REVIEW

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Ecstasy-induced rhabdomyolysis and its role in the development of acute renal failure

Maureen Cunningham

Ecstasy (MDMA) is widely used within the dance scene of the 1990s. In this article the growing trend of ecstasy usage among teenagers is considered, in particular, one of its potentially devastating side-effects, rhabdomyolysis. From consideration of the pathophysiology of rhabdomyolysis and its role in the development of acute renal failure, it becomes apparent that preventative measures and prompt treatment can prevent patients developing acute renal failure as a consequence of rhabdomyolysis.

INTRODUCTION

The use of drugs by young people has become prominent in the media by cases like that of Leah Betts, who died shortly after her 18th birthday following the use of ecstasy. Drug-induced rhabdomyolysis is not uncommon; heroin, methadone and temazepam have all been indicated throughout the literature as causes when a patient has been unconscious for some time allowing a crush injury to develop. The focus of this article is on the effects of ecstasy-induced rhabdomyolysis as ecstasy is a commonly used recreational drug among young people in the 16–25 age group (Brown et al 1995), with a wide variation in social class and occupations.

Rhabdomyolysis is a condition seen frequently in some intensive therapy units and is one of the many side-effects of ecstasy use. It is a clinical syndrome of muscle pain, weakness and brown urine due to the breakdown of muscle. This in turn liberates myoglobin into the extracellular fluid with the potential harmful effect of causing acute renal failure (ARF). Rhabdomyolysis was once called Meyer-Betz disease (Knochel 1982). A more appropriate term, myoglobinuria, has been used following the classic description of crush syndrome in patients injured during the bombing of London in World War II (Knochel 1982, Desforges 1990).

Renal failure following rhabdomyolysis is a side-effect of ecstasy use. O'Connor (1994) noted that out of nine documented deaths in Britain due to ecstasy use, five of the patients had rhabdomyolysis among their clinical symptoms; also seven out of 15 recorded cases of hyperpyrexia due to ecstasy, developed rhabdomyolysis. Drug-induced rhabdomyolysis complicated by ARF has a 46% mortality rate (Roth et al 1988).

ECSTASY (3,4 METHYLENEDIOXYMETHAMPHETAMINE OR MDMA)

Ecstasy was first synthesized in 1914 as an appetite suppressant. It was banned in 1977 (Saunders 1993) under the Misuse of Drugs Act 1971 as a class A drug (Wake 1995). This led to a back-street popularization of the drug and it has since maintained a high media profile. O'Connor (1994) stresses that one of the most worrying problems associated with ecstasy is its varied purity. Street specimens range from 50% to 100% purity with variable contaminants, which may include chalk, paracetamol, acid (LSD, lysergic acid), speed (amphetamines) or 'smack' (heroin) (Day 1996).

More common street names for ecstasy are 'white dove', 'E', 'XTC' or 'disco biscuit'. Most of the recent publicity has concentrated on the toxic effects (Gray & Hyde 1990; O'Connor 1994). Green and Goodwin (1996) suggest little attention is paid to the long-term effects, namely neurodegeneration. Robson (1996) agrees, suggesting that death may result due to a massive release and subsequent depletion of brain serotonin.

Henry (1992) suggests tolerance occurs with ecstasy use; some users increase their dose to as many as 10 or more tablets during an evening. It is used for many reasons including inducing a positive mood, increased sensuality, creating a sense of euphoria and a feeling of closeness to others (O'Connor 1994, Robson 1996). There are numerous negative effects of ecstasy including loss of appetite, insomnia, intracerebral haemorrhage, flash-backs and jaw stiffness (Day 1996). Regular users frequently chew gum to

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overcome the effects on their jaw muscles (Henry 1992). Individuals' reaction may be affected by their pre-existing known or unknown underlying conditions (O'Connor 1994). Brown (1987) suggests that exacerbation of coronary artery disease, underlying cardiomyopathy or asthma, may be life-threatening after ingestion of ecstasy. Rhabdomyolysis is one of the serious complications associated with ecstasy. Box 1 gives some advice on how to avoid ecstasy-related complications.

ECSTASY-INDUCED RHABDOMYOLYSIS

Over 100 causes of rhabdomyolysis are recorded in the literature. Box 2 gives a brief overview of some of them.

Box 1 Avoidance of ecstasy-related complications

1. Drink at least a pint of water or lots of soft drinks every hour to avoid dehydration. Not alcohol as this causes further dehydration
2. Don't dance for long periods: take a break
3. Don't mix drugs
4. Wear cool clothing, no hats: avoid being over heated
5. Sit in cool places, use 'time out rooms'
6. Know exactly what is contained in any tablet offered to you

Adapted from Gray & Heather (1990) with permission.

Box 2 Causes of rhabdomyolysis

Drugs
- heroin
- diazepam
- alcohol

Infections
- septic shock
- viral hepatitis
- influenza

Direct muscle injury
- crush injury
- vasculitis
- burns

Decreased energy production
- severe potassium depletion
- hypothermia
- diabetic ketoacidosis

Increased oxygen consumption
- status epileptics
- malignant hyperpyrexia
- exercise

Venom
- snake bite
- hornet
- brown spider

Adapted from Kiely & Kiely (1986) with permission.

How does ecstasy cause rhabdomyolysis?

There are three ways in which ecstasy may induce rhabdomyolysis by causing damage to muscle cells: (i) hyperpyrexia; (ii) increased energy consumption (dancing); or (iii) crush injury when a patient has been unconscious for some time.

Hyperpyrexia

Hyperpyrexia following ecstasy ingestion may set up a response in the body which leads to rhabdomyolysis. Ecstasy ingestion followed by prolonged spells of vigorous physical activity may lead to the development of hyperthermia (O'Connor 1994), it has an ability to disturb the thermoregulatory homeostasis greatly elevating the body temperature (>40°C). Animal studies show that the drug may cause excessive heat production due to serotoninergic mechanisms, which is greater at high ambient temperatures (Henry 1992). O'Connor (1994) describes how hyperpyrexia is associated with severe metabolic acidosis, muscle rigidity, disseminated intravascular coagulation (DIC) and hyperkalaemia. He goes on to state that death may occur within 2–60 h following admission to hospital. Acute renal failure follows when the tissues have been damaged allowing myoglobin to infiltrate the extracellular fluid, eventually leading to damage of the kidneys.

Ecstasy-induced increased energy consumption

Increased energy consumption through exercise, for example, dancing, is an important cause of rhabdomyolysis. Exercise by itself or in the presence of any of the many metabolic disturbances that impair energy production by muscle or oxygen delivery from the blood stream can lead to necrosis and rhabdomyolysis (Sweny 1989). Athletes with an increased muscle mass requiring more oxygen and therefore more blood supply to the muscles, have a predisposition to rhabdomyolysis if the supply does not meet the demand (Better 1992).

Crush injury

Crush injury is possible when a patient has been unconscious for some time following the ingestion of ecstasy. Damage to compressed muscle groups of the arms, legs and sacrum are not uncommon. Skeletal muscle injury may alter the integrity of the cell membrane sufficiently to allow the escape of cell contents into the extracellular fluid. The frequent involvement of muscle in trauma is not surprising. The muscles are the largest organ system in the body, comprising 40–50% of body weight (Better 1992). Failure to appreciate the importance of

muscle necrosis as the underlying problem in the crush syndrome may have disastrous consequences (Shaw et al 1994). Shaw et al (1994) state that there may be a delay in recognizing the signs of muscle compression and necrosis when a patient is first admitted to hospital with a drug overdose. A pressure difference of <30–40 mmHg between the compartment pressure and the diastolic pressure is associated with an inability of muscle to maintain a metabolic state (Shaw et al 1994). Indeed, peak muscular exertion may, within minutes, cause sarcolemmal fluid and electrolyte shifts that mimic those seen following crush injury (Better 1992).

DEVELOPMENT OF RHABDOMYOLYSIS

Rhabdomyolysis is a clinical syndrome resulting from muscle disintegration and myoglobinuria as the myoglobin released from damaged muscles is excreted (Kiely & Kiely 1986). It has been identified as a significant factor in ARF (Kiely & Kiely 1986) and accounts for 5–25% of all cases of ARF (Rowland 1984). Screaton et al (1992) describes a patient admitted to hospital following an ecstasy overdose with a temperature of 43.3°C; 2 h after admission the urine became dark-brown, positive for urinary myoglobin and plasma creatine kinase, suggestive of rhabdomyolysis. DIC was diagnosed soon after admission. He was treated with fresh frozen plasma, and forced alkaline diuresis induced with mannitol and bicarbonate. His neurological condition deteriorated resulting in death. It is important to understand the implications of the patient's signs and symptoms and his treatment.

Diagnosis of rhabdomyolysis

Myoglobinura or haemoglobinuria?

Both free-myoglobin and haemoglobin in the plasma can produce ARF (Sweny 1989). The pathogenesis of ARF with both pigments is very similar, but the causes of myoglobinuria and haemoglobinuria are completely different. The development of ARF depends on the amount of pigment released into the circulation as well as the state of hydration and acid base balance (Sweny 1989). Knochel (1982) describes the function of myoglobin as much like the function of haemoglobin in binding oxygen and facilitating its delivery to the site of oxidative metabolism in the muscle cells.

Myoglobin, a protein with a molecular weight of 17 000 D, is normally found in skeletal and smooth muscles (Keily & Kiely 1986). It is excreted readily by the kidneys and, under normal circumstances, only scant amounts are detectable in plasma. Davies (1995) states that serum levels of myoglobin are influenced by the glomerular filtration rate in the nephron of the kidney. The high renal clearance of myoglobin maintains low plasma concentrations unless muscle necrosis is marked, as in cases of rhabdomyolysis. Myoglobinuria is not detectable until plasma levels exceed 1.5 mg/dl, an amount equivalent to the dissolution of 100 gm of skeletal muscle (Kiely 1986).

The presence of myoglobinuria may not be obvious when a patient is admitted; urinary tests may be the only initial confirmation. Urinary dipstick test for blood will be positive, but no red cells will be seen under the microscope if myoglobin is present. Spun plasma which is clear in colour will assist in the diagnosis. If it is pink this indicates haemoglobinuria rather than myoglobinuria (Kiely & Kiely 1986, Sweny 1989). Myoglobin is not protein bound, therefore, it is filtered rapidly and excreted, allowing plasma to retain its normal colour (Rose 1996). Sweny (1989) indicates that the diagnosis may also be assisted by demonstrating a markedly elevated creatine kinase in the plasma. If appropriate action is not taken the patient is in danger of developing ARF.

DEVELOPMENT OF ACUTE RENAL FAILURE

Acute renal failure is the sudden onset of impaired function (failure to excrete waste products of metabolism) in a patient whose renal function was previously normal. The potential for reversal of ARF is an important distinction from chronic renal failure (CRF) (Hinds 1987, Luce & Pierson 1988). The progression of renal failure due to myoglobinuria is shown in Box 3.

The role of myoglobin in ARF is controversial and the subject of much debate and discussion. Davies (1995) suggests myoglobin is nephrotoxic when the urinary pH is 5.6 or less,

Box 3 Myoglobinuric renal failure

Increased plasma myoglobin
Decreased urinary pH
Myoglobin precipitates: causing cast formation
Acute tubular obstruction (caused by myoglobin precipitate and uric acid crystal precipitate)
Direct damage to epithelial wall of the renal tubules, resulting in back diffusion of ultrafiltrate
Reduced glomerular filtration caused by renal ischaemia and hypoperfusion of the kidneys
Renal necrosis

Adapted from Davies (1995) with permission.

precipitating in the renal tubules and causing tubular obstruction.

Myoglobin in the urine will cause it to be red/brown (Kiely & Kiely 1986, Rose 1996). It is freely filtered at the glomerulus and forms myoglobin casts in the nephron. These casts may appear in the urine as 'brown-sugar' casts (Sweny 1989). The key to diagnosing rhabdomyolysis is assessing a patient's history, to identify the potential for muscle damage, and observing for the appropriate biochemical abnormalities.

Biochemical abnormalities

Rhabdomyolysis may be silent and easily overlooked unless the appropriate biochemical tests are carried out. There are many changes which will take place when a muscle cell is damaged (Figure). When cells are damaged they lose their homeostatic mechanisms (Davies 1995). Sodium, water and calcium enter the cell and the intracellular contents escape. These cell contents include; enzymes such as creatine kinase, transaminase, lactate dehydrogenase and aldolase; the haem pigment, myoglobin; electrolytes such as potassium and phosphates; and purines (Molnar & Shearer 1996). The release of muscle constituents due to leakage of potassium and myoglobin may continue for up to 60 h after the release of a compressed limb (Shaw et al 1994). Under normal conditions myocitic volume is tightly regulated by the delicate balance struck between sarcolemmal leak and ionic extrusion capacity; mainly by sarcolemmal Na–K (sodium–potassium) and ATPase (adenosine triphosphate). During mechanical muscle damage or sometimes even merely severe exertion, this 'pump leak' balance is disturbed (Better 1992).

Acidosis

Metabolic acidosis is a prominent feature of all patients with ARF; as the kidneys are unable to buffer the acidosis (Cheney 1994) there is a decreased reabsorption of sodium bicarbonate and a decreased excretion of acids. Acidosis is an important factor as both myoglobin and haemoglobin break down under acidic conditions to produce ferrihaemate and globin. Ferrihaemate, with a molecular weight of 650 Da can enter tubular epithelial cells and has been shown to be toxic when added to kidney slices (Sweny 1989). Tubular damage is followed by the sloughing of cells and debris into the tubular lumen and may also allow the back diffusion of glomerular filtrate. Hypotension and ischaemia will contribute to tubular damage (Sweny 1989).

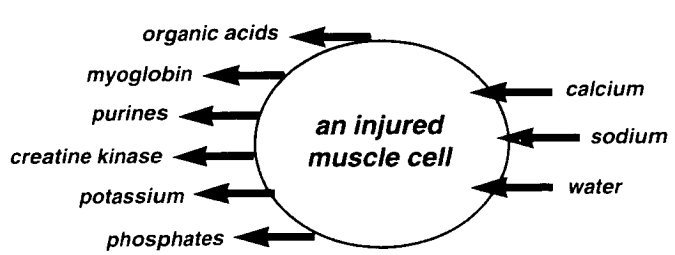

Figure Damage to a muscle cell. Adapted from Davies (1995) with permission.

Creatinine

Creatinine, a by-product of muscle catabolism, is derived from the breakdown of muscle (Kee 1994). Disproportionate rises in serum creatinine levels in relation to serum nitrogen levels are noted (Kiely & Kiely 1986). Plasma urate rises, this is derived from a release of purines, and plasma creatinine from release of muscle creatine.

Creatine kinase

Creatine phosphokinase (CPK or CK) is an enzyme found in high concentrations in the heart and skeletal muscles, and in low concentrations in the brain tissue. High levels of CK-MM are associated with rhabdomyolysis but may also be elevated in intramuscular injections and hypothyroidism (Kee 1994).

Blood urea nitrogen (BUN)

Urea is formed as an end product of protein metabolism and is excreted by the kidneys. An elevated BUN level indicates dehydration, prerenal failure or renal failure (Kee 1994). At levels >70–100 mg/dl uraemic symptoms will appear.

Potassium

The plasma potassium level rises as it is released from damaged cells. Hyperkalaemia becomes a life-threatening complication predisposing the patient to cardiac arrhythmias (Kiely & Kiely 1986). If acute tubular necrosis develops the damaged tubules are not able to excrete potassium, leading to reduced renal excretion and further increasing blood potassium. Acidosis may also increase hyperkalaemia when the kidneys have chosen to excrete hydrogen ions before potassium.

Hypocalcaemia

Hypocalcaemia ensues as a result of calcium phosphate deposition in soft tissue. Supplements of calcium should not be given until phosphate levels return to normal. Once membrane permeability increases or the ion pump fails there will be massive influx of calcium (Sweny 1989). Desforges (1990) states that most infused calcium is deposited in injured muscle, thereby aggravating rhabdomyolysis and causing metastatic calcification. Leakage of calcium will increase the energy requirements for ionic and volume extrusion and deplete cel-

lular ATP stores. At a later stage following trauma, increased catabolism and breakdown of large molecules into smaller ones would be expected to increase intracellular osmolality, and thus aggravate muscle swelling (Better 1992).

Hypercalcaemia

During the recovery phase of rhabdomyolysis, 20–30% of patients will develop hypercalcaemia due to the mobilization of calcium which has been deposited in the injured muscle. Rennie (1996) suggests that low renal blood flow is probably the single most important common denominator for the development of ARF; but damage by calcium and oxygen during reperfusion, and the production of vasoactive agents by the kidney itself are all likely to be involved.

Syndrome of inappropriate antidiuretic hormone secretion (SIADH)

There is a rare association between hyponatraemia and ecstasy ingestion. Ecstasy users are encouraged to drink large quantities of water at 'raves' (dances), but in the presence of SIADH this could lead to rapid hyponatraemia which could result in neurological damage or even death (Brown et al 1995).

Inadequate erythropoietin synthesis

One of the major functions of the kidneys is to stimulate red-blood cell production. Inadequate production of erythropoietin will cause anaemia. Monitoring of haemoglobin and haematocrit levels is essential (Handerhan 1991).

Prevention of renal failure

The protocol adopted by Charing Cross Hospital, London, UK for the prevention of renal failure apparently improves patients' prognoses (Rennie 1996). This is achieved by aiming at normovalaemia, normotension and decreasing action of the ion pump working in the thick ascending loop of Henle. The treatment aimed at prevention of ARF is summarized in Box 4. Early recognition is the key to preventing acute tubular necrosis and severe electrolyte imbalance (Kiely & Kiely 1986, Molnar & Shearer 1996), including identification of at-risk patients, for example, those with volume depletion, alcoholism, or drug-induced unconsciousness (Kiely & Kiely 1986). Maintenance of adequate intravascular volume to prevent hypoperfusion of the kidneys is essential. It has been shown that if adequate fluid replacement is delayed for more than 6 h the incidence of ARF rises rapidly (Molnar & Shearer 1996). Dehydration facilitates the development of ARF in the presence of myoglobinuria (Davies 1995). After ingestion of ecstasy as a recreational drug dehydration is a particular concern, since states of increased energy consumption, for instance, during dancing, may result in increased insensible water loss and leakage of fluid or 'third spacing' of fluid in damaged muscle cells, both of which can cause hypovolaemia (Kiely & Kiely 1986, Davies 1995). Maintenance of adequate urinary output is important, but the amount suggested as adequate is variable between authors; approximately 100–300 ml/h is suggested by Glassock (1984) and Davies (1995) for this situation. The patient's age, weight and previous medical conditions will be taken into account when deciding on the fluid balance. This will allow 'flushing of the kidneys' reducing cast formation, diluting urinary pigments and decreasing intrarenal renin release (Glassock 1984). It is suggested that 10 L of isotonic fluid should be administered within 12–24 h (Knochel 1982). In maintenance of acid-base balance, the use of sodium bicarbonate is controversial; it will induce metabolic alkalosis. Alkalizing the urine may decrease the nephrotoxicity of myoglobin in the urine (Knochel 1982); but a urinary pH value of 5.6 or less can cause myoglobin to dissociate and become more nephrotoxic (Knochel 1982). Correction of hyperkalaemia is one of the most important considerations for these patients due to the possibility of life-threatening arrhythmias. Intensive catabolism associated with rhabdomyolysis may require haemodialysis or haemofiltration to reduce serum urea, nitrogen, potassium and minimize fluid overload.

Box 4 Prevention of acute renal failure

1. Combat hypovolaemia with isotonic saline (1.5 l as soon as the trapped limb is free
2. Infuse hourly 500 ml of crystalloid and 22.4 meq bicarbonate
3. If diuresis is <300 ml/h give mannitol 1 g/kg per dose (Mannitol & bicarbonate should not be continued if a marked diuresis is not achieved, aim at 300 ml/h until myoglobin ceased)
4. If blood pH is >7.4, give 250 mg acetazolamide
5. Assess the need for a CVP and PA Catheter
6. Monitor vital signs hourly, including urine pH and volume
7. Monitor every 6 h osmolarity, electrolytes and blood gases

Adapted from Moshe (1992) with permission.

Nursing care

The hallmark of rhabdomyolysis is haemodynamic shock (Better 1992), this may be due to the occurrence of many of the previously stated abnormalities, including extracellular volume depletion and cardiovascular compli-

cations due to electrolyte imbalance. Treatment should begin early. If rhabdomyolysis is due to a crush injury, therapy can begin before the crushed limb is released, that is, before the haem pigment can be released into the circulation (Rose 1996).

Respiratory care

The patient may be intubated and ventilated in an attempt to assist maintenance of the acid-base balance, allow adequate oxygenation of the tissues and facilitate the removal of retained tracheobronchial secretions using aseptic suctioning techniques. The choice of induction agent by the anaesthetist is important as suxamethonium (a muscle relaxant) can worsen hyperkalaemia and precipitate cardiac arrhythmias (Walsh et al 1994). It is also a potential trigger for malignant hyperpyrexia (Walsh et al 1994), and therefore contraindicated in cases of ecstasy overdose. Samples for blood-gas measurement are taken via an arterial line; it is important for nurses caring for these patients to have a working knowledge of normal/abnormal blood-gas levels. These patients are prone to pulmonary oedema due to the infusion of large volumes of fluid. A chest X-ray will also be required to assess for signs of fluid overload and determine the position of the endotracheal tube following intubation. If the patient had been unconscious before admission, inhalation of gastric contents is a possibility (especially following drug overdose). The nurse will need to assess the air entry at regular intervals, and should send any specimens of tracheal secretion to the bacteriology department, also observing the patient and secretions for signs of infection.

Cardiovascular care

Correcting electrolyte imbalance is of utmost importance. As previously indicated hyperkalaemia is a potential life-threatening complication. Davies (1995) states that 75% of potassium is stored within muscles. When the muscle is damaged potassium leaks out; therefore, a cardiac monitor is used to observe for signs of hyperkalaemia, (ECG changes include peaked T-waves, widening of QRS complexes and flattened P-waves). The kidneys are unable to excrete the increasing potassium released, predisposing the patient to life-threatening arrhythmias. Hyperkalaemia is exacerbated by severe metabolic acidosis resulting from muscle breakdown (O'Connor 1994). The patient will have a central line inserted on admission facilitating the monitoring of fluid status. A pulmonary artery catheter may also be inserted to allow measurement of capillary artery wedge pressure and cardiac output, to guide more fine tuning of fluid balance. Disturbance in the renin–angiotensin–aldosterone system and changes in fluid status may induce hypotension (Handerhan 1991).

Fulminating DIC may be present on admission with severe bleeding requiring replacement of clotting factors (O'Connor 1994). Release of muscle extracts is thought to activate the clotting cascade (Molnar & Shearer 1996). It is important to assess for signs of anaemia, pale-coloured mucous membranes, conjunctiva and skin pallor. Also close observation for signs of bleeding from the gums, intravenous or arterial line insertion sites is necessary.

Renal care

When a patient is admitted to hospital because of ecstasy ingestion, a urinary catheter is inserted allowing the nurse to maintain an accurate fluid balance chart and monitor the acidity of the urine. Correction of hypovolaemia should be initiated before the patient arrives on the ward. As stated previously, 100–300 ml of urine output is required each hour to help prevent ARF developing. If the patient is oliguric, in established renal failure, or has a refractory hyperkalaemia, dialysis or haemofiltration will be required (O'Connor 1994). This will facilitate the removal of fluid, the correction of electrolyte and acid-base balance. The type of replacement therapy chosen will be determined by the patient's cardiovascular stability.

Forced alkaline diuresis

This is the subject of much controversy among many authors. Alkalinization of the urine has a dual purpose of preventing dissociation of myoglobin to ferrihaemate and being prophylactic against hyperkalaemia and acidosis (Molnar & Shearer 1996). Knochel (1982) and Harper (1990) on the other hand do not recommend the use of bicarbonate due to the potential harmful effects of aggravating hypocalcaemia by causing calcium salts to be deposited in the injured muscle. It is also stated that there is little evidence to support the notion that myoglobin is nephrotoxic, and that if myoglobin is given intravenously it will not have an effect on the renal system if there is a good urine output. It only begins to cause problems when the patient is acidotic (urinary pH of less than 5.6) or dehydrated. Rose (1996) indicates that raising the urinary pH to >6.5 can markedly diminish the renal toxicity of myoglobin. He goes on to suggest there is no clinical evidence that an alkaline diuresis is more effective than a saline diuresis and agrees there is a potential risk in alkalinization, since it promotes precipitation of calcium phosphate and induces or exacerbates hypocalcaemia. O'Connor (1994) warns of the dangers of

giving the fluid to induce a forced diuresis in a patient with ecstasy overdose, as it may worsen impending pulmonary oedema.

Better (1992) states that mannitol (an osmotic diuretic) may improve systemic haemodynamics, blood pressure and coronary blood flow; it stimulates the release of atrial natriuretic factor, protecting the kidneys. Johnston et al (1981) explains that mannitol elevates renal blood flow, and reduces renal vascular resistance by reducing afferent arteriolar resistance. Rose (1996) also suggests it acts as a free radical scavenger thereby minimizing cell injury. It is prescribed normally as 100 ml of 25% solution over 15 min. It is important to check the urinary pH hourly to ensure the aim of alkalizing the urine is being met.

Frusemide therapy

Loop diuretic agents have the theoretical disadvantage of acidifying the urine, whereas alkalinity is beneficial for these patients according to Desforges (1990). He goes on to say that loop diuretics may be required for elderly patients who cannot cope with large volumes of fluid.

Gastrointestinal care

Hypoglycaemia has been noted in some patients, so regular measurement of the patient's blood glucose is required. As previously stated the patient may be at risk of developing DIC, therefore, the stools and nasogastric contents will be observed for blood. The body will attempt to meet the changing metabolic requirements, hence nutritional support is required to lower BUN levels, sparing protein levels, preserving muscle, and assisting in the prevention of infection by supporting the immune system (Cheney 1994). A possible 70% of all patients with ARF develop infection (Cheney 1994).

Wound care

If rhabdomyolysis is due to compression of a limb, for example, after drug overdose, road traffic accident or cerebrovascular accident, it is important to assess the affected area and document the size and description of any wound. If compartmental syndrome is suspected (tender painful areas, which are swollen and hard) the intracompartmental pressure should be monitored and possibly decompression faciotomy preformed (Shaw et al 1994, Sweny 1989).

Medication

As with CRF, patients with ARF are intolerant of many drugs, there is a potential for prolonged sedation from sedative and analgesics, and bizarre neurological reactions to cimetidine, chlorpromazine and antibiotics are all too frequently encountered. Great care is required in the choice, timing and dosage of drugs used (Sweny 1989).

Temperature control

Screaton et al (1992) believes the most important consideration for a patient with rhabdomyolysis following ecstasy overdose is rapid cooling, as hyperpyrexia may well be the initiating factor for rhabdomyolysis and DIC. Currently, dantrolene is the drug of choice to control body temperature, in conjunction with passive and active cooling methods (O'Connor 1994). The patient should be cooled with ice packs, cold intravenous fluids and dantrolene 1 mg/kg, repeated after 20 min (Walsh et al 1994). Blood pressure, peripheral and core temperature should be closely monitored.

Anxiety and fear

As with all patients who are ventilated, good verbal and non-verbal communication are essential. Clear and simple explanations must be given at all times. The nurse must be aware of the possible reasons for the patient's anxiety, such as pain, fear over the possibility of losing a limb, drug withdrawal, possibility of long-term renal failure, or fear of death, as well as the unfamiliar sights and sounds of the intensive therapy area.

CONCLUSION

There is much debate about ecstasy, and whether it should be legalized. Saunders (1993) defends the use of ecstasy, disputing scientific evidence of brain damage, psychological damage, and death due to this cause. He suggests ecstasy has reduced football violence, is a treatment for emotional illness, helps patients to relax and express themselves freely and honestly. Green and Goodwin (1996) state:

> No-one should seriously consider legalising a compound that can be shown to cause long-term neurodegeneration in rodents and primates at doses that do not differ from those used recreationally by humans.

It is understandable how young people attending 'raves' are confused at the mixed message given by health professionals and the media. It is important that they be given practical information as to how to avoid the complications of taking ecstasy. Brown (1995) describes a herbal ecstasy which contains a number of herbs from around the world. It is said to be '100% natural with no side effects', it is legal in the UK but further research is required before it can be promoted as 'safe ecstasy'.

The debate will go on for some time and it is outwith the scope of this article to give an opinion. But it must be emphasized that there are many side-effects of ecstasy use which have not been publicized in the press. It has been shown in this paper how the serious effect of rhabdomyolysis is caused and how acute renal failure may be prevented. As Gray and Hyde (1990) states 'the number of people taking illicit drugs each year is on the increase'; therefore, the amount of people admitted to hospital each year with drug-related problems is ever-increasing. A study undertaken in Edinburgh, UK, examined the urine of 30 people attending a 'rave'; 12 tested were positive for ecstasy (Brown 1995). In another study, in Glasgow, UK, 135 participants of 'raves' were interviewed, 91.1% admitted to having tried ecstasy (Forsyth 1996), indicating the widespread use of ecstasy among young people. Forsyth (1996) emphasizes that the mixing of different drugs will increase the risk of drug-related harm.

Rhabdomyolysis is only one of the serious side-effects of which nurses should be aware. The treatment of rhabdomyolysis is again controversial. It seems the aim should be prompt treatment with intravenous saline. Unfortunately some cases go undetected until muscle injury is apparent and ARF is in progress. These patients may require mannitol and bicarbonate infusions.

Once acute renal failure is established the aim of treatment is to support the kidneys and preserve the remaining renal function, possibly with dialysis. It is important to prevent secondary insult to the kidney (hypotensive episodes or nephrotoxic drugs). It is only with awareness of this condition that nurses and doctors may be able to prevent some patients developing chronic renal failure.

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