

# TOXICOLOGY TODAY



## BUTTON BATTERY INGESTION

© Matteo69 | Dreamstime.com



by Alisha Shelton,  
PharmD Candidate 2012



Endoscopic view of button battery in stomach

TABLE 1<sup>2</sup>

Letter imprint	Cathode	Anode
L or LR	Manganese dioxide	Zinc
S or SR	Silver oxide	Zinc
P	Oxygen	Zinc
C or CR	Manganese dioxide	Lithium
B or BR	Carbon monofluoride	Lithium
G	Copper oxide	Lithium
M,N*	Mercuric oxide	Zinc

\*The sale of mercuric oxide batteries is prohibited under federal law<sup>3</sup>. Adapted from reference 2

### Introduction

Button batteries are small cell-type, disc-shaped batteries that differ in size, chemical composition, and voltage. They contain an anode and cathode. The anode is generally made of zinc or lithium; whereas the cathode is made of a plethora of chemicals (Table 1). Each disc battery has an imprint with one to two letters, and three to four numbers. The International Electrotechnical Commission<sup>2</sup> sets each imprint to help determine the difference in each battery.

Button batteries differ in diameter and thickness. The imprint code can be found on the battery package and on the battery itself. The letter

“R” is used to designate a round battery. The size of the battery can be important in evaluating the risk of the ingestion. Harmful outcomes are more common with ingestion of batteries with a diameter greater than or equal to 20 millimeters; approximately the same size as an American quarter. The nomenclature for lithium manganese dioxide batteries notes the diameter first in mm followed by the thickness. Diameter generally ranges from six to 25 mm and thickness from 12 to 77 mm. For example CR2032 has a diameter of 20 mm and a thickness of 3.2 mm.

### National Battery Ingestion Hotline

The National Bat-

tery Ingestion Hotline (NBIH) at the National Capital Poison Center in Washington, DC, was established in 1982. The NBIH phone number is 202-625-3333. It is open 24 hours a day, seven days a week with trained professionals available to answer questions about button battery ingestions. The NBIH collects button batteries after ingestion to study decomposition.<sup>3</sup>

### Epidemiology

US Poison Control Centers through the National Poison Data System (NPDS) reported 56,535 button battery ingestions from 1985 to 2009, of which 68% occurred in children less than 6 years of age.<sup>1</sup> While there has been

## IN THIS ISSUE

### Button Battery Ingestion

### Outreach Education: CDC CO Campaign

### Poison Pearls:

- Salicylate Toxicity
- CO Toxicity and Hyperbaric Oxygen Therapy

### Meet the UPCC Staff:

- Tom Davies

no demonstrated trend in reports of battery ingestions to US poison control centers, the proportion of cases resulting in a major or fatal outcome has risen nearly 7-fold over the last three years.<sup>1</sup> A recent study published in *Pediatrics* reviewed available data from NPDS, NBIH and the literature to identify outcome predictors and trends. A total of 13 fatalities have been reported from 1977-2011 from battery ingestion, all in children under the age of three years.<sup>1</sup> In all cases the batteries were lodged in the esophagus 10 hours to 2 weeks before removal or death. Death most frequently occurred as a result of blood loss secondary to development of a fistula into a major artery, especially aorto-esophageal fistulas.

A major (life-threaten-

ing) outcome occurred in 73 cases, 92% in children younger than 4 years and all occurring in children under the age of 10 years. In 41 (56%) patients the ingestion was not witnessed resulting in a delayed or missed diagnosis in 19 as a result of the non-specific symptoms and misidentification on radiograph. Severe esophageal burns and esophageal stenosis occurred in as little as 2.5 hours in three cases. Removal of the battery occurred within 24 hours in 50% of cases that resulted in a major outcome. Other significant clinical effects included tracheoesophageal fistulas, other esophageal perforations, esophageal strictures and stenosis, vocal cord paralysis, mediastinitis, cardiac or respiratory arrests, pneumothorax, (cont. on pg. 2)

## CDC LAUNCHES NEW AWARENESS CAMPAIGN

Each year, more than 400 people die in the US from accidental carbon monoxide (CO) poisoning. CO is found in combustion fumes, such as those produced by small gasoline engines, stoves, generators, lanterns, and gas ranges; or by burning charcoal and wood.

CO poisoning is increasingly recognized as a public health concern in the wake of large-scale disasters such as hurricanes, floods, and ice storms. Yet it is almost entirely preventable. With this increased awareness, the CDC has focused on surveillance, research, education, and partnerships as means to reducing mortality and morbidity from CO poisoning.

Using what was learned from its research activities and field investigations, CDC has developed an education strategy focusing on 3 key activities:

- A diagnostic tool for health professionals to assist in identifying CO poisoning in a clinical setting



- Maintenance of CDC's CO web site, including CDC-TV features, print materials, public service announcements available for download, and CO poisoning prevention guidance in 17 languages
- Audience profiling for a proposed national communication effort to raise awareness of CO poisoning risks and preventive behavior

The campaign has been launched and all tools are available on the newly designed web-site: [www.cdc.gov/co](http://www.cdc.gov/co). Links to the research, surveillance, clinical guidance, prevention education, and partners are on the main website.

### Tips to prevent CO poisoning:

- Have your heating system, water heater and other fuel burning appliances checked annually
- Install a battery-operated CO detector and check or replace the batteries twice a year
- Seek medical attention if you suspect CO poisoning and feel dizzy, light-headed, or nauseous – call the poison center at 1-800-222-1222
- Don't use a generator or other fuel burning device in or near the home or garage
- Don't run a car or truck inside a garage
- Don't heat your home with a gas oven

### (cont. from pg. 1) **Battery**

pneumoperitoneum, tracheal stenosis, tracheomalacia, aspiration pneumonia, empyema, lung abscess, and spondylodiscitis. Batteries with a diameter of 20-25 mm pose the greatest risk of a severe or fatal outcome (odds ratio 24.6). During the same time period that worsened outcomes were noted there was an increase in the use of lithium 20 mm button cells along with an increase in the number of ingestions of lithium button cells. Of note, in 64% of ingestions reported to the NBIH where interval of passage through the GI tract was known, the battery passed in the stool within 72 hours of ingestion.

### **Mechanism of Toxicity**

Traditionally, batteries have been thought to cause tissue injury by three primary mechanisms: generation of electrical current; leakage of battery contents and pressure necrosis. Current information suggests that the greatest damage comes from generation of hydroxide from the negative pole resulting in tissue damage. Injury can continue after removal of the battery as a result of either residual alkali or tissue weakness. Damage can occur within as little as 2 hours after ingestion of a battery that lodges in the esophagus. Lithium cells

can generate hydroxide more rapidly than other button cells, which may be the reason for the worsening outcomes with lithium button cells.

Some batteries contain heavy metals such as mercury or other metals such as lithium.<sup>4</sup> There are case reports of measurable mercury and lithium concentrations following battery ingestion but not symptomatic poisoning.<sup>5</sup> The damage generally occurs on the negative pole of the battery. Batteries that do not work to power a device still have an electrical charge that can harm the tissue in the esophagus.<sup>1</sup> It is important to get an accurate ingestion history of the patient as co-ingestion with a magnet can cause perforation and necrosis in the gut.<sup>6</sup>

### **Treatment**

Battery ingestion requires immediate attention. A radiograph should be obtained in children 12 years of age or younger or in anyone who has ingested a battery > 12 mm in diameter. An attempt should be made to identify the battery imprint. Children should remain NPO until esophageal position of the battery can be ruled out in case the patient may need to be anesthetized. If battery is found in the esophagus, immediate removal is indicated. Other criteria for removal include: patient

also ingested a magnet, or the battery is > 20 millimeters, or the patient is symptomatic. Otherwise, if the patient is asymptomatic the patient can be sent home to watch for passage of the battery in the stool. Another X-ray should be performed four days post ingestion if the battery has not passed yet. If the patient is > 12 years and battery is ≤ 12 millimeters and the patient is asymptomatic, only swallowed one battery, did not also co-ingest a magnet, has no pre-existing esophageal disease, and the patient or caregiver agrees to promptly seek evaluation if symptoms develop, the patient can be treated at home to watch for battery to pass in stool. If 14 days have passed and no battery has been excreted, an X-ray may need to be performed to check for passage.

### **Prevention**

Adults put batteries in their mouths for numerous reasons including checking charge on the battery and mistaking the battery as medication. Children like to put all sorts of objects into their mouths, including batteries. It is important to store and dispose of batteries out of reach of small children. Do not put used batteries on the counter – as it might be mistaken as medication or candy. Encourage use of electronics that require (cont. on pg. 4)

**SALICYLATE TOXICITY**

by Andrew Dorias, MD  
Emergency Medicine Resident

The salicylate poisoned patient may be tachypneic, lethargic, comatose, seizing, hyperthermic, or in cardiopulmonary arrest. Arterial blood gas will likely reveal a mixed respiratory alkalosis and metabolic acidosis. Providers should have a low threshold to check a salicylate level on a patient that presents with any of the above signs or symptoms. A therapeutic salicylate concentration is 15-30 mg/dL. Salicylates have a narrow therapeutic window and toxicity can occur close to the upper limit of the therapeutic range. More than one salicylate level a few hours apart is required as delayed absorption can occur and a false sense of security is possible with an initially low blood concentration.

Once the diagnosis of salicylate toxicity is made initiate therapy with IV hydration and sodium bicarbonate. Three ampoules of sodium bicarbonate in one liter of D5 W at 200 cc/hr is a typical starting point for an adult. Patients who are acidotic should receive a bicarbonate bolus of 1-2 mEq/kg. Alkalinizing the blood (pH 7.5) and urine (pH 8.0-9.0) traps the ionized form of the drug in these compartments. It is difficult to alkalinize the urine if a patient is hypokalemic as the kidneys will excrete hydrogen ions into the urine in an effort to retain potassium. Hence, administer potassium with the sodium bicarbonate infusion (20-40 meq/liter) unless the patient is in renal failure.



© Atir13 | Dreamstime.com

Frequent monitoring of arterial blood gas is necessary to detect systemic alkalosis.

If the patient is experiencing severe toxicity, one should consider hemodialysis. Indications are seizures, a salicylate concentration greater than 100 mg/dL, or a worsening acidosis in spite of treatment. Setting up for dialysis is often a cumbersome process involving multiple disciplines so consider this option early.

As a patient becomes increasingly acidemic, salicylate more easily crosses the blood brain barrier, where it exerts toxic effects. This is why the patient's respiratory drive is very important. A respiratory rate of 30-50 breaths/minute is not uncommon with severe toxicity and is a result of both the direct effect of the drug and a respiratory compensation to the developing anion gap metabolic acidosis. To take away the ventilatory drive can lead to sudden and severe acidemia and subsequent cardiovascular collapse. Thus, be careful intubating such patients for indications such as tachypnea and fear that the patient may tire. If intubation cannot be avoided, be sure to continue hyperventilating at a rate similar to the pre-intubation rate and check a blood pH 10-15 minutes later to access their acid-base status.

**References**

1. Chyka PA, Erdman AR, Christianson G, Wax PM, Booze LL, Manoguerra AS, Caravati EM, Nelson LS, Olson KR, Cobaugh DJ, Scharman EJ, Woolf AD, Troutman WG. Salicylate poisoning: an evidence-based consensus guideline for out-of-hospital management. *Clin Toxicol* 2007;45(2):95-131.
2. Poisoning and Drug Overdose 5th edition. Olsen et al. Lange, 2007:333-335.
3. Stolbach AI, Hoffman RS, Nelson LS. Mechanical ventilation was associated with acidemia in a case series of salicylate-poisoned patients. *Acad Emerg Med* 2008;15(9):866-9.

**CARBON MONOXIDE TOXICITY AND HYPERBARIC OXYGEN THERAPY**

Patrick Ockerse, MD  
Emergency Medicine Resident



© Danny Hooks | Dreamstime.com

Carbon monoxide (CO) binds tightly to hemoglobin (Hgb) displacing oxygen and causing hypoxia and inflammation. The

diagnosis can be made by measuring carboxyhemoglobin (COHgb) concentrations on blood gas analysis or with co-oximetry. It should be noted that standard pulse oximetry cannot distinguish between COHgb and O2 saturated Hgb. Patients can present with a variety of nonspecific complaints (e.g., headache, nausea, fatigue) and can have sequelae such as myocardial ischemia and delayed neurologic effects.

COHgb has a half life of about 300 minutes while breathing room air. This decreases to 60-90 minutes with supplemental oxygen via a reservoir "nonbreather" apparatus, and to about 20-30 minutes with hyperbaric oxygen therapy. The use of hyperbaric oxygen for CO toxicity remains controversial as there is no definitive data that proves it prevents the delayed neurologic complications. Initial randomized controlled trials have produced conflicting results and have study design flaws. There is a lack of standardization among studies regarding duration, frequency, and intensity of treatment, as well as evaluation for cognitive delays.

The American College of Emergency Physicians Clinical Policy (2008) states that "hyperbaric oxygen remains a treatment option but its use cannot be mandated" based on current evidence. Patients at high risk should have early hyperbaric consultation, especially those with moderate to severe toxicity (seizure or syncope, coma, altered mental status or confusion, abnormal neurologic examination, COHgb >25%, fetal distress in pregnancy, or any signs of end-organ damage; myocardial ischemia, or pulmonary edema). The major absolute contraindication for hyperbaric therapy is an untreated pneumothorax.

**TOXINS IN THE NEWS**

- **Quetiapine's** new drug label warns to avoid using it in combination with other drugs that may **prolong the QT interval**, including quinidine, procainamide, amiodarone, sotalol, other antipsychotic medications, certain antibiotics, pentamidine, levome-thadyl acetate, and methadone.
- McNeil Consumer Healthcare announced it is "reducing the maximum daily dose of Extra Strength Tylenol to lower risk of unintentional overdose from **acetaminophen**". It will list the maximum daily dose as six pills (3,000 mg) down from eight daily pills (4,000 mg).
- **Inorganic mercury** has been associated with "anti-aging", "beauty", and "skin lightening" cosmetic creams from foreign countries. Cases of toxicity have been reported.



© Kevin Zimarik | Dreamstime.com

## MEET THE UPCC STAFF

(cont. from pg. 2) **Battery**

a screwdriver to get into the battery compartment. Teach children that button batteries are not toys and should not be played with. Safety tips for button batteries can be found through the National Capital Poison Center's NBIH website: [www.poison.org/battery](http://www.poison.org/battery).

### Conclusion

Button battery ingestion can produce significant morbidity and mortality within a short period of time and can range from no effects to esophageal perforation and death. Battery ingestion needs to be identified as quickly as possible and the size of the battery should be noted. The NBIH and poison control centers are available 24/7 to answer questions and concerns about button battery ingestion.

### References

1. Litovitz T, Whitaker N, Clark L, et al. Emerging battery-ingestion hazard; clinical implications. *Pediatrics* 2010; 125:1168.
2. International Electrotechnical Commission. International Standard IEC 60086-1 Primary batteries – Part 1: General. 10th ed. Geneva, Switzerland. IEC 2006.
3. National Capital Poison Center. Button Batteries. Available at: <http://www.poison.org/battery>. Accessed October 31, 2011.
4. Environmental Protection Agency. Batteries. Available at: <http://www.epa.gov/mercury/consumer.htm#bat> Accessed October 31, 2011
5. Mallon P, White J, Thompson RL. Systemic absorption of lithium following ingestion of a lithium button battery. *Hum Exp Toxicol* 2004; 23:193-95
6. Shastri N, Leys C, Fowler M, Conners GP. Pediatric button battery and small magnet coingestion: two cases with different outcomes. *Pediatr Emerg Care* 2011;27(7):642-4.



### TOM DAVIES, PHARM.D

joined the poison control center team in June 2011, after becoming interested in toxicology in pharmacy school and during his 4th year drug information rotation. Tom has lived in Utah all of his life attending college at Weber State, Salt Lake Community and University of Utah. He studied multiple disciplines such as criminal justice and abnormal psychology before settling on pharmacy. Tom feels working at the UPCC as a pharmacist is an extremely unique and completely fulfilling career choice. Tom's favorite poison control calls are the ones that start with a frantic parent worried about the welfare of their child but end with unaffected child and the situation defused. In Tom's free time he enjoys: fishing, running, soccer, watching football and most of all hanging out with his two sons. His job at the poison control center has given him the opportunity to immediately affect caller lives for the better, interact with and assist healthcare professionals improve patient outcomes, continue to build on his toxicology knowledge through interactions with the extremely knowledgeable UPCC staff and most importantly, his oldest son believes he is super-hero because his dad works at UPCC. Tom plans to continue expanding his toxicology knowledge and is excited about the opportunity to teach pharmacy students about the fascinating field of toxicology.

## UTAH POISON CONTROL CENTER STAFF

### Executive Director/Editor

Barbara Insley Crouch, PharmD, MSPH

### Medical Director/Editor

E. Martin Caravati, MD, MPH

### Associate Medical Director

Douglas E. Rollins, MD, PhD

### Assistant Director

Heather Bennett, MPA

### Clinical Toxicology Fellow

Karen C. Thomas, PharmD, PhD, CSPI\*

### Specialists in Poison Information

Kathleen T. Anderson, PharmD, CSPI\*

Michael Andrus, PharmD, CSPI\*

Thomas Davies, PharmD

Bradley D. Dahl, PharmD, CSPI\*

Michael L. Donnelly, RN, BSN, CSPI\*

Ann Lystrup, RN, BSN, CSPI\*

Brittanie Hatch, PharmD, MS

Jeannett E. Madsen, RN, ASN, CSPI\*

Ed Moltz, RN, BSN, CSPI\*

Sandee Oliver, RN, BSN, CSPI\*

Cathie Smith, RN, BSN, CSPI\*

John Stromness, BS Pharm, RPh, CSPI\*

\*CSPI denotes Certified Specialist in Poison Information.

### Poison Information Providers

Lisa Chavez, BS

Angela Green, BS

Marilyn Redd

Kami Roake, BS

### Coordinator, Outreach Education

Marty C. Malheiro, MS, MCHES

### Health Educator

Sherrie Pace, BS, CHES

### Grant and Project Administration

David Craig

Kelly Teemant, BS, CHES/Publisher

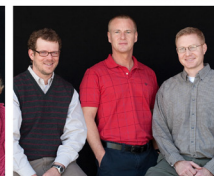
### Administrative Assistant

Brenda Clausing

Please send comments and suggestions for future articles to the editors of Toxicology Today at:

**585 Komas Dr., Suite 200  
Salt Lake City, Utah 84108**

Or send e-mail to  
[poison@hsc.utah.edu](mailto:poison@hsc.utah.edu)



## THANK YOU

*The Utah Poison Control Center expresses its sincere thanks to the health care professionals, public health officials and toxicology colleagues that work together to treat and prevent poisonings.*



UNIVERSITY OF UTAH  
COLLEGE OF PHARMACY

