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Blunt Abdominal Trauma With Minimal Free Fluid on CT

ABSTRACT & COMMENTARY

Source: Ochsner MG, et al. Significance of minimal or no intraperitoneal fluid visible on CT scan associated with blunt liver and splenic injuries: A multicenter analysis. *J Trauma* 2000;49:505-510.

IN MANY CENTERS, BEDSIDE ULTRASOUND (U/S) HAS BECOME A VALUable tool in the emergency assessment of the injured patient. It is generally accepted that, in experienced hands, U/S will detect fluid collections of 250 mL or more.[1] The purpose of this study was to describe the incidence and clinical importance of liver and spleen injuries with minimal (< 250 mL) or no free intraperitoneal fluid visible on CT scan.

All patients with liver and spleen injuries were identified retrospectively using medical records and CT scan review. Study inclusion criteria were liver and spleen injury, identification by CT scan, and minimal or no free fluid on CT scan. Minimal fluid was defined as less than 250 mL, and was calculated using Knudson's quantification scale.[2] Nine hundred thirty-eight patients with liver and spleen injury were identified. Two hundred sixty-seven (28%) met inclusion criteria and had minimal or no free fluid on CT. One hundred sixty-one had injury to the spleen and 125 had injury to the liver. Ninety-seven percent of included patients were managed nonoperatively; while eight patients (3%) required operative intervention for bleeding. There were no deaths or major complications. Compared to the liver, the spleen was significantly more likely to bleed (P = 0.01), but the grade of splenic injury was not related to hemorrhage risk.

COMMENT BY MICHAEL A. GIBBS, MD, FACEP

A growing number of studies document the excellent sensitivity, specificity, and accuracy of U/S as a screening tool to identify intra-abdominal hemorrhage.[1,3,4] As U/S gains acceptance, algorithms for its use must be developed. Clinicians should develop a clear understanding of what to do with both positive and negative scans. Currently, there are two accepted clinical scenarios for which the results of the U/S should trigger a clear response:

- In the clinically unstable blunt trauma patient, a positive U/S should prompt immediate exploratory laparotomy without further diagnostic testing.

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- In the clinically stable blunt trauma patient with a positive U/S, CT scanning of the abdomen should be performed to further define specific injuries.

Knowing what to do with a negative U/S is a bit trickier, but no less important. It is this author's opinion that:

- In the clinically unstable blunt trauma patient, a negative U/S should be followed by a diagnostic peritoneal aspirate.
- In the clinically stable blunt trauma patient with a significant mechanism of injury, a negative U/S should be followed by an abdominal CT scan or an appropriate period of clinical observation. Serial ultrasounds are another option in this setting.

This is not an U/S study. The retrospective design does have inherent weaknesses. Nonetheless, the results do remind us that a negative U/S does not always rule out significant injury. Studies like this one should stimulate future research in trauma U/S and help with the development of clinical pathways. As we continue to adopt U/S (or any other new technology) into the practice of emergency medicine, we must understand what it can and cannot do. ❖

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Failure of CPAP in Non-Hypercapnic Respiratory Insufficiency

ABSTRACT & COMMENTARY

Source: Delclaux C, et al. Treatment of acute hypoxemic non-hypercapnic respiratory insufficiency with continuous positive airway pressure delivered by a face mask. *JAMA* 2000;284:2352-2360.

THIS MULTICENTER, PROSPECTIVE, RANDOMIZED TRIAL compared the efficacy of continuous positive airway pressure (CPAP) plus oxygen to standard oxygen therapy (O_2 alone) in 123 intensive care unit (ICU) patients with acute respiratory insufficiency. At study entry, all patients had bilateral infiltrates on chest x-ray, and 17% were classified as pure cardiac decompensation; 83% had acute lung injury due to infections, adult respiratory distress syndrome (ARDS), near drowning, and other causes.

CPAP treatment failed to reduce the endotracheal intubation rate (34% CPAP vs 39% O_2), hospital mortality (31% vs 30%) or median ICU length of stay (6.5 vs 6.0 days). A higher number of adverse events occurred with CPAP treatment (18 vs 6, P = 001). Four patients in the CPAP group experienced cardiac arrest; no cardiac arrests occurred in the oxygen alone group (P = 0.14).

COMMENT BY STEPHANIE ABBUHL, MD, FACEP

This study helps further define the population in which non-invasive positive pressure ventilation (NPPV) should be considered. To date, the evidence supporting NPPV is strongest for patients with acute exacerbations of chronic obstructive pulmonary disease (COPD) with hypercarbia. In fact, in COPD patients, NPPV has been shown not only to decrease the need for intubation, but also has been associated with improved survival. The findings of several studies, including a related article in the *Journal of the American Medical Association,* have

suggested that the increased survival seen in COPD patients may be due, in part, to the avoidance of infectious complications, especially nosocomial pneumonia.[1]

However, this study raises some serious doubts as to the effectiveness of CPAP in patients with acute hypoxemic, non-hypercapnic respiratory failure primarily due to non-hydrostatic pulmonary edema. Not only was there no improvement in any of the main outcome measures for the CPAP group, but a significantly higher number of adverse events occurred with CPAP treatment. Most concerning, although alone not statistically significant, were the four cardiac arrests in the CPAP group that occurred at the time of intubation (3 patients), or at the time of removal of the CPAP mask for nursing care (1 patient). With the safety of CPAP in this group in question, it makes "just trying" NPPV an uncertain approach in these patients. ❖

Reference

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Prochlorperazine vs. Promethazine for Acute Nausea and Vomiting

ABSTRACT & COMMENTARY

Source: Ernst AA, et al. Prochlorperazine versus promethazine for uncomplicated nausea and vomiting in the emergency department: A randomized, double-blind clinical trial. *Ann Emerg Med* 2000;36:89-94.

VOMITING ASSOCIATED WITH GASTROENTERITIS OR GASTRITIS is a major problem confronting emergency department (ED) physicians. Opinions vary as to which agent best controls vomiting and avoids undesirable side effects and unnecessary expense. Physician preference often is arbitrary rather than scientific. Ernst and colleagues compared intravenous prochlorperazine (Compazine) and promethazine (Phenergan) in a randomized, double-blind trial involving 84 adult patients from two academic EDs. All cases involved acute nausea and vomiting (with or without diarrhea), inability to tolerate oral fluids, and some degree of volume contraction as judged by ED physicians. Patients with focal abdominal pain, underlying serious illness, bowel obstruction, altered sensorium, alcohol abuse, pregnancy, or complicated etiology for vomiting were excluded.

A 100 mm visual analog scale (VAS) was utilized to assess symptom severity at baseline and at 30 and 60 minutes to rate adequacy of relief over time. Forty-two patients received either 10 mg of prochlorperazine or 25 mg of promethazine intravenously, along with appropriate fluid therapy (1.1-3 L). Demographics included an average age of 29 years (18-44); 70% were female; 60% were white. The average number of vomiting episodes was 7.3 (2-16) in 24 hours; 55% of patients had concomitant diarrhea.

At 30 and 60 minutes, VAS symptom ratings for prochlorperazine were significantly better than for promethazine (20 vs 46 mm, $P = 0.004$; and 4.5 vs 26 mm, $P < 0.001$, respectively). Complete relief at 30 minutes was achieved in 33% of prochlorperazine patients compared to 16% of promethazine recipients ($P = 0.02$). Only 16% of patients required 60 minutes to achieve full relief with prochlorperazine, compared to 36% of those receiving promethazine. Four (9.5%) patients in the prochlorperazine group required additional anti-emetic therapy and were deemed "treatment failures" compared to 13 (31%) promethazine patients ($P = 0.03$). Of those given prochlorperazine, 38% experienced drowsiness, compared to 71% of those receiving promethazine ($P = 0.002$). Six patients in each group had akathisia, reversible with diphenhydramine.

COMMENT BY MICHAEL FELZ, MD

Both of these agents are phenothiazines, active as dopamine D_2 receptor antagonists and frequently used for nausea and vomiting. Yet physician preference, not data, often dictates therapy. This trial demonstrates clinical and statistical advantages for prochlorperazine in ED patients with acute nausea, vomiting, and apparent volume contraction. Prochlorperazine convincingly outperformed promethazine in every category assessed: time to complete relief (twice as fast), more complete relief (twice as good), fewer clinical failures (one-third as many), and less sedation (one-half as often).

Were all these cases due to Rotavirus? Norwalk agent? Viral gastroenteritis of some other etiology? The authors did not address causality specifically, but assumed that viral illnesses comprised the majority of abrupt vomiting cases among adults presenting to the ED. The major limitation is that children, who are afflicted with frequent "viral gastroenteritis" episodes, were excluded. Other anti-emetics, such as metoclopramide (Reglan), trimethobenzamide (Tigan), and ondansetron (Zofran), do not share the combined effectiveness, availability, tolerability, widespread ED usage, and relatively low cost of the two agents studied in this trial. I am now reconsidering how I treat gastroenteritis with vomiting, and how I teach residents about therapeutic advantages. It seems to me that prochlorperazine wins, hands down. ❖

Naloxone After Opioid Overdose: When to Discharge?

ABSTRACT & COMMENTARY

Source: Christenson J, et al. Early discharge of patients with presumed opioid overdose: Development of a clinical prediction rule. *Acad Emerg Med* 2000;7:1110-1118.

EMERGENCY MEDICINE AND TOXICOLOGY TEXTS COMmonly recommend observing patients after opioid overdose for four to 24 hours after the last dose of an opioid antagonist. This suggestion is based on concerns that longer-acting opioids may cause recurrent respiratory depression or delayed onset pulmonary edema. This study from St. Paul's Hospital in Vancouver, Canada, sought to develop a clinical prediction rule to identify patients who may be safely discharged only one hour after the last dose of the opioid antagonist, naloxone.

This prospective cohort study included 573 patients who received naloxone either 0.4 mg intravenously (IV) or 0.8 mg subcutaneously (SQ) during pre-hospital care or in the emergency department (ED). Exclusion criteria included death within one hour of naloxone administration, not having a name documented on the chart, and refusal to consent to follow-up. The investigators recorded the time of naloxone administration, and a formal assessment was planned for one hour later. If naloxone was re-administered prior to the completion of that hour, the clock was restarted and assessment again was planned for one hour later.

Telephone follow-up was attempted for patients who provided phone numbers. Subjects were asked whether they returned to the hospital for any reason within 24 hours of their one-hour assessment. If direct contact could not be made, indirect contacts (friends, family) were attempted to confirm patient status 24 hours post-discharge. A list of subjects who could not be contacted was matched with medical record databases of the six other hospitals in greater Vancouver, Department of Vital Statistics records, and the coroner's office records. For any return hospital visit within 24 hours, the reason for the visit and any adverse events were abstracted from the medical record.

Patients were classified into two groups: those with adverse events within 24 hours and those without. The investigators used classification and regression tree (CART) methodology to develop a decision rule to predict safe discharge. The rule predicted that patients with presumed opioid overdose can be discharged safely one hour after naloxone administration if they have: 1) the ability to mobilize as usual; 2) an oxygen saturation on room air greater than 92%; 3) a respiratory rate between 10-20 breaths/min; 4) a temperature between 35-37.5°C; 5) a heart rate between 50-100 beats/min; and 6) a GCS of 15. The prediction rule had a sensitivity of 99% and a specificity of 40% for predicting adverse outcomes within the first 24 hours.

COMMENT BY JACOB W. UFBERG, MD

This well-done, prospective study has made great strides toward identifying patients who may be safely discharged one hour after the administration of naloxone for opioid overdose. However, it does have several limitations, including direct phone follow-up with only 20% of subjects. However, commendable efforts were made toward contacting patients' friends and relatives and identifying outside hospital visits and deaths. Loss of subjects due to migration is unlikely, as the authors included visits to other Vancouver hospitals and the follow-up period was only 24 hours.

In addition, the authors do not specify the number and type of presumed opioid overdoses other than heroin. Nearly 86% of the study subjects admitted to heroin use prior to their ED visit, and others may have used heroin but denied it during the ED interview. This overwhelming majority of heroin overdoses leads me to believe that this prediction rule may not be applicable to patients who overdose on opioids other than heroin (e.g., orally ingested or longer-acting agents).

Lastly, it is important to note that this project was the development phase of this particular clinical prediction rule and may not be generalizable to other settings. The drug overdose profiles (e.g., mixing of opioids with other drugs of abuse) can differ significantly from region to region, as can practices regarding naloxone administration. In this study, 88% of patients received 0.8 mg naloxone SQ, which may have a longer duration of action than IV naloxone. This may differ from the standard route and dose of naloxone in other hospitals and EMS systems. ❖

Special Feature

Pharmacologically Facilitated Sexual Assault: A Review of "Date Rape" Drugs

By Jacob W. Ufberg, MD

DURING THE LAST SEVERAL YEARS, DRUG-FACILITATED sexual assault has received increased media exposure and public attention. Among the major reasons for

this are the emergence of several relatively new drugs that are easily procured, have a rapid onset of action, and produce anterograde amnesia—making them ideal agents for perpetrators of this crime. Although several agents have been identified as date rape drugs, this article focuses on three newer agents: flunitrazepam, gamma-hydroxybutyrate, and ketamine.

Flunitrazepam

A sedative-hypnotic, flunitrazepam (Rohypnol) is an intermediate-to-long acting benzodiazepine that is banned in the United States, but manufactured legally by Hoffman-LaRoche in Europe and Latin America, where it is used for the treatment of anxiety and insomnia. Based on weight, it is 10 times more potent than diazepam. Flunitrazepam, like other benzodiazepines, acts as an indirect gamma-aminobutyric acid (GABA) agonist. The effects of flunitrazepam are similar to those of other benzodiazepines, and its hypnotic and amnestic effects predominate over its anxiolytic, muscle relaxant, and anticonvulsant effects.

Flunitrazepam was originally manufactured in clear plastic blister packs, available as individually wrapped, 2 mg tablets embossed with the name Roche and the number two in a circle. Legally manufactured flunitrazepam has been reformulated to create an easily identifiable blue color when dissolved in clear beverages, and a haziness in colored beverages. However, illicitly manufactured flunitrazepam remains colorless, odorless, and tasteless when dissolved.[1] Common street names include Roofies, Rochies, Roches, Rope, Mexican Valium, R-2, and Roach-2, among others.

Following oral ingestion, flunitrazepam is 80-90% absorbed by the gastrointestinal tract. Onset of action is within 30 minutes, with peak blood levels achieved in approximately one hour. Flunitrazepam exerts its effects for up to eight hours, and has an elimination half-life of approximately 20 hours. It is hepatically metabolized, with active metabolites that are excreted in the urine; therefore, renal insufficiency will result in accumulation of metabolites and prolonged duration of action.

Symptoms of intoxication resemble those of other benzodiazepines. They include sedation, disinhibition, amnesia, muscular relaxation, respiratory depression, and possibly hypotension. Fatalities due to flunitrazepam overdose are rare unless the drug is taken with alcohol.[2] Emergency department (ED) treatment includes assessment of the ABCs, activated charcoal, symptomatic treatment, and consideration of the benzodiazepine antagonist flumazenil.[3]

Recognition of flunitrazepam toxicity is aided by understanding the usual course of events. During most drug-facilitated sexual assaults, a person at a bar or a party unknowingly ingests a beverage in which a 2 mg tablet of flunitrazepam has been dissolved. Later, the person may awaken partially clothed or nude in a strange place, with little or no memory of the events that occurred. Routine toxicologic testing may not detect flunitrazepam.

Gamma-Hydroxybutyrate (GHB)

Gamma-hydroxybutyrate (GHB) is a short-chain fatty acid derivative of GABA. It is found naturally in human serum, binding in the CNS mainly at a GHB-specific receptor site, and more weakly to the GABA receptor. It is a CNS depressant that is abused as a euphoriant and/or as an anabolic agent (purported to enhance muscle strength and growth hormone release despite no evidence as to its anabolic effects in humans). It is banned in the United States with the exception of FDA-approved research trials; however, it has been used clinically outside of the United States for anesthesia, and treatment of narcolepsy and opiate and alcohol addiction.[4]

GHB is easy to synthesize from common ingredients, with recipes available on the internet and in underground literature. Chemical precursors of GHB, include gamma-butyrolactone (GBL), 1,4 butanediol, and pine needle oil. Any of these chemicals can be readily converted to GHB in a home laboratory. The most common of these, GBL, is marketed as a dietary supplement in many health food stores under the names Blue Nitro and RenewTrient. Following ingestion, GBL is bioconverted to GHB, and has greater bioavailability than GHB. GHB may be produced in a powdered or a liquid form. When mixed in a drink, it is colorless and odorless and may have a salty taste or be tasteless.[1] Common street names for GHB include Grievous Bodily Harm, Liquid Ecstasy, Easy Lay, Salty Water, G-Riffick, Gamma-G, Soap (it is produced from GBL using a saponification reaction), and Georgia Home Boy, among others.

Following oral ingestion, GHB is rapidly absorbed. It is not protein-bound, and it readily crosses the blood-brain barrier. Onset of action occurs within 15 minutes, and duration of action typically is between one and three hours. GHB has an elimination half-life of 27 minutes. The typical euphoric dose of GHB is 10-25 mg/kg. An oral dose of 25 mg/kg of GHB initiates REM sleep, and 60 mg/kg typically induces coma. The primary route of GHB elimination of is through cellular respiration with elimination of carbon dioxide.

GHB intoxicated patients present with a profound decrease in level of consciousness. Emesis is common, and may continue several hours after the sedative effects have worn off. Many patients also may have mild

hypothermia, bradycardia, hypoventilation, and anterograde amnesia.[5] An unusual feature of GHB intoxication is agitation upon stimulation despite prolonged hypoxia or apnea. ED treatment of GHB intoxication includes airway protection and ventilation support if necessary, treatment of symptomatic bradycardia with atropine, and treatment for co-ingestions. Most patients will awaken spontaneously within three hours. There is evidence to indicate that neostigmine or physostigmine may reverse the CNS-depressant effects of GHB, and these agents may be considered in severe cases.[1,4,5]

When used in drug-facilitated sexual assault, victims typically ingest a beverage to which GHB has been added. The drug induces a rapid decrease in level of consciousness, and usually produces complete anterograde amnesia, such that the victim will not remember the assault even if she remained conscious throughout. Like flunitrazepam, routine toxicologic testing will not detect GHB.

Ketamine

Ketamine is a rapid-acting general anesthetic that is a derivative of phencyclidine. It produces profound anesthesia and analgesia while preserving respiratory function and airway protective reflexes. It is used mainly for procedural sedation in the United States, but is used for general anesthesia in third world countries where extensive anesthetic equipment is not available. Ketamine's mechanism of action is complex; it interacts with multiple binding sites including opioid, NMDA, glutamate, nicotinic, muscarinic, and monoaminergic receptors. The analgesic effects are partially mediated by the opioid μ-receptor. However, most of ketamine's analgesic, amnestic, and neurologic effects are due to non-competitive antagonism of the NMDA receptor.[6]

Recreational users of ketamine and perpetrators of drug-facilitated sexual assault generally obtain the drug through theft from sources such as hospitals or veterinary clinics.[6] It is manufactured by Parke-Davis and is available commercially in vials containing 10, 50, or 100 mg per mL. Recreational abusers mix ketamine with cocaine, amphetamines, flour, talc, or vitamins in order to achieve a dissociative hallucinatory state. It can be taken through intravenous, intramuscular, intranasal, or oral routes, with the oral route being used in acquaintance rape. Street names include Special K, K, Super Acid, Green (the color of crystalline ketamine), Purple (the color when mixed with vitamin B_{12}), and Super C.[7]

Following oral ingestion, peak plasma concentrations occur within 30-45 minutes. It has a high volume of distribution, and is approximately 12% protein-bound, with an elimination half-life of two hours. It is metabolized in the liver, with active metabolites that are one-sixth to one-tenth as potent as ketamine. Renal insufficiency may prolong the duration of action, as the kidney excretes the metabolites.

Ketamine intoxication produces profound analgesia, catalepsy, and amnesia. The patient may have nystagmus, and corneal reflexes remain intact. Patients appear to be awake, but are non-communicative and lack physical control. The airway remains patent and the patient maintains respiratory effort, although mild respiratory depression may be present. Salivary and tracheobronchial secretion is stimulated. Ketamine increases heart rate, blood pressure, and cerebral blood flow, leading to increased intracranial pressure. Adverse events include moderate-to-severe emergence reactions, hypertensive crisis, respiratory depression, laryngospasm, stridor, dystonia, and seizures.[7]

ED treatment of ketamine intoxication is mostly supportive. Airway control and ventilatory support must be considered, but usually are not necessary. Naloxone can be considered for reversal of opioid-dependent effects. Seizures can be treated in the standard fashion. Dystonia can be treated with diphenhydramine or benztropine. Hypersecretion can be reversed with atropine or glycopyrrolate. Emergence reactions should be treated with reassurance, a dark, quiet room, and benzodiazepines. Hypertensive crisis can be treated with alpha- and beta-blockade and verapamil. Consideration must be given to possible co-ingestants. Gastric decontamination can be performed for recent ingestions or co-ingestants. There is no known antidote for ketamine.[7]

Similar to flunitrazepam and GHB, patients who are sexually assaulted after ingesting ketamine will have little or no recollection of the event. Routine toxicology screens do not test for ketamine. Assays for serum ketamine levels exist, although they are not commonly available in most institutions.

Summary

Drug-facilitated sexual assault is becoming increasingly common. Flunitrazepam, GHB, and ketamine are being used with increasing frequency due to their rapid onset of action, and the production of disinhibition, muscular control loss, and profound anterograde amnesia. Emergency physicians should be aware of these drugs, their effects, the clinical presentation of acutely intoxicated patients, and treatment modalities, as well as the fact that these agents are not detected on routine toxicologic screening. In cases of drug-facilitated sexual assault, physicians should contact their local or state police crime laboratory or a private laboratory in their area. It also is important to note that urine samples that have been refrigerated or frozen can be tested days or weeks later. ❖

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CME Questions

7. Minimal (< 250 mL) or no fluid seen on abdominal CT after blunt abdominal trauma:
- a. is consistent with significant splenic but not hepatic injury.
- b. is consistent with significant hepatic but not splenic injury.
- c. could be consistent with either significant splenic or hepatic injury.
- d. guarantees the absence of significant splenic or hepatic injury.

8. All of the following statements regarding noninvasive positive pressure ventilation (NPPV) are true *except*:
- a. Several studies have shown that NPPV can reduce the need for endotracheal intubation in some patients with acute exacerbations of chronic obstructive pulmonary disease (COPD).
- b. NPPV has been shown to increase survival in some COPD patients who present with hypercarbia.
- c. In the study by Delclaux et al, treatment with CPAP in patients with acute hypoxemic, non-hypercapnic, respiratory failure was associated with a decrease in the need for intubation when compared to the oxygen alone group.
- d. In the study by Delclaux et al, there was a significantly higher number of adverse events in the CPAP group than in the oxygen alone group.

9. In the face of acute respiratory distress, which of the following clinical entities is most likely to benefit from NPPV?
- a. Spontaneous pneumothorax
- b. Adult respiratory distress syndrome (ARDS)
- c. Trilobar pneumonia
- d. Acute hypercarbic exacerbation of COPD

10. For relief of acute nausea and vomiting of presumed viral etiology in adults, the study by Ernst and colleagues indicated that:
- a. Prochlorperazine is equal to promethazine.
- b. Prochlorperazine is statistically superior to promethazine.
- c. Promethazine is statistically superior to prochlorperazine.
- d. Neither prochlorperazine nor promethazine is effective.

11. In the Ernst et al study comparing prochlorperazine and promethazine in acute nausea and emesis, some patients in each group developed:
- a. anhedonia.
- b. anencephaly.
- c. akathisia.
- d. tardive dyskinesia.

12. In the Vancouver study of predictors of safe discharge after naloxone for opioid overdose, which of the following was a clinical requirement?
- a. Respiratory rate between 10 and 20 breaths/min
- b. Heart rate less than 60 beats/min
- c. Diastolic blood pressure greater than 80 mmHg
- d. Minimental status score of 11

13. Which of the following is *not* a valid criticism of the Vancouver study of predictors of safe discharge after naloxone administration for opioid overdose?
- a. The project was meant to develop a prediction rule, and needs to be validated before implementation.
- b. Most patients overdosed on codeine, and thus the data do not generalize well to other sites.
- c. Route and dose of naloxone administration may vary from site to site.
- d. Follow-up was incomplete, despite the good faith efforts of the investigators.

14. A 20-year-old female is brought in from a party where she has been drinking alcohol. Her friends became alarmed when she became rapidly obtunded, with multiple episodes of vomiting. She presents with a temperature of 96°F, a pulse of 50 bpm, and a blood pressure of 100/56 mmHg. She is comatose, and does not respond to administration of naloxone and oxygen. Her fingerstick blood sugar is normal. After intubation for airway protection, she awakens four hours later, is extubated, and recalls none of the evening's events after arrival at the party. She most likely ingested:
- a. flunitrazepam.
- b. gamma-hydroxybutyrate.
- c. ketamine.
- d. repaglinide.

15. Pharmacologic agents recognized as being used for drug-assisted sexual assault include all of the following *except*:
- a. flunitrazepam.
- b. gamma-hydroyxbutyrate.
- c. ketamine.
- d. repaglinide.

16. During the ED work-up of a victim of possible drug-assisted sexual assault, which of the following would be surprising?
- a. Profound hypotension requiring pressor support
- b. Eventual complete recovery of normal sensorium without pharmacologic intervention
- c. Amnesia for the event
- d. A negative urine toxicology screen

Computer Oversight

By Ken Grauer, MD

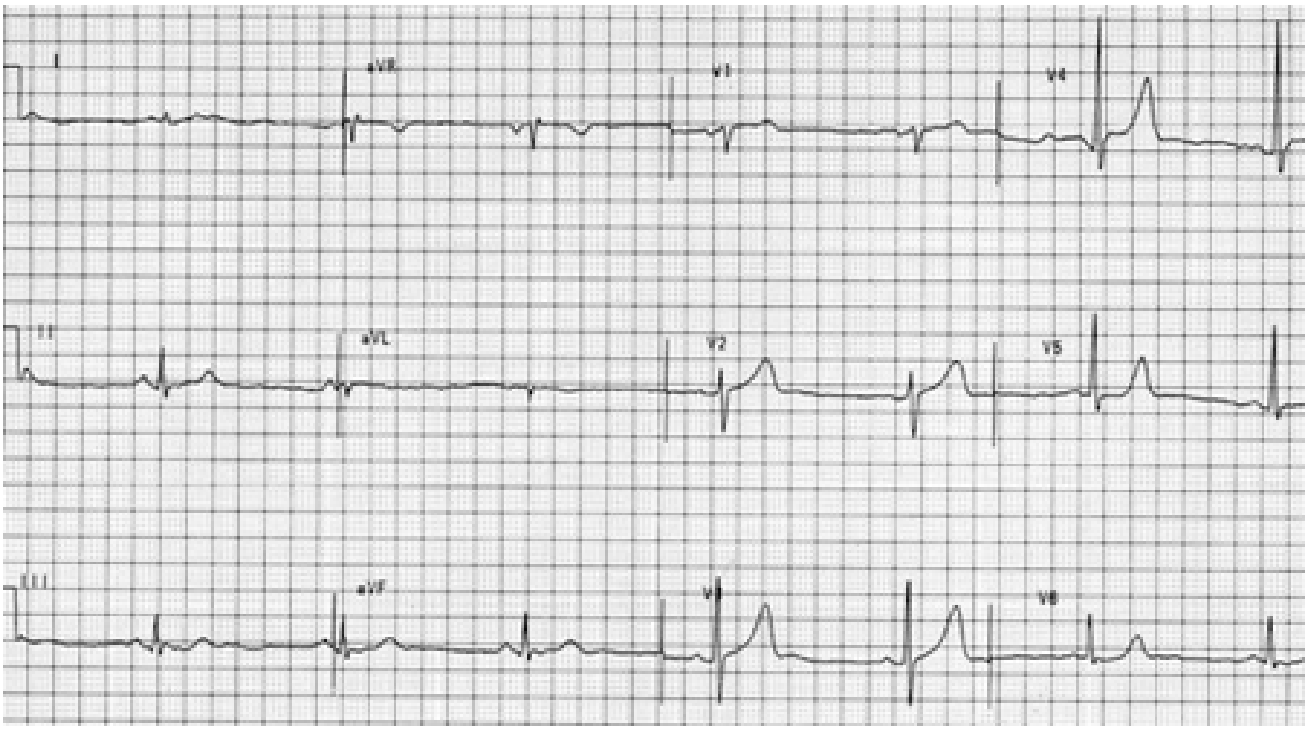

Figure. ECG obtained from a 62-year-old man who was seen in an ambulatory care setting.

Clinical Scenario. This ECG generated a computerized interpretation of "sinus bradycardia—otherwise normal ECG." Do you agree with this interpretation?

Interpretation. The rhythm is sinus bradycardia at a rate of 50 beats/minute. The mean QRS axis and all intervals are normal. QRS amplitude is relatively decreased in the standard limb leads. Transition is normal and occurs between leads V_2 and V_3. There is no sign of chamber enlargement. The most remarkable finding is the presence of tall peaked T waves in most precordial leads. In addition, the ST segment is distinctly flat in leads V_4 through V_6, instead of manifesting the normal smooth upslope with gradual transition into the T wave (as seen in leads V_2 and V_3).

Although hyperkalemia is clearly suggested by T wave appearance in this tracing, serum potassium was not increased. Other than hyperkalemia, T wave peaking in anterior precordial leads may be seen as a normal variant or as a manifestation of myocardial ischemia.

Anterior leads typically reflect a mirror image view of ischemic events that occur in the posterior wall. The "mirror image" view of T wave peaking would be deep symmetric T wave inversion, or a pattern suggestive of ischemia. In support of the interpretation that T wave peaking in anterior precordial leads might reflect posterior ischemia is the finding of ST segment flattening in lateral precordial leads. Such ST flattening may be a subtle sign of coronary artery disease. Clinical correlation would be needed in this case to determine the relevance of these subtle but suggestive ECG signs of potential ischemic heart disease. ❖

In Future Issues:

Phenylpropanolamine and CVA