy has to be determined by the effect of impaired flow to the involved organ. Patients who present some degree of mesenteric artery
occlusion without bowel necrosis, renal artery occlusion without renal shutdown, or aortic thrombosis without distal ischemia can be treated with anticoagulation and careful clinical monitoring (Fig. 16). When end-organ compromise, or tissue necrosis, is threatened emergency thrombectomy is indicated. The intimal insult brought about vasospasm of the vasa vasorum may result in a secondary aortic dissection. The dissection initiates at the site of the intimal injury and propagates in the subintimal location as a hematoma or as a dissection. The cocaine user, after blunt trauma, may be suspected of having this syndrome when there is a sudden ‘tearing’ chest or abdominal pain which cannot be explained on the basis of deceleration injury. The principles of therapy follow those that are seen in patients who have dissecting aortic injuries following trauma.

CONCLUSION

Cocaine can present many unusual problems to the acute care surgeon. Multiple organ toxicities may occur and aggravate the detrimental effects of hemorrhagic shock and sepsis resulting in a much higher mortality rate. Identification of cocaine use is supported by an aggressive approach by the physician team in terms of getting a complete history. This should be followed by monitoring for substance abuse in patients presenting with emergency problems. Sudden death in a patient who seems to be recovering should be suspected of being related to in hospital cocaine ingestion. Friends of the patient may bring in the cocaine which may be of purer strength and, thus, lead to the catastrophic cardiac events. Once cocaine utilization is identified, one should monitor for the effects of cocaine on each organ system. This will allow the therapeutic use of beta blockade, sedatives, and anticonvulsants as indicated by the patient’s general state. This approach will help decrease the likelihood of the cocaine being an added factor to a patient having a poor outcome.

REFERENCES