Systematic review of clinical adverse events reported after acute intravenous lipid emulsion administration

Background
Intravenous lipid emulsions (ILEs) were initially developed to provide parenteral nutrition. In recent years, ILE has emerged as a treatment for poisoning by local anesthetics and various other drugs. The dosing regimen for the clinical toxicology indications differs significantly from those used for parenteral nutrition. The evidence on the efficacy of ILE to reverse acute toxicity of diverse substances consists mainly of case reports and animal experiments. Adverse events to ILE are important to consider when clinicians need to make a risk/benefit analysis for this therapy.

Methods
Multiple publication databases were searched to identify reports of adverse effects associated with acute ILE administration for either treatment of acute poisoning or parenteral nutrition. Articles were selected based on pre-defined criteria to reflect acute
use of ILE. Experimental studies and reports of adverse effects as a complication of long-term therapy exceeding 14 days were excluded.

**Results**

The search identified 789 full-text articles, of which 114 met the study criteria. 27 were animal studies, and 87 were human studies. The adverse effects associated with acute ILE administration included acute kidney injury, cardiac arrest, ventilation perfusion mismatch, acute lung injury, venous thromboembolism, hypersensitivity, fat embolism, fat overload syndrome, pancreatitis, extracorporeal circulation machine circuit obstruction, allergic reaction, and increased susceptibility to infection.

**Conclusion**

The emerging use of ILE administration in clinical toxicology warrants careful attention to its potential adverse effects. The dosing regimen and context of administration leading to the adverse events documented in this review are not generalizable to all clinical toxicology scenarios. Adverse effects seem to be proportional to the rate of infusion as well as total dose received. Further safety studies in humans and reporting of adverse events associated with ILE administration at the doses advocated in current clinical toxicology literature are needed.

Full text available from: [http://dx.doi.org/10.3109/15563650.2016.1151528](http://dx.doi.org/10.3109/15563650.2016.1151528)

**Use of intravenous fat emulsion in the emergency department for the critically ill poisoned patient**


Abstract and full text available from: [http://dx.doi.org/10.1016/j.jemermed.2016.02.008](http://dx.doi.org/10.1016/j.jemermed.2016.02.008)

**Antipsychotic-related fatal poisoning, England and Wales, 1993–2013: impact of the withdrawal of thioridazine**


**Context**

Use of second generation antipsychotics in England and Wales has increased in recent years whilst prescription of first generation antipsychotics has decreased.

**Methods**

To evaluate the impact of this change and of the withdrawal of thioridazine in 2000 on antipsychotic-related fatal poisoning, we reviewed all such deaths in England and Wales 1993–2013 recorded on the Office for National Statistics drug poisoning deaths database. We also reviewed antipsychotic prescribing in the community, England and Wales, 2001–2013.

**Use of routine mortality data**

When an antipsychotic was recorded with other drug(s), the death certificate does not normally say if the antipsychotic caused the death rather than the other substance(s). A second consideration concerns intent. A record of "undetermined intent" is likely to have been intentional self-poisoning, the evidence being insufficient to be certain that the individual intended to kill. A record of drug abuse/dependence, on the other hand, is likely to have been associated with an unintentional death.

**Accuracy of the diagnosis of poisoning**

When investigating a death in someone prescribed antipsychotics, toxicological analysis of
biological samples collected post-mortem is usually performed. However, prolonged attempts at resuscitation, or diffusion from tissues into blood as autolysis proceeds, may serve to alter the composition of blood sampled after death from that circulating at death. With chlorpromazine and with olanzapine a further factor is that these compounds are notoriously unstable in post-mortem blood.

**Deaths from antipsychotics**

There were 1544 antipsychotic-related poisoning deaths. Deaths in males (N = 948) were almost twice those in females. For most antipsychotics, the proportion of deaths in which a specific antipsychotic featured either alone, or only with alcohol was 30–40%, but for clozapine (193 deaths) such mentions totalled 66%. For clozapine, the proportion of deaths attributed to either intentional self-harm, or undetermined intent was 44%, but for all other drugs except haloperidol (20 deaths) the proportion was 56% or more. The annual number of antipsychotic-related deaths increased from some 55 per year (1.0 per million population) between 1993 and 1998 to 74 (1.5 per million population) in 2000, and then after falling slightly in 2002 increased steadily to reach 109 (1.9 per million population) in 2013.

**Intent**

The annual number of intentional and unascertained intent poisoning deaths remained relatively constant throughout the study period (1993: 35 deaths, 2013: 38 deaths) hence the increase in antipsychotic-related deaths since 2002 was almost entirely in unintentional poisoning involving second generation antipsychotics. Clozapine, olanzapine, and quetiapine were the second generation antipsychotics mentioned most frequently in unintentional poisonings (99, 136, and 99 deaths, respectively). Mentions of diamorphine/morphine and methadone (67 and 99 deaths, respectively) together with an antipsychotic were mainly (84 and 90%, respectively) in either unintentional or drug abuse-related deaths.

**Deaths and community prescriptions**

Deaths involving antipsychotics (10 or more deaths) were in the range 11.3–17.1 deaths per million community prescriptions in England and Wales, 2001–2013. Almost all (96%) such deaths now involve second generation antipsychotics. This is keeping with the increase in annual numbers of prescriptions of these drugs overall (<1 million in 2000, 7 million in 2013), largely driven by increases in prescriptions for olanzapine and quetiapine. In contrast, deaths involving thioridazine declined markedly (from 40 in 2000 to 10 in 2003–2013) in line with the fall in prescriptions for thioridazine from 2001.

**Conclusions**

The removal of thioridazine has had no apparent effect on the incidence of antipsychotic-related fatal poisoning in England and Wales. That such deaths have increased steadily since 2001 is in large part attributable to an increase in unintentional deaths related to (i) clozapine, and (ii) co-exposure to opioids, principally diamorphine and methadone.

Full text available from: http://dx.doi.org/10.3109/15563650.2016.1164861

**Missed opportunities?: an evaluation of potentially preventable poisoning deaths**


**Introduction**

Although most poisoning deaths are not preventable with current medical technology, in some cases different management decisions may have prevented fatal outcomes.
**Objective**
This study aims to review reported poisoning-related deaths for preventability to provide insight to improve future care.

**Methods**
Fatality abstracts published in the US National Poison Data System (NPDS) Annual Reports (2008–2012) were analyzed. Preventability was graded using a Likert scale of 1 (definitely non-preventable) to 6 (definitely preventable). Two medical toxicologists screened all cases. Cases deemed definitely not preventable (score 1) by both reviewers were excluded from further review and considered to be "non-preventable". All cases considered at least possibly preventable by either screener were reviewed by a multidisciplinary panel of 5 physicians for preventability scoring. Differences were resolved by consensus. Cases determined to be "preventable" (scores 4–6) were characterized by type of improvement issue involved (diagnosis, treatment, monitoring, other) and recurring scenarios.

**Results**
Of 390 published abstracts, 78 (20.0%) deaths were considered at least possibly preventable by at least one screener. Of these, 34 (8.7%) deaths were determined to be "preventable" by the panel. Inter-observer agreement by weighted kappa analysis was 0.58 for screening, 0.24 for preventability, and 0.44 for specific aspects of care. The most common toxicants were salicylates (n = 9), opioids (n = 4), toxic alcohols (n = 3), fluoride containing product (n = 3), and bupropion (n = 3). The most common improvement opportunities involved treatment and monitoring.

**Discussion**
Most of the ingested substances in preventable deaths have delayed GI absorption or require metabolic activation to produce a delayed effect (such as salicylates, opioids, and toxic alcohols), and therefore provide an opportunity for early recognition and successful interventions. Most improvement opportunities are clearly described in the literature but may be not recognized.

**Conclusions**
Based on an analysis of published NPDS data, a considerable number of poisoning-related deaths reaching medical attention may be preventable. The most common scenarios involved in potentially preventable poisoning fatalities related to monitoring and treatment. Salicylates and opioids were the most common agents involved in preventable deaths.

Full text available from: [http://dx.doi.org/10.3109/15563650.2016.1157721](http://dx.doi.org/10.3109/15563650.2016.1157721)

**Could chest wall rigidity be a factor in rapid death from illicit fentanyl abuse?**

**Background**
There has been a significant spike in fentanyl-related deaths from illicit fentanyl supplied via the heroin trade. Past fentanyl access was primarily oral or dermal via prescription fentanyl patch diversion. One factor potentially driving this increase in fatalities is the change in route of administration. Rapid intravenous (IV) fentanyl can produce chest wall rigidity. We evaluated post-mortem fentanyl and norfentanyl concentrations in a recent surge of lethal fentanyl intoxications.

**Methods**
Fentanyl related deaths from the Franklin County coroner’s office from January to
September 2015 were identified. Presumptive positive fentanyl results were confirmed by quantitative analysis using liquid chromatography tandem mass spectrometry (LC/MS/MS) and were able to quantify fentanyl, norfentanyl, alfentanyl, and sufentanyl.

Results
48 fentanyl deaths were identified. Mean fentanyl concentrations were 12.5 ng/ml, (range 0.5 ng/ml to >40 ng/ml). Mean norfentanyl concentrations were 1.9 ng/ml (range none detected to 8.3 ng/ml). No appreciable concentrations of norfentanyl could be detected in 20 of 48 cases (42%) and were less than 1 ng/ml in 25 cases (52%). Elevated fentanyl concentrations did not correlate with rises in norfentanyl levels. In several cases fentanyl concentrations were strikingly high (22 ng/ml and 20 ng/ml) with no norfentanyl detected.

Discussion
The lack of any measurable norfentanyl in half of our cases suggests a very rapid death, consistent with acute chest rigidity. An alternate explanation could be a dose-related rapid onset of respiratory arrest. Deaths occurred with low levels of fentanyl in the therapeutic range (1-2 ng/ml) in apparent non-naïve opiate abusers. Acute chest wall rigidity is a well-recognized complication in the medical community but unknown within the drug abuse community. The average abuser of illicit opioids may be unaware of the increasing fentanyl content of their illicit opioid purchase.

Conclusion
In summary we believe sudden onset chest wall rigidity may be a significant and previously unreported factor leading to an increased mortality, from illicit IV fentanyl use. Fentanyl and norfentanyl ratios and concentrations suggest a more rapid onset of death given the finding of fentanyl without norfentanyl in many of the fatalities. Chest wall rigidity may help explain the cause of death in these instances, in contrast to the typical opioid-related overdose deaths. Intravenous heroin users should be educated regarding this potentially fatal complication given the increasingly common substitution and combination with heroin of fentanyl.

Full text available from: http://dx.doi.org/10.3109/15563650.2016.1157722

Diglycolic acid, the toxic metabolite of diethylene glycol, chelates calcium and produces renal mitochondrial dysfunction in vitro

Context
Diethylene glycol (DEG) has caused many cases of acute kidney injury and deaths worldwide. Diglycolic acid (DGA) is the metabolite responsible for the renal toxicity, but its toxic mechanism remains unclear.

Objective
To characterize the mitochondrial dysfunction produced from DGA by examining several mitochondrial processes potentially contributing to renal cell toxicity.

Materials and methods
The effect of DGA on mitochondrial membrane potential was examined in normal human proximal tubule (HPT) cells. Isolated rat kidney mitochondria were used to assess the effects of DGA on mitochondrial function, including respiratory parameters (States 3 and 4), electron transport chain complex activities and calcium-induced opening of the mitochondrial permeability transition pore. DGA was compared with ethylene glycol tetraacetic acid (EGTA) to determine calcium chelating ability. DGA cytotoxicity was assessed using lactate dehydrogenase leakage from cultured proximal tubule cells.
**Results**

DGA decreased the mitochondrial membrane potential in HPT cells. In rat kidney mitochondria, DGA decreased State 3 respiration, but did not affect State 4 respiration or the ADP/O ratio. DGA reduced glutamate/malate respiration at lower DGA concentrations (0.5 mmol/L) than succinate respiration (100 mmol/L). DGA inhibited Complex II activity without altering Complex I, III or IV activities. DGA blocked calcium-induced mitochondrial swelling, indicating inhibition of the calcium-dependent mitochondrial permeability transition. DGA and EGTA reduced the free calcium concentration in solution in an equimolar manner. DGA toxicity and mitochondrial dysfunction occurred as similar concentrations.

**Discussion**

DGA inhibited mitochondrial respiration, but without uncoupling oxidative phosphorylation. The more potent effect of DGA on glutamate/malate respiration and the inhibition of mitochondrial swelling was likely due to its chelation of calcium.

**Conclusion**

These results indicate that DGA produces mitochondrial dysfunction by chelating calcium to decrease the availability of substrates and of reducing equivalents to access Complex I and by inhibiting Complex II activity at higher concentrations.

Full text available from: [http://dx.doi.org/10.3109/15563650.2016.1162312](http://dx.doi.org/10.3109/15563650.2016.1162312)

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**Incidence and patterns of cardiomyopathy in carbon monoxide-poisoned patients with myocardial injury**


**Objectives**

Sustained myocardial injury is a significant predictor of mortality in carbon monoxide (CO) poisoning. There are few reports in the literature regarding the presence of CO-induced cardiomyopathy from early stages in the emergency department (ED). We prospectively investigated the early incidence of CO-induced cardiomyopathy and its patterns in patients with cardiomyopathy.

**Materials and methods**

During a 10-month period, transthoracic echocardiography (TTE) was performed in 43 consecutive patients with CO poisoning and myocardial injury, which was defined as elevated high-sensitive troponin I within 24 h after ED arrival. Measurements of left ventricular ejection fraction and wall motion abnormalities were performed to evaluate cardiac function. If a patient had CO-induced cardiomyopathy, we measured cardiac function at the time of patient admission, day 1, day 2, and once within seven days of hospitalization.

**Results**

The incidence of cardiomyopathy was as high as 74.4% (32 of 43 patients) in CO-poisoned patients with myocardial injury based on initial ED results. Echocardiographic patterns included non-cardiomyopathy (25.6%), global dysfunction (51.2%), and Takotsubo-like cardiomyopathy (23.2%). Patients in the global dysfunction group had significantly more normalized cardiac dysfunction within 72 h than did those in the Takotsubo-like cardiomyopathy group (81.8% vs. 22.2%, $p = 0.001$).

**Discussion and conclusion**

Patients with CO poisoning and myocardial injury experienced cardiomyopathy, including reversible global dysfunction and a Takotsubo-like pattern. Investigation of cardiomyopathy
needs to be considered in patients with CO poisoning and myocardial injury.

Full text available from: [http://dx.doi.org/10.3109/15563650.2016.1162310](http://dx.doi.org/10.3109/15563650.2016.1162310)

**Management of cocaine-induced myocardial infarction: 4–year experience at an urban medical center**


Abstract and full text available from: [http://dx.doi.org/10.14423/SMJ.0000000000000430](http://dx.doi.org/10.14423/SMJ.0000000000000430)

**Acute animal and human poisonings from cyanotoxin exposure – A review of the literature**

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Abstract and full text available from: [http://dx.doi.org/10.1016/j.mpmed.2015.12.004](http://dx.doi.org/10.1016/j.mpmed.2015.12.004)

**Drugs of abuse**


Abstract and full text available from: [http://dx.doi.org/10.1016/j.mpmed.2015.12.030](http://dx.doi.org/10.1016/j.mpmed.2015.12.030)

**Corrosives**


Abstract and full text available from: [http://dx.doi.org/10.1016/j.mpmed.2015.12.007](http://dx.doi.org/10.1016/j.mpmed.2015.12.007)

**Plants**


Abstract and full text available from: [http://dx.doi.org/10.1016/j.mpmed.2015.11.017](http://dx.doi.org/10.1016/j.mpmed.2015.11.017)

**Metabolic effects of poisoning**


Abstract and full text available from: [http://dx.doi.org/10.1016/j.mpmed.2015.11.013](http://dx.doi.org/10.1016/j.mpmed.2015.11.013)

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Cyanide.  

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Gases
Meulenbelt J.  
Irritant gases.  

Helium

**Household products**


**Hydrazine**


**Hydrogen sulphide**


**Methanol**


**Methyl tertiary butyl ether**


**Nanoparticles**


**Oxygen**


**Paraphenylenediamine**


**Perfluorinated compounds**


**Petrol (gasoline)**


**Phosphine**


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General
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**Silicon**


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**General**


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General

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Jellyfish


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Hymenoptera

Microorganisms
Algae

Cyanobacteria

Scorpions

Snake bites


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### INDEX

<table>
<thead>
<tr>
<th>Compound</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abrus Precatorius</td>
<td>38</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>28</td>
</tr>
<tr>
<td>Acetone</td>
<td>30</td>
</tr>
<tr>
<td>Acetylcysteine</td>
<td>18</td>
</tr>
<tr>
<td>Air pollution</td>
<td>30</td>
</tr>
<tr>
<td>Alcohol</td>
<td>30</td>
</tr>
<tr>
<td>Algae</td>
<td>39</td>
</tr>
<tr>
<td>Alkyl Nitrites</td>
<td>21</td>
</tr>
<tr>
<td>Aloe vera</td>
<td>38</td>
</tr>
<tr>
<td>Alpha-chloralose</td>
<td>37</td>
</tr>
<tr>
<td>Aluminium</td>
<td>35</td>
</tr>
<tr>
<td>Amfetamines</td>
<td>21</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>22</td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>29</td>
</tr>
<tr>
<td>Anoxicillin</td>
<td>22</td>
</tr>
<tr>
<td>Anaesthetics</td>
<td>22</td>
</tr>
<tr>
<td>Analytical toxicology</td>
<td>8</td>
</tr>
<tr>
<td>Animals, general</td>
<td>39</td>
</tr>
<tr>
<td>Anisomorpha buprestoides</td>
<td>39</td>
</tr>
<tr>
<td>Antiarrhythmic drugs</td>
<td>22</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>22</td>
</tr>
<tr>
<td>Antibodies</td>
<td>20</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>22</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>22</td>
</tr>
<tr>
<td>Antidepressants</td>
<td>23</td>
</tr>
<tr>
<td>Antidotes</td>
<td>18</td>
</tr>
<tr>
<td>Antihistamines</td>
<td>23</td>
</tr>
<tr>
<td>Antimalarial Drugs</td>
<td>23</td>
</tr>
<tr>
<td>Antineoplastic Drugs</td>
<td>23</td>
</tr>
<tr>
<td>Antipsychotics</td>
<td>23</td>
</tr>
<tr>
<td>Antitussiculcurs Drugs</td>
<td>22</td>
</tr>
<tr>
<td>Antivenom</td>
<td>19</td>
</tr>
<tr>
<td>Antiviral Drugs</td>
<td>23</td>
</tr>
<tr>
<td>Arsenic</td>
<td>35</td>
</tr>
<tr>
<td>Asbestos</td>
<td>31</td>
</tr>
<tr>
<td>Azacitidine</td>
<td>23</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>26</td>
</tr>
<tr>
<td>Benzene</td>
<td>31</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>23</td>
</tr>
<tr>
<td>Beta2 Agonists</td>
<td>24</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>20</td>
</tr>
<tr>
<td>Beta-Blockers</td>
<td>24</td>
</tr>
<tr>
<td>Biological warfare</td>
<td>38</td>
</tr>
<tr>
<td>Biomarkers</td>
<td>8</td>
</tr>
<tr>
<td>Bisphenol A</td>
<td>31</td>
</tr>
<tr>
<td>Bispyridinium-non-oxide-compounds</td>
<td>19</td>
</tr>
<tr>
<td>Bleomycin</td>
<td>23</td>
</tr>
<tr>
<td>Body packers</td>
<td>9</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>20</td>
</tr>
<tr>
<td>Cadmium</td>
<td>35</td>
</tr>
<tr>
<td>Caffeine</td>
<td>24</td>
</tr>
<tr>
<td>Calcinol</td>
<td>29</td>
</tr>
<tr>
<td>Calcium Channel Blockers</td>
<td>24</td>
</tr>
<tr>
<td>Cannabis</td>
<td>24</td>
</tr>
<tr>
<td>Capsaicin</td>
<td>31</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>22</td>
</tr>
<tr>
<td>Carbon</td>
<td>31</td>
</tr>
<tr>
<td>Carbon black</td>
<td>31</td>
</tr>
<tr>
<td>Carbon monoxide</td>
<td>31</td>
</tr>
<tr>
<td>Carbon tetrachloride</td>
<td>31</td>
</tr>
<tr>
<td>Carcinogenicity</td>
<td>9</td>
</tr>
<tr>
<td>Cardiototoxicity</td>
<td>9</td>
</tr>
<tr>
<td>Cement</td>
<td>31</td>
</tr>
<tr>
<td>Chemical warfare, general</td>
<td>38</td>
</tr>
<tr>
<td>Chemicals, general</td>
<td>30</td>
</tr>
<tr>
<td>Chlorine</td>
<td>31</td>
</tr>
<tr>
<td>Chloroquine</td>
<td>24</td>
</tr>
<tr>
<td>Cholinesterase Inhibitors</td>
<td>24</td>
</tr>
<tr>
<td>Ciclosporin</td>
<td>26</td>
</tr>
<tr>
<td>Cisplatin</td>
<td>23</td>
</tr>
</tbody>
</table>
Propylene glycol ................................................................. 34
Prothrombin complex concentrate .................................... 20
Proton Pump Inhibitors ...................................................... 28
Psychiatric aspects .......................................................... 17
Pyrethroid insecticides, general ......................................... 37
Quaternary ammonium ...................................................... 34
Radiation ........................................................................... 34
Reprotoxicity ...................................................................... 17
Ricin .................................................................................... 38
Risk assessment ................................................................. 17
Risperidone ......................................................................... 23
Rodenticides ........................................................................ 37
Salicylates ........................................................................... 29
Sarin ..................................................................................... 8
Scorpions ............................................................................. 39
Silica ..................................................................................... 34
Silicon ................................................................................... 36
Simeprevir ............................................................................ 23
Snake bites .......................................................................... 39
Sodium hydroxide ............................................................. 34
Sodium oxybate ................................................................. 20
Sodium polystyrene sulfonate ............................................. 20
Solvents ............................................................................... 34
St. John's wort ..................................................................... 39
Substance abuse ............................................................... 29
Suicide ............................................................................... 18
Superwarfarin ..................................................................... 37
Synthetic Cannabinoids .................................................... 25
Synthetic Cathinones ........................................................ 25
Tebuconazole ...................................................................... 37
Thallium .............................................................................. 36
Theophylline ....................................................................... 29
Titanium .............................................................................. 36
Toluene ................................................................................. 34
Toxicology, general ............................................................ 8
Tranexamic Acid .................................................................. 29
Tricyclic Antidepressants .................................................. 29
True vipers ......................................................................... 40
Tryptamines ......................................................................... 29
Twostriped walkingstick ..................................................... 39
Vincristine ........................................................................... 23
Viperinae ............................................................................ 40
Vitamins ............................................................................... 29
VX ....................................................................................... 38
Warfarin .............................................................................. 22
Water pollution ................................................................. 30
Wood dusts ......................................................................... 34
Wood smoke ....................................................................... 34

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