cardiovascular involvement to be severe in nature, and a frequent cause of death in such cases. In our study[1] of 10 cases we found cardiovascular involvement in 6 out of 10 cases – Acute pulmonary oedema was observed in 2. Interesting ECG findings were seen in 6 cases and in 2 cases ECG changes appeared even before the patient lapsed into shock. The various ECG changes observed were ST-T wave changes (elevation/depression), supra-ventricular tachycardia with conduction defects, atrial fibrillation A-V dissociation and LBBB.

In a subsequent study[2] of 25 patients we observed pulmonary oedema in 2 patients and cardiac arrhythmias in 16 patients. Arrhythmias were variable like chaotic atrial tachycardia (2), atrial fibrillation (4), ventricular tachycardia (1), wandering pacemaker (3), complete heart block (1), bundle branch block (2), and sinus arrest (1). One of our patients developed extensive pericardial rub on the fourth day after ingestion of poison. This rub persisted for seven days. There was however no cardiomegaly or pericardial fluid and the patient recovered completely. Ten of our patients expired. In all 10 cases a common factor was that they had consumed three or more tablets of aluminium phosphide. All these patients were admitted in severe shock and were delirious. Metabolic acidosis was present in all these patients and transaminases were raised. Further, their shock did not respond to therapy and they died within one to six hours after hospitalization.

Unfortunately the treatment of aluminium phosphide is still supportive[3] and includes gastric lavage with 1 in 5000 solution of potassium permanganate and intravenous infusion with dextrose saline. Dopamine hydrochloride and hydrocortisone hemisuccinate is given in every case for treatment of shock. Correction of electrolytes and acid base disturbances is important and prob[illegible] is the sheet-anchor of treatment. We have used calcium gluconate (10 ml 10% IV six hourly) as a membrane stabilising agent and achieved some success. The observation of Singh *et al* [1] regarding magnesium levels in these cases are interesting and require further elucidation.

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REPLY FROM THE AUTHORS

The Editor,

It is clear that Khosla and his colleagues are international experts on aluminium phosphide (ALP) intoxication particularly with reference to cardiovascular manifestations. We agree that ALP poisoning is usually associated with cardiac arrhythmias, raised blood levels of transeminases (SGOT, SGPT) suggestive of acute damage to myocardial and liver cells. There is experimental evidence[1], some of which is substantial, that injury to the myocardial cell is associated with increased efflux of magnesium and potassium and influx of sodium and calcium. While cellular magnesium and potassium have been suggested to maintain the integrity of the myocardial cell, sodium and calcium may be arrhythmogenic[2]. Thus, parenteral administration of saline and calcium gluconate in patients with cardiac cell damage, particularly with cardiac tachyarrhythmias, may be dangerous in patients with ALP intoxication (unless there is deficiency of these cations). We are following the same treatment as advised by Prof. Khosla, except for saline and calcium. There is clinical and experimental evidence that parenteral administration of calcium salts may be associated with increased excretion of magnesium and vice versa. Indeed calcium is a natural antagonist of magnesium in both cardiac and arterial smooth cell[3]. While we are grateful to Prof. Khosla for taking interest in our article, we would suggest restricting the use of calcium only to those patients of ALP poisoning who have associated calcium deficiency. The best results obtained may be due to a bias and the use of calcium in less serious patients. Multicentre, double blind and randomized studies should be organised by Prof. Khosla to confirm the use of cations among these patients, for which I would like to cooperate with him.

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OCULAR ATROPINE INDUCED PSYCHOSIS – IS THERE A DIRECT ACCESS ROUTE TO THE BRAIN?

The Editor,

Anticholinergics like atropine in large toxic doses can lead to restlessness, irritability, hallucinations and de-

lirium. The large doses required for detectable central effects reflect the difficulty of penetration of the drug into the central nervous system. Rarely atropine used in eye drops has selectively produced toxic psychosis without peripheral manifestations like bradycardia. This suggests that the eye drops have direct access to the brain rather than through the nasolacrimal duct, as is traditionally believed. The authors report a case which corroborates previous records of this phenomenon with ocular atropine, and make this novel speculation.

A 10 year old boy was admitted with a history of delirium with hallucinations, and talking and behaving abnormally – all of which started on the day of admission. He had been seen in another hospital a week back and advised atropine eye drops for pain and redness of the eye, with dimness of vision. There was no previous history of psychosis in this child or his family.

On examination he was delirious, talking irrelevantly and not oriented in time or place. His heart rate was 90 per minute. There was no evidence of meningism. His left eye showed evidence of chronic iridocyclitis with a complicated cataract. The iris pattern was completely distorted, there was a dense synechiae and a festooned pupil. There were no keratic precipitates or a flare. The mouth and skin were moist, there was no rash and no difficulty in swallowing or micturition.

A diagnosis of chronic iridocyclitis with atropine psychosis was made and the child was switched over to phenylephrine drops.

Within 48 hours of stopping atropine the child's behaviour had reverted to normal without antipsychotic medication. His synechiae between 9 o'clock and 12 o'clok position had also broken. He was discharged after 3 days and has had no further psychotic episodes on follow up.

Systemic absorption of atropine eye drops is postulated to occur either from the nasal mucosa after the drug has transversed the nasolacrimal duct, or from the intestinal tract if it is swallowed. Systemic effects of atropine overdose like tachycardia and dryness of the skin occur at much lower doses than the central anticholinergic actions, and this is a reflection of the difficulty the drug has in reaching the brain. However delirium has been reported with relatively small doses of atropine instilled into the eye[1,2]. Central anticholinergic action in the absence of undue peripheral manifestations, as in our case, has been reported previously in adults[3]. It is as if atropine eye drops, in susceptible people, have direct access to the brain, so that small doses can be concentrated there to produce the central anticholinergic action, without systemic effects. We feel that the present concept of systemic absorption via the nasolacrimal duct, from the nasal and intestinal mucosa is inadequate to explain such a central action of atropine eye drops without peripheral manifestations.

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AYURVEDIC DRUGS – ARE THEY REALLY SAFE?

The Editor,

Many ayurvedic drugs are popularised as innocent, safe and effective medicines and are available over the counter without prescriptions. Many of these, at times, prove fatal.

BPT a 25 year old male, was admitted with palpitations, abdominal pain, vomiting, paraesthesiae in all limbs following ingestion of 10 tablets of 'Shankhvati' for abdominal pain 2 hours earlier. The patient was conscious, with irregularly irregular pulse (170/min.), BP 120/80 mm of Hg, without signs of CCF. Systemic examination was normal except for irregularly-irregular heart sounds. Patient had no past history of cardiac disease. ECG showed supraventricular tachycardia with aberrancy with nodal escape rhythm. 100 mgms of xylocaine bolus was given and there was immediate conversion to sinus rhythm. ECG taken at intervals of 24, 48 and 72 hours was normal. The patient did not require any other drug therapy.

'Shankhvati' used for abdominal pain of varied aetiology is an ayurvedic medicine containing mercury, sulphur, 'Vachhnaag' (aconite) and many other trace elements. 'Vachhnaag' (aconite) is a known cardiotoxic and neurotoxic compound. It can produce arrhythmias like VPCs, ventricular tachycardia, ventricular fibrillation, nodal rhythm, bundle branch block, and supraventricular tachycardia.[1] Symptoms may commence immediately or within few minutes of ingestion of a poisonous dose, but the fatal period is usually from 1 to 5 hours. It can be delayed upto 24 hours.[1] The common cause of death is VF and respiratory failure.[1] The fatal dose of aconite is 1 to 2 gms of aconite roots.[1]

Gohel *et al*[2] reported one patient of 'Vachhnaag' (aconite) poisoning following accidental ingestion of lotion used for poliomyelitis.