

Blood (5 packs [2–12]) and fresh plasma (4 packs [2–12]) transfusions were required within the first 24 hours. Hemorrhages (9/57), thrombosis (3/57) or lower limb ischemia (4/57) seemed equivalent to previous series using more complicated anticoagulation protocols. **Conclusions:** Poisoned patients present at ECLS time with important alteration in their clotting tests, associated with various degrees of hepatocellular failure, DIC, defibrination, as well as dilution. A simple heparin protocol appears optimal to reduce complications in these critical situations.

190. 1-Benzylpiperazine (BZP) and 3-trifluoromethylphenylpiperazine (TFMPP) Detection in Recreational Drug Users Presenting to the Emergency Department

Wood DM,¹ Button J,² Lidder S,¹ Ramsey J,² Holt DW,² Dargan PI.¹ ¹Guy's & St Thomas' Poisons Unit, Guy's & St Thomas' NHS Foundation Trust, London; ²Analytical Unit, St George's, University of London, London, UK.

Background: Recreational drug use is common throughout Europe and is increasing. Legally available synthetic compounds with actions similar to controlled recreational drugs have previously been sold as 'recreational drugs' within nightclubs (1). **Case series:** Three male patients (aged 18, 18 and 19) presented to the Emergency Department (ED) following use of supposed 'MDMA' purchased within a nightclub. All had ingested 4 of the purchased tablets over the course of an evening. Following ingestion, they developed symptoms of anxiety, nausea, vomiting, non-specific abdominal pain and dizziness. On presentation they had features of sympathomimetic toxicity (tachycardia, bruxism and dilated pupils); none were pyrexial; one had inducible clonus, hypertonia and hyperreflexia. They were treated with IV fluids and oral diazepam and admitted for observation. All were asymptomatic after 6 hours observation and were discharged home with no long-term sequelae. **Results:** Serum samples obtained from them on admission were subsequently analysed by gas chromatography-mass spectrometry (GC-MS). BZP (260–270 ng/mL) and TFMPP (30–60 ng/mL) were detected in all of the samples obtained; no other recreational drugs or other compounds, apart from diazepam given in the ED, were detected with extended toxicological screening. **Conclusion:** We report here a case series of 3 patients who presented following ingestion of a combination of BZP and TFMPP, purchased as supposed 'MDMA', who developed features of sympathomimetic toxicity with associated anxiety. Clinical Toxicologists should be aware of the possibility that patients presenting following use of recreational drugs, may have unknowingly ingested 'novel' or legally available drugs. Increased surveillance using toxicological screening is necessary to monitor trends in recreational drug use and the emergence of novel recreational drugs. **Reference:** 1. Wood DM, Dargan PI, Button J, et al. Collapse, reported seizure and an unexpected pill. *Lancet* 2007; **369**: 1490.

191. Development of the P-CAP (Poisoned Clubbers Avoidance Project) Ambulance Referral Guidelines

Wood DM,¹ Allid G,² Greene SL,¹ Lovitt C,³ Heather K,¹ Jones AL,⁴ Drake N,⁵ Dargan PI.¹ ¹Guy's & St Thomas' Poisons Unit, Guy's & St Thomas' NHS Foundation Trust, London; ²Lambeth Police LGBT Liaison Team, London; ³Department of Health, London, UK; ⁴University of Newcastle, NSW, Australia; ⁵Emergency Department, Guy's and St Thomas' NHS Foundation Trust, London, UK.

Objective: Recreational drug use and acute toxicity associated with recreational drugs is common amongst clubbers. Therefore many large clubs have 'club first-aiders', who undertake the initial assessment of individuals with acute toxicity. We became aware of a number of instances in which patients with significant poisoning were not identified by 'club first-aiders' resulting in delayed transfer to and initiation of appropriate management in hospital. The 'Safer Clubbing' initiative provided guidance to promote the safety of clubbers (1), but did not include recommendations on when individuals with acute recreational drug toxicity should be referred to hospital. **Methods:** A working party of Clinical Toxicologists, Emergency Physicians, the Metropolitan Police and Club owners/'first-aiders' was established. Guidelines for when an ambulance should be called for those with recreational drug toxicity were developed to try and identify those with significant toxicity requiring immediate hospital assessment. These guidelines were audited six months after introduction. **Results:** The audit identified training needs for 'club first-aiders' in assessing recreational drug toxicity. A training module was developed and delivered to them and the guidelines were edited to clarify areas of uncertainty identified. After the training, 12/13 (92.3%) and 13/13 (100%) of 'club first-aiders' were confident in the assessment of poisoned clubbers and use of the P-CAP guidelines respectively. **Conclusions:** This initiative has demonstrated the benefits of a multi-disciplinary, multi-agency approach to the management of clubbers with recreational drug toxicity. Wider dissemination of these guidelines through an update of "Safer Clubbing" is planned which should lead to improved pre-hospital management of poisoned clubbers. **Reference:** 1. Safer clubbing: Guidance for licensing authorities, club managers and promoters http://www.cityoflondon.gov.uk/Corporation/our_services/law_order/community_safety/drugs_action_team/dat_clubbing.htm

192. Withdrawn

193. Detection of the Novel Recreational Drug Diphenyl-2-pyrrolidinemethanol (D2PM) Sold 'legally' in Combination with Glauconine

Wood DM,¹ Button J,² Lidder S,¹ Ovaska H,¹ Ramsey J,² Holt DW,² Dargan PI.¹ ¹Guy's & St Thomas' Poisons Unit, Guy's & St Thomas' NHS Foundation Trust, London; ²Analytical Unit, St George's, University of London, London, UK.

Background: There is increasing use of synthetic 'herbal highs' sold legally within Europe, either through self-purchase from high-street shops/the internet, or sold as 'recreational drugs' by illegal dealers (1). The European Monitoring Centre for Drugs and Drug Abuse (EMCDDA) review the legal status of novel recreational drugs, but can only do so when more information on their use and clinical effects becomes available. **Case report:** A 20 year old male presented to the ED with ischaemic sounding chest pain and palpitations lasting 1 hour following ingestion of 3 legally purchased 'BZP-free herbal highs' and one 'sniff' of amyl nitrate. He had features of sympathomimetic toxicity with hypertension (213/109), tachycardia (125) and dilated pupils; neurological examination and temperature were normal. A 12-lead ECG and methaemoglobin concentration (0.5%) were normal on presentation; Troponin T at 12

hours was not elevated (<0.01 microgram/L). He was treated with oral diazepam 10 mg and his tachycardia and hypertension settled. Following 12 hours observation he was asymptomatic and he was discharged with no long-term sequelae. **Results:** A serum sample obtained on admission to the ED was analysed by gas chromatography-mass spectrometry (GC-MS). Glauconine and D2PM were detected; no other recreational drugs or other compounds, apart from diazepam given in the ED, were detected with extended toxicological screening. **Conclusion:** We report a case of ingestion of a 'BZP-free herbal high', which contained a combination of Glauconine and D2PM. This is the first report of recreational use of D2PM. Involvement of clinical toxicologists and increased toxicological screening will improve the detection of novel recreational drugs and assist legislative authorities. **Reference:** 1. Wood DM, Dargan PI, Button J, et al. Collapse, reported seizure - and an unexpected pill. *Lancet* 2007; **369**: 1490.

194. Chronic Volatile Nitrite Neurotoxicity

Halcomb SE,¹ Griffey RT,¹ Parks DA,² Mullins ME.¹ ¹Division of Emergency Medicine, Washington University, St. Louis; ²Department of Internal Medicine, Washington University, St. Louis, US.

Objective: Nitrous oxide abuse has been associated with a disabling polyneuropathy that resembles subacute combined degeneration of the cord coupled with pernicious anemia. To date, no other nitrites have been reported to cause a similar syndrome. We report a case of chronic amyl and butyl nitrite abuse that resulted in the development of profound ataxia and pernicious anemia. **Case report:** A 65 year old man with a history of migraine headaches and chronic renal insufficiency presented to our ED complaining of sudden onset of bilateral lower extremity weakness that resulted in a fall. The patient described a two year history of increasing balance problems especially when he was walking in the dark or climbing stairs. This progressive ataxia was accompanied by a cognitive decline which the patient characterized as memory loss. Of note, the patient had been diagnosed with pernicious anemia by his primary care physician around the same time as these symptoms began, and was started on B-12. Upon questioning, the patient admitted to using "poppers" for sexual excitation 2–3 times per week since 1980. On exam the patient had normal vital signs, paratonia, a wide based gait, intention tremor, a slight decrease in vibration sense and dysdiadochinesis. Brain and brainstem MRI revealed frontal and cerebellar atrophy. The patient's symptoms improved during his admission and he was discharged with close neurologic follow-up. Several months later, he presented to the ED with a mild recurrence of his symptoms which improved with observation. However, he did exhibit a persistent residual effect upon discharge. **Discussion:** Nitrous oxide oxidizes cyanocobalamin and irreversibly inhibits the enzyme methionine synthase. This prevents the synthesis of methionine and tetrahydrofolate, which can lead to diminished myelin production and decreased DNA synthesis producing neuropathy and a pernicious anemia-like syndrome. We propose that chronic abuse of volatile nitrites may have caused this patient's symptoms. **Conclusion:** Chronic volatile nitrite abuse may lead to a syndrome similar to subacute combined degeneration of the cord.

195. Amphetamine-Induced Cardiomyopathy

Armstrong JM,¹ Pascu OC,¹ Daly FFS,^{2,3} McCoubrie D,^{2,3} Graudins A,³ Murray L.^{1,3} ¹Toxicology Service, Sir Charles Gairdner Hospital, Perth; ²Toxicology Service, Royal Perth Hospital, Perth; ³NSW Poisons Information Centre, Childrens Hospital at Westmead, Sydney, Australia.

Objective: To describe three cases of acute amphetamine-induced cardiomyopathy, with a discussion of pathophysiology and literature review. **Case series:** Case one presented 10 hours after IV amphetamine with cardiogenic shock and pulmonary oedema, and died in the Emergency Department. Two further cases with similar presentations subsequent to amphetamine use required ICU admission and multiple inotropic agents, with an intra-aortic balloon pump used in one case. Both surviving cases had markedly decreased ejection fractions on initial echocardiography, with resolution to normal by the time of discharge. Pathophysiological changes may be similar to the catecholamine-induced cardiomyopathy seen in pheochromocytoma. **Conclusion:** Amphetamine-induced cardiomyopathy can present in extremis with cardiogenic shock. Extraordinary measures such as mechanical circulatory support are justified as complete resolution of the toxicity is possible. **References:** 1. Watts DJ, McColester L. Methamphetamine-induced myocardial infarction with elevated troponin I. *Am J Emerg Med* 2006; **24**: 132–4. 2. Turnipseed SD, et al. Frequency of acute coronary syndrome in patients presenting to the emergency department with chest pain after methamphetamine use. *J Emerg Med* 2003; **24**: 369–73. 3. Wijetunga M, et al. Crystal methamphetamine-associated cardiomyopathy: tip of the iceberg? *J Tox Clin Tox* 2003; **41**: 981–6.

196. Neurological Complications Related to Cocaine Use

Carcelen ME,¹ Cervello A,¹ Gullen C,¹ Brocalero A,¹ Castillo A,¹ Climent B,² Garcia D.² ¹Neurology Department, Consorcio Hospital General Universitario, Valencia; ²Clinical Toxicology Unit, Consorcio Hospital General, Valencia, Spain.

Objective: To evaluate the impact that the extensive use of cocaine is producing on the nervous system. **Methods:** Retrospective study in patients hospitalized in the Neurology Department of our hospital between September 2004 and May 2007. Patients with stated use of cocaine and having positive levels at admission. Patients without objective data on its use were excluded. **Results:** 20 cases with an average age of 34.5 years (range 16–48 years). 12 men and 8 women. Toxicological history: tobacco use 85%, alcohol abuse 65%, cannabis 30%, designer drugs 15% and opiates 10%. Intranasal administration 95%. Main diagnosis: ischemic stroke in 7 cases (35%) and haemorrhagic stroke in 3 cases (15%), headache in 2 cases (10%), psychiatric disorder in 5 cases (25%), seizure crisis in 2 cases (10%), and other diagnoses (CCT) in 1 case (5%). Secondary diagnoses: cephalgia in 3 cases (15%), seizure crisis in 1 case (5%), discopathy in 2 (10%), leukoencephalopathy in 1 case (5%) and others in 3 cases (15%). The average number of hospitalization days: 8.8 days (range 3–15 days). Neurological history: 6 cephalgia cases (30%), 1 case of polyradiculoneuritis (5%). Psychiatric history: 3 cases of mood disorders (15%) and 1 case of personality disorder (5%). Cardiovascular risk factor: 3 cases of dyslipidemia (15%), 1 case of diabetes mellitus (5%) and 1 case of high BP (5%). Laboratory data revealed dyslipidemia in 6 cases (30%) and acute rhabdomyolysis in 1 case (5%). Neurological sequelae in 5 cases (25%); 2 cases with motor deficit, 1 case with sensory deficit, 1 case with sensory - motor, language and visual deficit and 1 case of vigilant coma. Fatal cases: 2 cases (10%): 1 by massive ischemic stroke and posterior hernia and 1 by brain stem - encephalic