

Current Awareness in Clinical Toxicology

Editors: Sarah Cage MSc and Allister Vale MD

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CURRENT AWARENESS PAPERS OF THE MONTH

Should β -blockers be used in the treatment of cocaine-associated acute coronary syndrome?

Page RL, II, Utz KJ, Wolfel EE. *Ann Pharmacother* 2007; 41: 2008-13.

Objective: To critically evaluate the 30 year debate of β -blocker use in cocaine-induced acute coronary syndrome (CIACS).

Data sources: An Ovid Medline In-Process and Other Non-Indexed Citations, Ovid Medline Daily, and Ovid Medline (1966-August 21, 2007) search of the medical literature was conducted using the key terms cocaine, myocardial infarction, acute coronary syndrome, and adrenergic β -antagonists.

Study selection and data extraction: All clinical trials, case reports, national cardiovascular guidelines, and reviews published in English were evaluated. Case reports were included based on whether (1) acute coronary syndrome was suspected, (2) a β -blocker was used during the treatment course, and (3) objective and subjective patient-specific information was documented.

Data synthesis: Three case reports and 2 placebo-controlled trials were identified that used 4 β -blockers (atenolol, labetalol, metoprolol, propranolol). Three national guidelines addressed β -blocker use. Although published data are limited, propranolol and labetalol exert minimal to no effect on alleviating cocaine-induced coronary vasoconstriction. None of the evaluated national guidelines recommends β -blockers as first-line agents in CIACS management.

Conclusion: β -Blockers should not be considered first-line agents for controlling chest pain in patients with documented CIACS. If long-term β -blockade is warranted, its benefits should be weighed against recurrent use of cocaine and possible exacerbation of acute coronary syndrome. Given that carvedilol exhibits ancillary pharmacologic properties beneficial in CIACS, and post-myocardial infarction mortality data are available regarding its use, this agent could be considered to be appropriate therapy.

Survey of cases of paracetamol overdose in the UK referred to National Poisons Information Service (NPIS) consultants

Thanacoody HKR, Good AM, Waring WS, Bateman DN. *Emerg Med J* 2008; 25: 140-3.

Background: Paracetamol is the most common means of drug overdose in the UK. Guidance on management is available to junior doctors through TOXBASE, the online resource managed by the UK National Poisons Information Service (NPIS) and in poster form. TOXBASE is supported by NPIS units and further by a UK national rota of clinical toxicologists. A study was undertaken to

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examine reasons why calls about paracetamol are referred to consultants to better understand issues in managing this common poisoning.

Methods: Calls relating to paracetamol overdose referred by a poisons information specialist to the duty NPIS consultant between 1 May 2005 and 30 April 2006 were identified from the database and the number of TOXBASE accesses during the same time period was determined. Enquiries that resulted in consultant referral were classified into six categories.

Results: Calls referred to NPIS consultants pertain mainly to patients who present late, staggered overdoses, adverse reactions to N-acetylcysteine, and interpretation of blood results. This information has been used to inform the development of TOXBASE so that comprehensive advice is readily available to end users.

Conclusions: The operation of a national consultant rota enables information on difficult or unusual cases of poisoning to be pooled so that treatment guidelines can be developed to optimise treatment throughout the UK.

Use of benzodiazepines and benzodiazepine receptor agonists during pregnancy: neonatal outcome and congenital malformations

Wikner BN, Stiller CO, Bergman U, Asker C, Källén B. *Pharmacoepidemiol Drug Saf* 2007; 16: 1203-10.

Background: Exposure to Benzodiazepines (BZD) during foetal life has been suggested to contribute to neonatal morbidity and some congenital malformations, for example, orofacial clefts. Here we aimed to study the neonatal outcome and congenital malformations in neonates whose mothers reported use of BZD and/or hypnotic benzodiazepine receptor agonists (HBRA) during pregnancy.

Methods: In the Swedish Medical Birth Register we identified 1979 infants whose mothers (n = 1944) reported use of BZD and/or HBRA in early pregnancy. An additional 401 infants were studied, born to 390 mothers who were prescribed such drugs during late pregnancy. Neonatal outcome including congenital malformations after exposure was compared with that of all births (n = 873 879).

Results: An increased risk for preterm birth and low birth weight was detected in the exposed population. The rate of relatively major congenital malformations was moderately increased among infants exposed in early pregnancy (adjusted OR = 1.24, 95%CI 1.00-1.55), not explained by known teratogenic maternal co-medication. A higher than expected number of infants with pylorostenosis or alimentary tract atresia (especially small gut) was found. This was, however, based on only seven infants for each group of malformation without association to any specific BZD or HBRA. The earlier proposed increased risk for orofacial clefts was not confirmed in our study.

Conclusions: Maternal use of BZD and/or HBRA may increase the risk for preterm birth and low birth weight and cause neonatal symptoms, but does not appear to have a strong teratogenic potential. The tentative association with pylorostenosis and alimentary tract atresia needs confirmation.

Assessment of cognitive brain function in ecstasy users and contributions of other drugs of abuse: results from an fMRI study

Jager G, De Win MML, van der Tweel I, Schilt T, Kahn RS, Van den Brink W, van Ree JM, Ramsey NF. *Neuropsychopharmacology* 2008; 33: 247-58.

Abstract: Heavy ecstasy use has been associated with neurocognitive deficits in various behavioral

and brain imaging studies. However, this association is not conclusive owing to the unavoidable confounding factor of polysubstance use. The present study, as part of the Netherlands XTC Toxicity study, investigated specific effects of ecstasy on working memory, attention, and associative memory, using functional magnetic resonance imaging (fMRI). A large sample (n=71) was carefully composed based on variation in the amount and type of drugs that were used. The sample included 33 heavy ecstasy users (mean 322 pills lifetime). Neurocognitive brain function in three domains: working memory, attention, and associative memory, was assessed with performance measures and fMRI. Independent effects of the use of ecstasy, amphetamine, cocaine, cannabis, alcohol, tobacco, and of gender and IQ were assessed and separated by means of multiple regression analyses. Use of ecstasy had no effect on working memory and attention, but drug use was associated with reduced associative memory performance. Multiple regression analysis showed that associative memory performance was affected by amphetamine much more than by ecstasy. Both drugs affected associative memory-related brain activity, but the effects were consistently in opposite directions, suggesting that different mechanisms are at play. This could be related to the different neurotransmitter systems these drugs predominantly act upon, that is, serotonin (ecstasy) vs dopamine (amphetamine) systems.

The dark side of ecstasy: neuropsychiatric symptoms after exposure to 3,4-methylenedioxymethamphetamine

Karlsen SN, Spigset O, Slordal L. Basic Clin Pharmacol Toxicol 2008; 102: 15-24.

Abstract: 3,4-Methylenedioxymethamphetamine (MDMA, ecstasy) is a known neurotoxin in animals. This review discusses the history, pattern of use, pharmacology, acute and long-term effects of MDMA. Emphasis is given to the concern that MDMA may induce long-term cognitive and psychiatric effects. MDMA is an illegal substance, and investigations of the effects of exposure in human beings have limitations and weaknesses. There are numerous studies suggesting a correlation between MDMA exposure and psychopathology, and that the psychotropic effects may be long-lasting or permanent. However, it is not possible to conclude that there is a causal relationship between exposure and the increased psychopathology observed in MDMA users. Longitudinal studies are needed to assess whether MDMA causes persistent cognitive impairment and/or psychiatric symptoms in human beings.

A community-based evaluation of sudden death associated with therapeutic levels of methadone

Chugh SS, Socoteanu C, Reinier K, Waltz J, Jui J, Gunson K. Am J Med 2008; 121: 66-71.

Background: Published case reports have associated the therapeutic use of methadone with the occasional occurrence of sudden cardiac death. Because of the established utility of this drug and with the eventual goal of enhancing safety of use, we performed a community-based study to evaluate this association.

Methods: During a 4-year period, we prospectively evaluated all patients who consecutively had sudden cardiac death and underwent investigation by the medical examiner in the metropolitan area of Portland, Ore. Case subjects of interest were those with a therapeutic blood level of methadone (<1 mg/L), and case comparison subjects were those with no methadone identified. Patients with recreational drug use or any drug overdose were excluded from either group. Detailed autopsies were conducted, including the detection and quantification of all substances in the blood.

Results: A total of 22 sudden cardiac death cases with therapeutic levels of methadone (mean 0.48 ± 0.22 mg/L; range 0.1-0.9 mg/L) were identified (mean age 37.0 ± 10 years, 68% were male) and compared with 106 consecutive sudden cardiac death cases without evidence of methadone (mean age 42 ± 13 years, 69% were male). The most common indication for

methadone use was pain control (n = 12, 55%). Among cases receiving methadone therapy, sudden death-associated cardiac abnormalities were identified in only 23% (n = 5), with no clear cause of sudden cardiac death in the remaining 77% (n = 17). Among cases with no methadone, sudden death-associated cardiac abnormalities were identified in 60% (n = 64, P = .002).

Conclusion: The significantly lower prevalence of cardiac disease in the case group implicates methadone, even at therapeutic levels, as a likely cause of sudden death. These findings point toward an association between methadone and occurrence of sudden death in the community. Clinical safeguards and further prospective studies specifically designed to enhance safety of methadone use are warranted.

A Parkinsonian syndrome in methcathinone users and the role of manganese

Stepens A, Logina I, Liguts V, Aldins P, Eksteina I, Platkajis A, Martinsone I, Terauds E, Rozentale B, Donaghy M. N Engl J Med 2008; 358: 1009-17.

Background: A distinctive extrapyramidal syndrome has been observed in intravenous methcathinone (ephedrone) users in Eastern Europe and Russia.

Methods: We studied 23 adults in Latvia who had extrapyramidal symptoms and who had injected methcathinone for a mean (\pm SD) of 6.7 ± 5.1 years. The methcathinone was manufactured under home conditions by potassium permanganate oxidation of ephedrine or pseudoephedrine. All patients were positive for hepatitis C virus, and 20 were also positive for the human immunodeficiency virus (HIV).

Results: The patients reported that the onset of their first neurologic symptoms (gait disturbance in 20 and hypophonia in 3) occurred after a mean of 5.8 ± 4.5 years of methcathinone use. At the time of neurologic evaluation, all 23 patients had gait disturbance and difficulty walking backward; 11 patients were falling daily, and 1 of these patients used a wheelchair. Twenty-one patients had hypophonic speech in addition to gait disturbance, and one of these patients was mute. No patient reported decline in cognitive function. T1-weighted magnetic resonance imaging (MRI) showed symmetric hyperintensity in the globus pallidus and in the substantia nigra and innominata in all 10 active methcathinone users. Among the 13 former users (2 to 6 years had passed since the last use), lesser degrees of change in the MRI signal were noted. Whole-blood manganese levels (normal level, $<209 \text{ nmol.L}^{-1}$) averaged 831 nmol.L^{-1} (range, 201 to 2102) in the active methcathinone users and 346 nmol.L^{-1} (range, 114 to 727) in former users. The neurologic deficits did not resolve after patients discontinued methcathinone use.

Conclusions: Our observation of a distinctive extrapyramidal syndrome, changes in the MRI signal in the basal ganglia, and elevated blood manganese levels in methcathinone users suggests that manganese in the methcathinone solution causes a persistent neurologic disorder.

Protective ventilation strategies in the management of phosgene-induced acute lung injury

Parkhouse DA, Brown RF, Jugg BJ, Harban FM, Platt J, Kenward CE, Jenner J, Rice P, Smith AJ. Mil Med 2007; 172: 295-300.

Abstract: Phosgene is a chemical widely used in the plastics industry and has been used in warfare. It produces a life-threatening pulmonary edema within hours of exposure, to which no specific antidote exists. This study aims to examine the pathophysiological changes seen with low tidal volume ventilation (protective ventilation (PV)) strategies compared to conventional ventilation (CV), in a model of phosgene-induced acute lung injury. Anesthetized pigs were instrumented and exposed to phosgene (concentration \times time (Ct), $2350 \text{ mg.min.m}^{-3}$) and then ventilated with intermittent positive pressure ventilation (tidal volume (TV) = 10 ml.g^{-1} ; positive

end expiratory pressure, 3 cm H₂O; frequency, 20 breaths/min; fractional concentration of inspired oxygen, 0.24), monitored for 6 hours after exposure, and then randomized into treatment groups: CV, PV (A) or (B) (TV, 8 or 6 ml.kg⁻¹; positive end expiratory pressure, 8 cm H₂O; frequency, 20 or 25 breaths/min; fractional concentration of inspired oxygen, 0.4). Pathophysiological parameters were measured for up to 24 hours. The results show that PV resulted in improved oxygenation, decreased shunt fraction, and mortality, with all animals surviving to 24 hours compared to only three of the CV animals. Microscopy confirmed reduced hemorrhage, neutrophilic infiltration, and intra-alveolar edema.

Cholinesterase reactivators: the fate and effects in the organism poisoned with organophosphates/nerve agents

Bajgar J, Kuca K, Jun D, Bartosova L, Fusek J. *Curr Drug Metab* 2007; 8: 803-9.

Abstract: Understanding the mechanism of action of organophosphates (OP)/nerve agents - irreversible acetylcholinesterase (AChE, EC 3.1.1.7) inhibition at the cholinergic synapses followed by metabolic dysbalance of the organism - two therapeutic principles for antidotal treatment are derived. The main drugs are anticholinergics that antagonize the effects of accumulated acetylcholine at the cholinergic synapses and cholinesterase reactivators (oximes) reactivating inhibited AChE. Anticonvulsants such as diazepam are also used to treat convulsions. Though there are experimental data on a good therapeutic effects of reactivators, some attempts to underestimate the role of reactivators as effective antidotes against OP poisoning have been made. Some arguments on the necessity of their administration following OP poisoning are discussed. Their distribution patterns and some metabolic and pharmacological effects are described with the aim to resolve the question on their effective use, possible repeated administration in the treatment of OP poisoning, their peripheral and central effects including questions on their penetration through the blood brain barrier as well as a possibility to achieve their effective concentration for AChE reactivation in the brain. Reactivation of cholinesterases in the peripheral and central nervous system is described and it is underlined its importance for the survival or death of the organism poisoned with OP. Metabolization and some other effects of oximes (not connected with AChE reactivation) are discussed (e.g. forming of the phosphorylated oxime, parasympatholytic action, hepatotoxicity, behavioral changes etc.). An universality of oximes able to reactivate AChE inhibited by all OP is questioned and therefore, needs of development of new oximes is underlined.

Effects of occupational exposure in pesticide plant on workers' serum and erythrocyte cholinesterase activity

Joshaghani HR, Ahmadi AR, Mansourian AR. *Int J Occup Med Environ Health* 2007; 20: 381-5.

Objectives: The determination of cholinesterase activity has been commonly applied in the biomonitoring of exposure to organophosphates and carbamates and in the diagnosis of poisoning with anticholinesterase compounds. One of the groups who are at risk of pesticide intoxication are the workers engaged in the production of these chemicals.

Aims: The aim of this study was to assess the effect of pesticides on erythrocyte and serum cholinesterase activity in workers occupationally exposed to these chemicals.

Methods: The subjects were 63 workers at a pesticide plant. Blood samples were collected before they were employed (phase I) and after 3 months of working in the plant (phase II). Cholinesterase level in erythrocytes (EChE) was determined using the modified Ellman method, and serum cholinesterase (SChE) by butyrylthiocholine substrate assay.

Results: The mean EChE levels were 48±11 IU/g Hb in phase I and 37±17 IU/g Hb in phase II (paired t-test, mean = -29; 95% CI = -43-14), p < 0.001). The mean SChE level was 9569 ±

2496 IU/l in phase I, and 7970 ± 2067 IU/l in phase II (paired t-test, mean = 1599; 95% CI = 1140-2058, $p < 0.001$). There was a significant increase in ALT level ($p < 0.001$) and a decrease in serum albumin level ($p < 0.001$).

Conclusion: In view of the significant decrease in EChE and SChE levels among pesticide workers, it seems that routine assessment of cholinesterase level in workers employed in such occupations and people handling pesticides should be made obligatory.

The epidemiology, evaluation, and management of stingray injuries

Diaz JH. J La State Med Soc 2007; 159: 198-204,quiz 204,230.

Abstract: A descriptive analysis and review of the world's salient scientific literature on stingray injuries was conducted in light of recent high-profile cases of fatal and near-fatal thoracic stingray injuries to guide clinicians in evaluating and managing stingray injuries. Data was extracted from observational and longitudinal studies over the period, 1950-2006, to permit (1) a stratification of stingray injuries as bites, penetrating lacerations with and without envenoming, and combinations of deeply penetrating and envenoming wounds; and (2) an assessment of new management strategies for thoracoabdominal penetrating trauma and non-healing, necrotic stingray wounds. Unlike their Chondrichthyes classmates, the sharks, stingrays are docile and non-aggressive; and will not attack with their spined tails, unless provoked. Although some occupations are predisposed to stingray injuries, most stingray injuries can be avoided by observing sea floors and adopting simple practices when wading, swimming, diving, or fishing in temperate oceans and some tropical freshwater river systems. All stingray injuries should be managed initially with wound irrigation to dislodge retained spine fragments and envenoming tissues and warm water immersion to inactivate heat-labile toxins.

Systemic antivenom and skin necrosis after green pit viper bites

Chotenimitkhun R, Rojnuckarin P. Clin Toxicol 2008; 46: 122-5.

Introduction. As systemic effects of viper venoms can be neutralized by antivenom, local tissue damage, particularly necrosis of the skin, has become a more significant problem. The goal of this study is to evaluate the effectiveness of antivenom in preventing dermatonecrosis in envenomated patients with severe coagulopathy.

Methods. Retrospective review of medical records of patients who were envenomated by green pit vipers (*Trimeresurus albolabris* or *T. macrops*) following bites on fingers or toes and who came to Chulalongkorn hospital from 1996 to 2006.

Results. 1,886 records of suspected green pit viper bite patients were reviewed: 243 cases fit the inclusion criteria; 1,643 cases were excluded: uncertain diagnosis (931), bites at other sites (508), inadequate follow-up (196), and necrosis on presentation (8 patients). One-third of the 243 study cases (80 patients) received green pit viper F (ab')₂ antivenom an average of 21 hours after envenomation for the treatment of severe coagulopathy. The other 163 study patients were treated symptomatically. After a 3-day follow-up, the percentage of patients who developed gangrene among those who received antivenom (7 of 80 patients) was 8.8% and the percentage of patients who developed gangrene among those who did not receive antivenom (12 of 163 patients) was 7.4 % ($p = 0.9$).

Conclusion. Dermatonecrosis occurs after the systemic administration of antivenom. Earlier administration of antivenom and other treatment modalities should be explored in a prospective study.

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