

# Emerging Battery-Ingestion Hazard: Clinical Implications



**WHAT'S KNOWN ON THIS SUBJECT:** Most button battery ingestions are benign, although batteries that are lodged in the esophagus, especially large-diameter cells, may produce serious injury and must be removed. Damage is predominantly from an external current that causes electrolysis of tissue fluids, generating hydroxide.



**WHAT THIS STUDY ADDS:** Increasingly frequent, devastating complications from button battery ingestions are associated with 20-mm lithium cells. Removal from the esophagus must be accomplished within 2 hours. Misdiagnoses and delays have caused perforations, strictures, fistulas, vocal cord paralysis, exsanguination, and 13 reported deaths.

## abstract



**OBJECTIVES:** Recent cases suggest that severe and fatal button battery ingestions are increasing and current treatment may be inadequate. The objective of this study was to identify battery ingestion outcome predictors and trends, define the urgency of intervention, and refine treatment guidelines.

**METHODS:** Data were analyzed from 3 sources: (1) National Poison Data System (56 535 cases, 1985–2009); (2) National Battery Ingestion Hotline (8648 cases, July 1990–September 2008); and (3) medical literature and National Battery Ingestion Hotline cases (13 deaths and 73 major outcomes) involving esophageal or airway button battery lodgment.

**RESULTS:** All 3 data sets signal worsening outcomes, with a 6.7-fold increase in the percentage of button battery ingestions with major or fatal outcomes from 1985 to 2009 (National Poison Data System). Ingestions of 20- to 25-mm-diameter cells increased from 1% to 18% of ingested button batteries (1990–2008), paralleling the rise in lithium-cell ingestions (1.3% to 24%). Outcomes were significantly worse for large-diameter lithium cells ( $\geq 20$  mm) and children who were younger than 4 years. The 20-mm lithium cell was implicated in most severe outcomes. Severe burns with sequelae occurred in just 2 to 2.5 hours. Most fatal (92%) or major outcome (56%) ingestions were not witnessed. At least 27% of major outcome and 54% of fatal cases were misdiagnosed, usually because of nonspecific presentations. Injuries extended after removal, with unanticipated and delayed esophageal perforations, tracheoesophageal fistulas, fistulization into major vessels, and massive hemorrhage.

**CONCLUSIONS:** Revised treatment guidelines promote expedited removal from the esophagus, increase vigilance for delayed complications, and identify patients who require urgent radiographs. *Pediatrics* 2010;125:1168–1177

**AUTHORS:** Toby Litovitz, MD,<sup>a,b,c</sup> Nicole Whitaker, BSN, Med,<sup>a</sup> Lynn Clark, BSN,<sup>a</sup> Nicole C. White, BA,<sup>d</sup> and Melinda Marsolek, BA, MPH<sup>a</sup>

<sup>a</sup>National Capital Poison Center, Washington, DC; <sup>b</sup>Department of Emergency Medicine, Georgetown University School of Medicine, Washington, DC; <sup>c</sup>Department of Emergency Medicine, The George Washington University School of Medicine, Washington, DC; and <sup>d</sup>University of Virginia School of Medicine, Charlottesville, Virginia

### KEY WORDS

disc battery ingestion, button battery ingestion

### ABBREVIATIONS

NPDS—National Poison Data System  
NBIH—National Battery Ingestion Hotline  
OR—odds ratio

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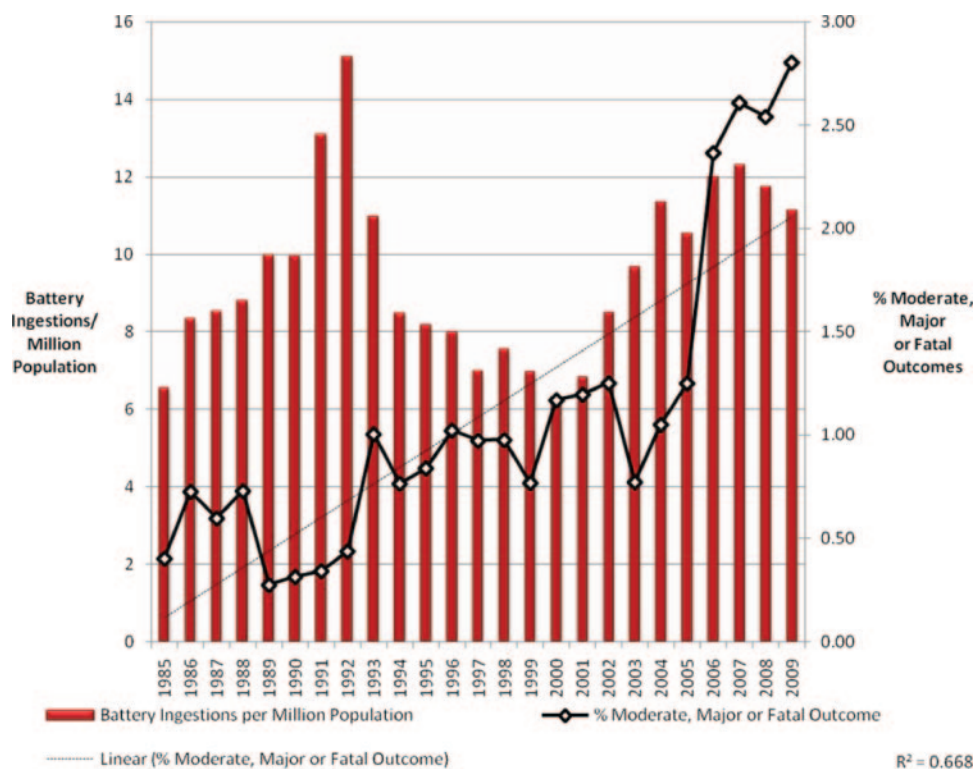
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Address correspondence to Toby Litovitz, MD, National Capital Poison Center, 3201 New Mexico Ave, Suite 310, Washington, DC 20016. E-mail: [toby@poison.org](mailto:toby@poison.org)

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**FIGURE 1**

NPDS button-battery ingestion frequency and severity (for moderate, major, and fatal outcomes), according to year.

In 1992, we analyzed 2382 battery ingestion cases. Most were benign, with no deaths and only 2 (0.1%) major effects.<sup>1</sup> Button batteries\* that were lodged in the esophagus posed the greatest risk, requiring prompt removal. Button cells of <15 to 18 mm in diameter generally passed through the gut uneventfully, and removal was rarely indicated for batteries beyond the esophagus.

Recent cases suggest that the nature of battery ingestions has evolved considerably. This investigation was undertaken to reassess the clinical course of battery ingestions and refine treatment guidelines.

## METHODS

Three data sources were examined:

1. National Poison Data System (NPDS): All 56 535 button battery ingestion cases that were reported

\*The terms "button battery" and "button cell" are used interchangeably. Lithium button batteries are often referred to as lithium coin cells.

to US poison centers (1985–2009) were obtained from the American Association of Poison Control Centers.† NPDS data are collected by specialists in poison information at each US poison center by using standardized data definitions.<sup>2,3</sup> Limited case detail restricted this analysis to frequency and outcome trends (see "Supplemental Information," which is published at [www.pediatrics.org/content/full/125/6/1168](http://www.pediatrics.org/content/full/125/6/1168)).

2. National Battery Ingestion Hotline (NBH): All 8648 battery ingestions (button and cylindrical) that were reported from July 1, 1990, through September 30, 2008 were analyzed. NBH data on clinical course and battery characteristics were used to determine outcome predictors and refine treatment guidelines.

†The American Association of Poison Control Centers disclosure statement is available at [www.poison.org/AAPCCdisclosureStatement.asp](http://www.poison.org/AAPCCdisclosureStatement.asp).

Since 1982, the National Capital Poison Center has maintained a 24/7 hotline to provide treatment guidance for battery ingestions (202-625-3333). Ingestions reported through June 1990 were previously published<sup>1,4,5</sup> (see "Supplemental Information").

3. All 13 fatal and 73 major outcome (life-threatening or disabling) cases involving esophageal or airway button battery lodgment reported in the literature or to the NBH at any time were obtained. Cases with sufficient clinical detail from the medical literature (36), NBH (43 cases, 1982–2009), both the literature and the NBH (6), or the media (1) were used to refine clinical guidelines and determine the urgency of battery removal<sup>1,4–46</sup> (see case lists at [www.poison.org/battery/FatalCases.asp](http://www.poison.org/battery/FatalCases.asp) and [www.poison.org/battery/SevereCases.asp](http://www.poison.org/battery/SevereCases.asp)).

This study was exempted from full review by the Georgetown University institutional review board.

## RESULTS

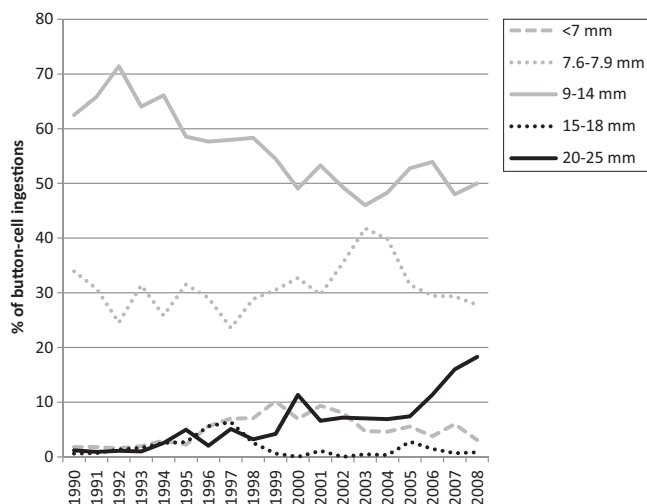
### NPDS: Frequency and Outcome Trends

NPDS data show 6.3 to 15.1 reported button battery ingestions per million population annually but fail to demonstrate a consistent upward or downward frequency trend (Fig 1). In 2007 to 2009, 3461 to 3758 button battery ingestion cases were reported to US poison centers annually. Of 56 535 button battery ingestions reported to the NPDS (1985–2009), 68.1% occurred in children who were younger than 6 years and 20.3% in children who were aged 6 through 19 years (Tables 5 and 6, which are published as supplemental information at [www.pediatrics.org/content/full/125/6/1168](http://www.pediatrics.org/content/full/125/6/1168)).

Although clinically significant (moderate, major, or fatal) outcomes occurred in only 1.3% of button cell ingestions during the 25-year period, the percentage of cases with clinically significant outcomes increased 4.4-fold from the first 3 years (0.596%) to the last 3 years (2.65%; linear regression,  $R^2 = 0.67$ ,  $P < .0001$ ; Fig 1). Focusing only on the most serious outcomes (Fig 5, which is published as supplemental information at [www.pediatrics.org/content/full/125/6/1168](http://www.pediatrics.org/content/full/125/6/1168)), there was a 6.7-fold increase in the rate of major or fatal outcomes by the final 3-year period (0.443%, 2007–2009) compared with the initial 3-year period (0.066%, 1985–1987). In comparison, there was a twofold increase in the percentage of major or fatal outcomes for all human poison exposures (all substances) reported to US poison centers.

### NBIH: Series Description and Battery Characteristics

During the 18.25-year period, 8648 battery ingestion cases were reported to



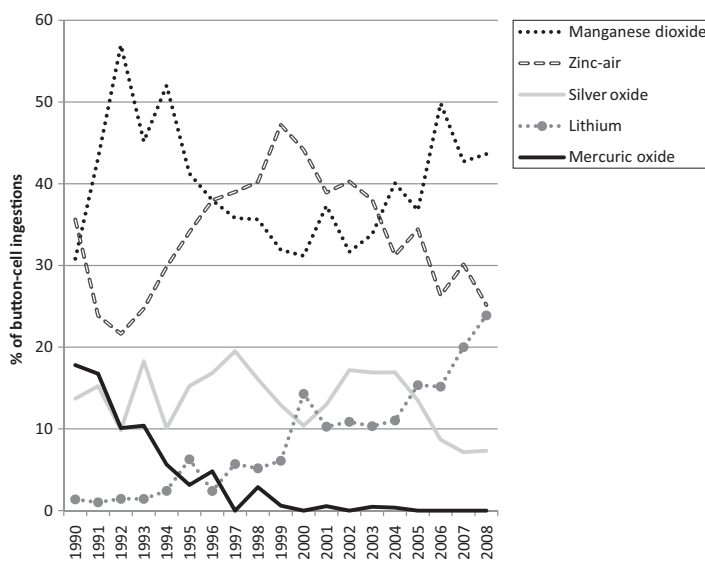
**FIGURE 2** Temporal changes in diameter of ingested button cells (NBIH).

the NBIH, including 8161 button cell and 487 cylindrical cell (eg, AA, AAA) ingestions. Ingestion was confirmed in 81.9% of cases, by radiograph (76.7%), battery presence in stool or emesis, or endoscopic retrieval. Children who were younger than 6 years were involved in 62.5% of button cell ingestions.

Four battery sizes accounted for 95% of ingested button cells with known diameter: 11.6 mm (55.1%); 7.8 to 7.9 mm (30.6%); 20 mm (6.4%); and 5.8 mm

(3.0%). Large-diameter cells ( $\geq 20$  mm), implicated in only 6.7% of ingested button cells during the entire period, increased from 1% of cases in 1990–1993 to 18% in 2008 (diameter known for 60.6%; Fig 2).

When button battery chemistry was known (57.7%), 41.7% were manganese dioxide/alkaline, 31.8% were zinc-air, and 13.1% were silver oxide. Whereas lithium cells composed only 9.0% of ingested button batteries during the entire period, 24% were lithium cells in 2008,



**FIGURE 3** Temporal changes in chemical systems of ingested button cells (NBIH).

**TABLE 1** Medical Outcome According to Diameter of Ingested Button Battery

Parameter	≤7 mm		7.6–7.9 mm		9–14 mm		15–18 mm		20–25 mm	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
No effect	177	80.82	1264	83.43	2310	82.29	60	81.08	171	51.98
Minor effect	11	5.02	53	3.50	174	6.20	9	12.16	48	14.59
Moderate effect	2	0.91	7	0.46	54	1.92	0	0.00	45	13.68
Major effect	0	0.00	0	0.00	2	0.07	0	0.00	30	9.12
Death	0	0.00	0	0.00	0	0.00	0	0.00	1	0.30
Unrelated	5	2.28	40	2.64	49	1.75	3	4.05	5	1.52
Unknown	24	10.96	151	9.97	218	7.77	2	2.70	29	8.81
Total	219	100.00	1515	100.00	2807	100.00	74	100.00	329	100.00

NBIH data, cases with known diameter (July 1990–September 2008 only).

**TABLE 2** Medical Outcome According to Cell Type, Number of Button Cells Ingested, and Button Cell Chemistry

Parameter	Button Cells	Cylindrical Cells	No. Ingested <sup>a</sup>		Button-Cell Chemistry					
			1	>1	Lithium	Manganese Dioxide	Mercuric Oxide	Silver Oxide	Zinc-Air	
Total cases	8161	487	7455	706	424	1967	203	615	1498	
Known outcome	7210	360	6591	619	380	1826	186	552	1340	
Outcome (% of cases with known outcome)										
No effect	88.06	73.89	88.56	82.71	63.68	89.43	89.78	90.04	92.09	
Minor effect	7.05	17.78	6.63	11.47	15.00	6.79	5.91	6.34	3.58	
Moderate effect	1.79	4.72	1.73	2.42	11.58	1.75	3.76	1.09	0.37	
Major effect	0.57	0.28	0.61	0.16	7.89	0.11	0.00	0.00	0.00	
Death	0.03	0.28	0.02	0.16	0.53	0.00	0.00	0.00	0.00	
Unrelated	2.51	3.06	2.46	3.07	1.32	1.92	0.54	2.54	3.96	

NBIH data (July 1990–September 2008 only). Lithium cells were more likely associated with clinically significant outcomes compared with each other button cell chemistry (logistic regression,  $P < .001$  versus zinc-air [OR: 65.2], silver oxide [OR: 22.5], manganese dioxide [OR: 13.1], and mercuric oxide [OR: 6.5]).

<sup>a</sup> Button cells only.

**TABLE 3** Age According to Outcome for Button Cell Ingestion Cases

Age, y	No Effect		Minor Effect		Moderate Effect		Major Effect		Death		Unknown or Unrelated		Total	
	<i>n</i>	Row %	<i>n</i>	Row %	<i>n</i>	Row %	<i>n</i>	Row %	<i>n</i>	Row %	<i>n</i>	Row %	<i>n</i>	Column %
<1	370	76.13	49	10.08	14	2.88	9	1.85	0	0.00	44	9.05	486	6.0
1	962	78.34	76	6.19	44	3.58	16	1.30	1	0.08	129	10.50	1228	15.0
2	874	80.18	66	6.06	21	1.93	7	0.64	1	0.09	121	11.10	1090	13.4
3	807	79.98	60	5.95	10	0.99	3	0.30	0	0.00	129	12.78	1009	12.4
4	558	76.54	66	9.05	13	1.78	0	0.00	0	0.00	92	12.62	729	8.9
5	361	78.31	40	8.68	2	0.43	0	0.00	0	0.00	58	12.58	461	5.6
6–19	912	74.82	94	7.71	18	1.48	4	0.33	0	0.00	191	15.67	1219	14.9
20–59	287	72.84	16	4.06	1	0.25	1	0.25	0	0.00	89	22.59	394	4.8
≥60	1083	83.50	38	2.93	6	0.46	0	0.00	0	0.00	170	13.11	1297	15.9
Unknown	135	54.44	3	1.21	0	0.00	1	0.40	0	0.00	109	43.95	248	3.0
Total	6349	77.80	508	6.22	129	1.58	41	0.50	2	0.02	1132	13.87	8161	100.0

NBIH data (July 1990–September 2008 only).

up from 1.3% in 1990–1993. Figure 3 shows the rise in lithium cells beginning in the late 1990s and a precipitous drop in ingestions of mercuric oxide cells in the early 1990s.

Imprint code was known for 56.7% of ingested button cells but often obtained after battery passage. Imported button cells occasionally had no imprint code.

Discharge state, known in 68.6% of cases, included 20.9% new, 65.8% partially spent (enough residual energy to power products), and 13.3% spent (“dead”).

### NBIH: Gut Transit Times and Use of Emetics, Endoscopy, and Surgery

See “Supplemental Information.”

### NBIH: Outcome Predictors

Of 33 major outcome or fatal cases with known diameter, 31 (93.9%) involved button batteries that were ≥20 mm (Table 1). No clinically significant % (moderate, major, or fatal) outcomes were observed with 15- to 18-mm cells, suggesting better outcomes compared with 20- to 25-mm cells ( $P < .0001$ , Fish-

er's exact test). Lithium cells were more likely associated with clinically significant outcomes than were other chemistries (Table 2).

Age was an important predictor of severity. All fatalities and 85% of major effects occurred in children who were younger than 4 years (Table 3). A major effect or death occurred in 12.6% of children who were younger than 6 years and ingested 20- to 25-mm batteries.

Logistic regression was used to identify button cell ingestion outcome predictors (Table 7, which is published as supplemental information at [www.pediatrics.org/content/full/125/6/1168](http://www.pediatrics.org/content/full/125/6/1168)). Battery diameter of 20 to 25 mm was the most important predictor of a clinically significant outcome (odds ratio [OR]: 24.6;  $P < .001$ ), followed by age younger than 4 years (OR: 3.2;  $P < .0001$ ) and ingestion of  $>1$  battery (OR: 2.1;  $P = .02$ ). Chemical system was not included in the model because it is highly correlated with diameter. When both diameter and chemistry were known ( $n = 4199$ ), 99.3% of 20- to 25-mm ingested button cells were lithium, 82.5% of lithium cells were 20 to 25 mm, 84.3% of zinc-air cells were  $<8$  mm, 87.8% of manganese dioxide cells were 9 to 14 mm (usually 11.6 mm), and all silver oxide cells were  $<15$  mm. It was not possible to determine the contribution of the lithium chemistry to ingestion severity for large-diameter cells, because only 2 ingestions were known to involve 20- to 25-mm non-lithium batteries. The outcome of ingestions of small lithium cells ( $<20$  mm) was no worse than the outcome of ingestions of other small button cells ( $P = .196$ , Fisher's exact test).

Of 221 ingestions of whole hearing aids (containing batteries), 78.7% were by individuals who were aged  $\geq 60$  years. When outcome was known (195 cases), 89.2% had no effect, 4.1% had a minor effect, and 1 patient with concomitant medical problems had a moderate ef-

fect. There were no major effects or deaths.

Cylindrical cell ingestions showed proportionately more minor and moderate effects (Table 2). Outcome was unknown more than twice as often because of the nature of cylindrical cell ingestions (at least 43% were intentional, suicidal, or associated with a neuropsychiatric disorder; another 6% were by incarcerated individuals). In addition, 37.8% of cylindrical cell ingestions involved multiple cells compared with 8.7% of button cell ingestions ( $P < .001$ ,  $\chi^2$ ).

New cells were 3.2 times more likely to be associated with clinically significant outcomes compared with spent cells when only 20- to 25-mm cells were assessed ( $P = .04$ , logistic regression); however, discharge state was not a predictor of outcome when all button cell sizes were considered. Co-ingestion of a battery and a magnet was reported in 2 NBIH cases. Both required  $\geq 1$  surgical procedure to resect necrotic bowel as a result of entrapment of the intestinal wall between the battery and the magnet. The association of battery corrosion and outcome is discussed in "Supplemental Information."

### **NBIH and Medical Literature: Clinical Issues and the Urgency of Removal**

Thirteen fatalities were identified (1977–2009), 9 (69%) in the most recent 6 years (2004–2009). All fatalities occurred in 11-month-old to 3-year-old children. Only 1 ingestion was witnessed. The diagnosis was missed by health care providers in 7 of the 13 deaths because of nonspecific presenting symptoms of vomiting, fever, lethargy, poor appetite, irritability, cough, wheezing, and/or dehydration. Batteries were in the esophagus for 10 hours to 2 weeks before removal or death. Exsanguination as a result of

esophageal fistulas into major arteries occurred in 9 patients, including 7 aorto-esophageal fistulas.<sup>7,11,12,46</sup> Delayed, unanticipated, and uncontrollable massive bleeding occurred up to 18 days after battery removal.

From 2000 to 2009, 92.1% of batteries that were identified in major and fatal ingestions were 20-mm lithium cells. Of major and fatal ingestions with known imprint code ( $n = 34$ , 2000–2009), most were CR 2032 (70.6%) or CR 2025 (20.6%). In 7 major or fatal outcome cases, batteries were  $<20$  mm (11.6–16 mm), and age, when known (5 of 7), ranged from 22 days to 10 months.

Of 73 major outcome cases, 91.8% occurred in children who were younger than 4 years (range: 22 days to 9 years;  $<1$  year: 28.8%; 1 year: 43.8%; 2 years: 11.0%; 3 years: 8.2%). Forty-one (56.2%) major outcome cases were unwitnessed (30.1% witnessed, 13.7% unknown). Nineteen (46.3%) of the unwitnessed ingestions were initially misdiagnosed compared with 1 witnessed ingestion (4.5%, battery mistaken for coin). Although most misdiagnoses involved failure to recognize the ingestion because of nonspecific symptoms, misdiagnoses also occurred when ingested batteries were misidentified as electrocardiogram electrodes, external objects,<sup>20,36</sup> or coins on radiograph or were above the uppermost radiograph border.

The battery was lodged in the esophagus for just 2 to 2.5 hours in 3 major outcome cases, causing severe burns, esophageal stenosis that required repeated dilation, a tracheostomy for persistent stridor, or bilateral vocal cord paralysis. Estimated time to removal was within 4 hours in 7 cases, within 8 hours in 17 cases (cumulative total), within 12 hours in 27 cases, within 24 hours in 36 cases, within 2 days in 42 cases, within 3 days in 48 cases, within 1 week in 60 cases, within



2 weeks in 67 cases, within  $\geq 6$  weeks in 72 cases, and completely unknown in 1 case. Delayed removal followed failure to seek medical care promptly, misdiagnosis, failure to recognize the need for urgent removal, transfer for pediatric endoscopy, or insistence on fasting before administering anesthesia.

Complications in major outcome cases included tracheoesophageal fistulas (35 cases [47.9%]), other esophageal perforations (17 cases [23.3%]), esophageal strictures or stenosis usually requiring repeated dilations (28 cases [38.4%]), vocal cord paralysis from recurrent laryngeal nerve damage (7 cases [9.6%]), mediastinitis, cardiac or respiratory arrests, pneumothorax, pneumoperitoneum, tracheal stenosis or tracheomalacia, aspiration pneumonia, empyema, lung abscess, and spondylodiscitis. Tracheoesophageal fistulas became symptomatic up to 9 days after battery removal (with even longer delays to diagnosis), strictures were delayed by weeks to months, and the single case of spondylodiscitis presented nearly 6 weeks after battery removal. Many patients required a tracheostomy, feeding tube, repeated esophageal dilation, and/or surgical esophageal or tracheal repair. See additional detail in “Supplemental Information.”

## DISCUSSION

All 3 data sets signaled worsening outcomes for button battery ingestions, paralleling the increase in household use and ingestion of 20-mm lithium coin cells. Lithium, the lightest metal, offers electrochemical efficiency, high-energy density, long shelf-life, and cold tolerance.

The association of 20-mm lithium cells with severe outcomes sheds light on the mechanism of battery-induced local injury. Previous experiments have invoked 3 factors, in order of importance<sup>15,47–52</sup>:

1. generation of an external electrolytic current that hydrolyzes tissue fluids and produces hydroxide at the battery’s negative pole,
2. leakage of alkaline electrolyte (hydroxide), and
3. physical pressure on adjacent tissue (which, alone, does not cause significant injury).

Unlike other button batteries, lithium coin cells contain a mildly irritating organic electrolyte instead of an alkaline electrolyte; therefore, leakage does not cause the local injury. Furthermore, 20-mm lithium cells are 3-V cells (twice the 1.5 V of other button cells), have a higher capacitance, and generate more current; therefore, lithium cells generate more hydroxide, more rapidly, than other button cells. The implication of lithium cells in most severe outcomes establishes the major injury mechanism as the generation of an external current, electrolysis of tissue or mucosal fluids, and local generation of hydroxide, rather than leakage. This is further supported by our finding (in large-diameter cells) that new cells were 3.2 times more likely to be associated with clinically significant outcomes compared with spent cells. (Although unable to power a product, spent cells have sufficient residual voltage and capacitance to generate an external current, produce hydroxide, and cause disastrous outcomes.) Because virtually all currently marketed large-diameter button cells in household use are lithium cells, we were unable to identify clinical data to confirm theoretical and animal observations showing that the lithium chemistry worsens outcomes compared with other chemistries, independent of the effect of the large diameter.<sup>47</sup>

The external electrolytic current generates hydroxide at the negative battery pole; therefore, the anatomic position and orientation of a battery lodged in the esophagus may predict

the specific subsequent injury. The most severe burns, delayed perforations, and fistulas are anticipated in the area adjacent to the lodged negative battery pole. Injury continues for days to weeks after battery removal, because of residual alkali or weakened tissues. Clinical expectations can be guided by the 3-Ns mnemonic “negative–narrow–necrotic”: the negative battery pole, identifiable as the narrow side on lateral radiograph, causes the most severe necrotic injury. The narrower negative pole is best identified on a film that is truly lateral with respect to the battery; obliqueness may obscure this determination. On direct visualization, the negative pole is the flat surface without imprint code or “+” sign. Clinicians are cautioned to refer to the negative and positive poles and avoid the terms “anode” and “cathode” because these terms reverse depending on context—that is, whether the frame of reference is the expected internal battery electrochemical process or the external electrolytic reaction that occurs when the battery is immersed in electrolyte.

Serious battery ingestion complications are related to local corrosive injury rather than systemic poisoning from battery contents. The US Mercury-Containing and Rechargeable Battery Management Act<sup>53</sup> of 1996 banned the sale of mercuric oxide button cells, and no mercuric oxide battery ingestions have been reported to the NBH since 2004. Despite *in vitro* data suggesting that minute amounts of other metals may be absorbed from batteries<sup>54</sup> and an asymptomatic 5-year-old with a serum lithium concentration transiently elevated (within the usual therapeutic range) after ingestion of a lithium cell,<sup>55</sup> no case of significant poisoning has been reported from these other metals. A rash attributed to nickel allergy may occur after ingestion of nickel-plated cells

(28 cases reported in our previous series).<sup>1</sup> Although endoscopists frequently describe a black precipitate and tissue discoloration surrounding the retrieved or impacted battery and a similar black precipitate is sometimes seen in the stool, the precipitate is the result of corrosive dissolution of the battery can and does not indicate heavy metal poisoning.

Clinicians missed the diagnosis of a battery lodged in the esophagus in at least 27% of major outcome and 54% of fatal cases because of nonspecific presentations, especially in unwitnessed ingestions. Unfortunately, 92% of fatalities and 56% of major outcome ingestions were unwitnessed, and most occurred in very young, often nonverbal children. A strategy to avoid these misdiagnoses, short of heightened vigilance or the overuse of radiographs, remains elusive, because patient presentations generally suggested other, more common diagnoses.

The window of opportunity for injury-free removal of an esophageal battery is <2 hours, considerably shorter than previously reported, possibly related to the greater voltage and capacitance of the lithium cell. Animal experiments confirm the rapid onset of severe injury observed in humans.<sup>13,15,47</sup> Delays introduced by late presentation, misdiagnosis, limited access to endoscopists, referral to a tertiary care facility, or concern about anesthesia induction after eating undoubtedly contributed to the severity of complications. Hemorrhage occurred in 12 of the 13 deaths, and no reported patients with massive delayed hemorrhage survived. Thus, anticipating delayed hemorrhage on the basis of battery position, performing serial diagnostic studies, prolonging in-hospital observation for patients who are at risk (on the basis of battery location and degree of injury), and intervening promptly when bleeding de-

**TABLE 4** Item Diameters for Comparison

Item	Diameter (mm)
Pencil eraser	6–7
Dime	18
Penny	19
Nickel	21
Quarter	24

Coin specifications, United States Mint.

velops might prove life-saving. Specific clinical recommendations on the basis of burn location and severity need to be developed, addressing issues such as frequency of scoping or imaging, duration of hospitalization, interval to initiation of feeding, and use of antibiotics and steroids.

Although outcome is determined by battery diameter and chemistry, these parameters are initially unknown in >40% of cases. Imprint code can be determined from a companion or replacement battery, package label, or product instructions (often on the Web). Lithium-cell imprints are standardized. For the CR 2032, the cell most frequently implicated in major and fatal outcomes, “CR” represents the lithium manganese dioxide chemistry, “20” is the diameter in millimeters, and the final 2 digits (“32”) indicate height (3.2 mm). In the absence of an imprint code, the parent or patient can compare the battery or slot diameter with standard items (Table 4). Batteries that are larger than a penny are of immediate concern, and batteries that are bracketed in diameter by a penny and a nickel should be assumed to be 20-mm lithium cells. Radiographs usually overestimate battery diameter unless magnification is corrected.

### Revised Triage and Treatment Guideline

Our findings inform a revised NBIH guideline for button battery ingestions (Fig 4; updated at [www.poisson.org/battery/guideline.asp](http://www.poisson.org/battery/guideline.asp)). An urgent initial radiograph is required for most

battery ingestions, but the requirement is waived in asymptomatic ingestions of ≤12-mm button batteries in patients who are older than 12 years, because significant complications are unlikely. (The 12-mm cutoff captures most ingested batteries [5.8–11.6 mm], and outcome data in this age and size range are robust.) In contrast, younger children always require an immediate radiograph to exclude an esophageal battery, even when asymptomatic, because 36% of patients with batteries lodged in the esophagus were initially asymptomatic.<sup>1</sup>

Batteries that are in the esophagus must be removed within 2 hours. Batteries that are in the stomach or beyond in an asymptomatic patient should be left to pass spontaneously, unless a magnet was co-ingested, with inspection of the stool or possible repeat radiograph in 10 to 14 days to confirm passage. A co-ingested magnet mandates prompt removal.

Although the supporting data are not definitive, children who are younger than 6 years and have ingested ≥15-mm batteries should have another radiograph 4 days after ingestion (increased from the previous 2–3 days) to confirm that the battery has moved beyond the stomach and endoscopic retrieval if still retained in the stomach. Earlier retrieval is indicated when any symptoms are evident, because these may indicate gastric ulceration or undetected previous esophageal lodgment. (For additional detail, see “Supplemental Information.”)

The revised guideline addresses pitfalls that have haunted clinicians. Radiographs should be examined for the battery’s double-rim or halo effect on anteroposterior radiograph or step-off on the lateral view, to make sure the “coin” or “electrocardiogram electrode” is not really a battery.<sup>35</sup> Endoscopic removal of esophageal batteries (rather than blind retrieval by





balloon catheter or magnet) is essential to determine the extent of injury and anticipate complications. Endoscopists should avoid pushing an esophageal battery into the stomach, because the esophageal perforation risk may increase.

Specific complications should be anticipated on the basis of injury location and structures that are adjacent to the hydroxide-generating negative pole. Tracheoesophageal fistulas became symptomatic within 9 days after battery removal, but the most frequent fatal complication—fistulization into an artery—manifested as late as 18 days after removal. Despite a paucity of specific guidance, the clinician must determine the length of observation, duration of esophageal rest, and need for serial imaging or endoscopy/bron-

choscopy on the basis of injury severity and location. Patients who are at risk of perforation into major vessels must be monitored closely as inpatients, with serial imaging and stool guaiacs, and early intervention if perforation is imminent or any evidence of bleeding develops.

### Ineffective Interventions

See “Supplemental Information.”

### Limitations

See “Supplemental Information.”

### CONCLUSIONS

Serious and fatal button battery ingestions are occurring with increasing frequency as a result of the emergence of the 20-mm lithium coin cell as a popular household battery. To improve

outcomes, health professionals must consider the diagnosis (particularly in unwitnessed ingestions), accurately discern batteries from coins, immediately remove batteries that are lodged in the esophagus because severe injury can occur in just 2 hours, and anticipate delayed complications. Primary prevention efforts must also be escalated.<sup>56</sup>

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