Clinical Toxicology

ISSN: 0009-9309 (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/ictx18

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To cite this article: David C. Perry & Bruce E. Ratcliffe (1975) Heroin and Cocaine Adulteration, Clinical Toxicology, 8:2, 239-243, DOI: 10.3109/15563657508988068

To link to this article: https://doi.org/10.3109/15563657508988068

CLINICAL TOXICOLOGY 8(2), pp. 239-243 (1975)

EDITORIAL

Heroin and Cocaine Adulteration*

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Heroin and cocaine are two of the most extensively adulterated drugs on the present illicit market. Use of both drugs is widespread, and both command high prices on the street. Consequently, a dealer's incentive to adulterate—"step on" or "cut"—a batch of pure drug with other cheaper substances is strong. In dealing with the phenomenon of heroin and cocaine use/abuse, it is important to understand that the white powder an addict purchases for $10 a bag may contain a mixture of substances, with different effects and possible dangers, besides (or even instead of) the heroin it was sold as. This problem becomes especially acute when the drug is injected, for adulterants are most likely to prove dangerous when used intravenously. Since heroin and cocaine are most commonly loose white powders in pure form, there are hundreds of possible adulterants a dealer could use. In this article we shall examine the ones most often used, especially the potentially dangerous ones.

NONDRUG CUTS

Many substances not chemically classified as drugs are used as adulterants or diluents. Sugars, most commonly lactose, inositol, and mannitol, are widely used. Lactose, often called "milk sugar," is a disaccharide similar to sucrose often used in infant nutritional formulas. Inositol is an isomer of glucose and is considered one of the vitamin B complex. Mannitol is a monosaccharide (like glucose) sometimes used clinically as an osmotic diuretic. These sugars are regularly used in pharmaceutical preparations, and can be considered relatively safe; however, most medical knowledge of these substances is based on oral use.

*Reprinted from PharmChem Newsletter, 3(2) (1974).

Other nondrug cuts include substances such as talc, flour, and cornstarch. It is difficult to determine the extent of their use, since analysis for them is rarely done. While pharmacologically inert, these substances are far from harmless. They are largely insoluble in blood. Upon injection, they can become emboli and lodge in arterioles and capillary beds, obstructing blood flow and causing serious pulmonary, renal, and neurological damage, as well as tissue anoxia resulting in necrosis and fibrosis. Recently, there have been numerous cases reported of addicts with talc and cornstarch emboli in the lungs and eyes, serious problems which can lead to respiratory failure and blindness.

ADULTERATION WITH OTHER DRUGS. SOME TRENDS

Of alleged cocaine samples submitted for analysis at PharmChem in 1973, 73% contained cocaine as the only drug. Another 21% consisted of cocaine adulterated with synthetic local anesthetics such as procaine, while the remaining 6% contained other drugs such as amphetamine, caffeine, phencyclidine, or else no drug at all. Other street drug analysis programs have shown roughly the same trend, with perhaps a slightly higher percentage of samples adulterated with local anesthetics. The illicit price of cocaine shows little correlation with purity.

A broad picture of heroin adulteration can be seen by examining the urinalysis results performed by PharmChem for methadone clinics throughout the country (over 4000 samples weekly). Both quinine and procaine are routinely detected in samples which are positive for heroin. Procaine cuts appear to be widespread in California and the Southwest, especially in the brown heroin allegedly from Mexico. Heroin from Northern California, the Northwest, and the Midwest is less commonly cut with procaine, while in the East procaine use is uncommon; however, its use appears to be increasing. The drug of choice for heroin adulteration in the East, particularly in New York and Detroit, is quinine. Quinine is seen less commonly in the Midwest and rarely in the West and Southwest. Neither drug is a common adulterant in Canadian heroin.

LOCAL ANESTHETICS

All local anesthetics, with perhaps the exception of cocaine, have similar structures and physiological effects. Cocaine is unique in

that it also has stimulant and euphoric properties. Pharmacologically, local anesthetics prevent both the generation and conduction of nerve impulses, affecting both the sensory and motor nerves. These drugs are rapid acting and of short duration. The initial effects wear off in one to two hours after administration. The interruption of nerve transmission by local anesthetics also makes them toxic agents. High blood levels of local anesthetics result in convulsions and respiratory collapse, conditions which can rapidly lead to death if not immediately checked. The surest method of achieving high blood levels is by intravenous injection. Thus, these drugs are commonly used topically and intramuscularly. When intravenous use is necessitated, strict controls and precautions are obeyed.

Of the dozens of local anesthetics used medicinally, only a few are seen on the illicit market. Procaine (Novocaine) is by far the most common, followed by lidocaine (Xylocaine), benzocaine, and tetracaine. The degree of toxicity varies, as shown by studies performed on rats and rabbits. Procaine is the least toxic, lidocaine and cocaine are about 2-3 times as toxic as procaine, and tetracaine is about 7 times as toxic as procaine. Benzocaine is relatively insoluble and is considered quite safe when used topically. However, this lack of solubility renders benzocaine extremely hazardous if injected. Blood clots and serious complications are distinct possibilities. Approximately 3% of the cocaine samples analyzed by PharmChem contained benzocaine.

The primary dangers of these drugs arise from intravenous use, a practice still common among some cocaine users and routine with heroin users. Even the relatively safe procaine has been known to be highly toxic and even lethal at doses as low as 10 mg when injected into a hypersensitive individual.

QUININE— THE UNKINDEST CUT OF ALL

Quinine is a bitter tasting white powder extracted from the bark of the cinchona tree. Quinine became famous as the original antimalarial, although today it has been largely replaced by synthetics. Most commonly available as its sulfate salt, quinine has a wide variety of seemingly unrelated pharmacological properties: as an anti-pyretic and analgesic to control fever and mild pain, as a skeletal muscle relaxant to relieve nocturnal muscle cramps and related symptoms, as a smooth muscle relaxant to induce labor and abortion (illicitly), as a sclerosing agent in the treatment of varicose veins, and as a local anesthetic.

The toxic effects of quinine are equally far-reaching. Even small

doses can induce symptoms of cinchonism: tinnitus (ringing in the ears), headaches, visual disturbances, vertigo, nausea, vomiting, and diarrhea. Quinine also possesses marked irritant properties. Oral use can cause gastric disturbances; topical use and intramuscular injection can cause painful tissue irritation and lesions; and intravenous use can lead to thrombosis of the injected vein and a definite and alarming hypotension (drop in blood pressure), especially when injected rapidly. More severe poisoning can lead to renal damage, acute hemolytic anemia, and overstimulation of the CNS leading to coma and eventual death by respiratory arrest. The average lethal dose is 8 gm, although death has been recorded from doses as low as 0.4 gm. A number of cases of oral overdose have been recorded involving women attempting to induce abortion.

Quinine was first used as a heroin adulterant in the early 1940s, allegedly to stem an epidemic of malaria sweeping through New York's addict population. Its use continued for other reasons. Its bitter taste prevented buyers from being able to judge the quality of heroin sold, and it also added to the "rush" of a heroin injection (very likely due to quinine's hypotensive effect). Although quinine is a relatively toxic drug, it has a "good" reputation with the general public. However, recent studies of "heroin overdose" deaths have indicated that quinine may be one of the primary agents responsible for these deaths.

The incidence of sudden deaths from heroin injection has dramatically risen since the early 1940s (about the same time as the introduction of quinine in heroin), and these tragic deaths have played a major role in the public outcry over the "heroin epidemic." When a coroner is faced with the task of determining the cause of death of a known narcotics user, evidence of recent use of narcotics is generally all that is needed to issue a certificate stating "death from narcotic overdose." The following are quotes from two recent papers concerned with New York heroin deaths, the authors of which include the Chief and Deputy Chief Medical Examiners of New York City, Dr. Milton Helpern and Dr. Michael M. Baden:

> It is concluded that the main cause of death in New York City addicts is an idiosyncratic reaction to an intravenous injection of unspecified material(s) and probably not a true pharmacological overdose of narcotics.
>
> There has been an increase in the "acute reaction" type of death in recent years. In many cases it seems to be not a simple overdosage but an overwhelming

shock-like reaction due to hypersensitivity to the injected material; the presence of quinine may be a contributing factor.*

The observable symptoms in these deaths bear a strong similarity to deaths from acute reaction to quinine (true narcotic overdoses are generally much slower), and furthermore, the incidence of these kinds of deaths is greatest in those areas where quinine adulteration is most common. This is not to say that true heroin overdoses do not occur, or that quinine is responsible for all the narcotics-related sudden deaths. Another possibility is the trend toward alcohol and barbiturate use by narcotic users. Such "mixing" was formerly shunned by most addicts, but periodic shortages of street heroin have become more common recently and these droughts may encourage use of these drugs as temporary substitutes. Both alcohol and barbiturates are synergistic with narcotics, greatly increasing the possibility of accidental overdose when used simultaneously.

Whatever the ultimate causes, this information has failed to reach both the narcotic user and the general public, and the deaths from "heroin overdose" continue.

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*Editorial note:

(a) Immediate (or "wet") heroin O.D.'s are a result of diacetylmorphine bolus I.V. ergo pulmonary smooth muscle spasm and exitus.

(b) Slow (or "dry") heroin O.D.'s are a result of diacetylmorphine metabolites: morphine, methymorphine, and (?) 6-monoacetylmorphine, ergo CNS respiratory depression and exitus.

R.T.R.

P.S. A little naloxone for (a) 0.8 ng. A lot of naloxone for (b) 1.6 mg and up.