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Button Battery Ingestions

A Review of 56 Cases

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• **A retrospective analysis of 56 button (miniature) battery ingestions was conducted. This represents the largest series in the literature studying this problem. Impaction of these foreign bodies, most frequently in the esophagus (five cases), was a uniform predictor of severe morbidity. In the remaining 51 cases, the battery traversed the esophagus without incident; only four of these ingestions produced symptoms, and there was only one case with any severe complications. In 33 asymptomatic patients, the battery passed spontaneously through the gastrointestinal tract. Fourteen patients underwent endoscopic or operative procedures or both despite the absence of symptoms. Unanticipated mucosal erosions were noted in seven of these patients, although no symptoms or sequelae developed. Initial chest roentgenogram and observation for symptoms will detect ingestors at risk of complications. Operative or endoscopic intervention should be withheld in the absence of these clinical indicators. Button batteries can routinely be allowed to pass spontaneously.**

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EXTRAPOLATIONS from data reported to the National Poison Center Network (NPCN)[1] for 1980 indicate that between 510 and 850 button (miniature) battery ingestions occur annually in the United States. Button batteries were cited in 1.7 of every 10,000 reported poisonings in 1980. The estimated actual annual incidence of poison exposures is 3 to 5 million. This high incidence reflects advances in electronic miniaturization and the increasing availability of these cells in homes. The battery industry has predicted rapid growth for the calculator and watch battery markets[2]; thus, the frequency of these ingestions can only be expected to increase.

See also pp 2502, 2504, and 2509.

Although a large number of button battery ingestions occur each year, only six cases have been reported in the medical literature.[3-8] The reports of two deaths[3,4] and two seriously ill children,[7,8] however, have introduced a bias into the medical literature that is apparent to clinicians and poison center directors who have managed multiple cases uneventfully.

Clinical presumption of alarming morbidity and mortality has generated increasing controversy regarding the appropriate medical management of button battery ingestors. Opinions range from those who would "let nature take its course" (as though managing the ingestion of a penny), to advocacy of mandatory, urgent operative intervention.[7]

In an initial effort to resolve this conflict, this study was conducted to identify features of serious cases (which might allow accurate prediction of increased risk) and to determine whether button batteries can pass spontaneously without adverse consequence.

METHODS

Button battery ingestions were studied with respect to size, components, locations, and passage of the battery and the ingestor's age, symptoms, and therapy. Data were compiled from seven sources: three major battery manufacturers (Union Carbide [12 cases], Ray-O-Vac [seven], and Duracell [one]), the Consumer Product Safety Commission (five), the National Button Battery Ingestion Study (five), and the medical literature (six). (Cases reported to a particular manufacturer often involved other manufacturer's products.) There was no overlap in reporting or duplication of cases. The single largest source of data was the National Poison Center Network Computer System for Documenting Poison Exposures (20 cases). Each computer-retrieved case was investigated through a detailed questionnaire to the reporting poison center to obtain data in depth. Of the five cases reported to the Consumer Products Safety Commission, four (cases 45 and 50 through 52) are also reported by Votteler et al in this issue (p 2504). Cases reported to battery manufacturers often contained sufficient identifying data to allow telephone investigation by contacting the treating physician. Nonetheless, 36 additional suspected ingestions had to be excluded, since no outcome data could be obtained. With the exception of the five cases reported to the National Button Battery Ingestion Study, this series is retrospective.

RESULTS

Fifty-six cases were collected between January 1978 and July 1982 from the United States (52), Britain (two), Canada (one), and South Africa (one).

The age distribution of patients ingesting button batteries in the 50 cases for which the exact age was known ranged from 11 months to 90

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Table 1.—Batteries Lodged in the Esophagus

Case/Age/Sex	Source	Description of Battery: System*	Diameter, mm	Use	Treatment	Clinical Course
1/2½ yr/M	Blatnick et al[3]	MnO_2	23.0	Movie camera	Endoscopic removal from esophagus at thoracic inlet; steroids; antibiotics	Presented with 24 hr vomiting, fever, inability to talk or swallow; death 9 days later secondary to massive tracheoesophageal fistula and exsanguination
2/16 mo/F	Shabino & Feinberg[4]	MnO_2	23.0	Camera flash	Endoscopic removal from upper esophagus; right tube thoracostomy; antibiotics; mediastinal drainage; feeding gastrostomy	Presented 4 days after ingestion with vomiting, fever, tachypnea, dehydration; right tension hydropneumothorax; death secondary to perforation of aortic arch
3/25 mo/M	Vottelor[7]	. . .	21-23	. . .	Endoscopic removal from upper esophagus; sacrospinalis muscle flap to control massive tracheoesophageal fistula; colon interposition after 7 mo	Presented 5 days after ingestion with cyanosis, tachypnea, dysphagia; tracheoesophageal fistula; cardiac arrest (successful resuscitation)
4/5 yr/F	Union Carbide	MnO_2	23.0	. . .	Endoscopic removal within 4 hr from lower esophagus; steroids for 4-6 wk	Odynophagia developed within 4 hr of ingestion; second-degree burns of lower esophagus; no sequelae
5/16 mo/. . .	NPCN & Milmoe†	MnO_2	23.0	. . .	Endoscopic removal within 6 hr of ingestion from esophagus (level of cricopharyngeus); steroids; antibiotics	Symptomatic child examined shortly after ingestion, barium swallow 7-10 days after ingestion demonstrated dye extravasation into mediastinum that resolved in 6 wk; no sequelae

*MnO_2 indicates manganese dioxide.
†NPCN indicates National Poison Center Network. Gregory Milmoe, MD, oral communication, April 1982.

years. Seventy-eight percent of cases occurred in children younger than 5 years, a percentage comparable with poisonings in general.[1,9] The remaining six cases were also pediatric, although the exact age was not specified.

In five cases (Table 1), including three ingestions previously reported in the medical literature,[3,4,7] the button cell lodged in the esophagus. In four instances the button battery arrested in the proximal esophagus (generally at the level of the cricopharyngeus). All five children were symptomatic, and the only two deaths in the entire series occurred in this group. Battery ingestion in the three most severe cases (1, 2, and 3) was initially unsuspected and medical evaluation delayed.

Large button cells were involved in all five cases in which the battery lodged in the esophagus. These batteries have diameters of 21 to 23 mm (about the size of a quarter) compared with the usual, smaller button cell. Indeed, in four of these five cases, the identical battery was implicated: a manganese dioxide, 23.0×5.8-mm, 7-g cell with imprint code EPX 825 (or PX 825) and potassium hydroxide electrolyte. (While the composition of the cell in case 3 is unknown, the diameter was in excess of 21 mm.) Of note, this 23-mm manganese dioxide button cell was implicated in only one of 28 cases with known cell dimensions in which the battery passed beyond the esophagus (see case 48 below). In the other 27 cases, battery diameters were either 7.9, 11.6, or 15.6 mm.

The button cell passed spontaneously beyond the esophagus in the remaining 51 cases. Thirty-three of these patients were entirely asymptomatic and received no endoscopic or surgical intervention (Table 2). Fourteen additional asymptomatic cases underwent endoscopy, surgery, or both. No mucosal lesions were noted in seven of these patients (Table 3). In the other seven patients (Table 4), although also asymptomatic, surgical or endoscopic intervention revealed unanticipated gastrointestinal tract mucosal lesions. Of the 51 ingestors in whom the battery passed beyond the esophagus, only four were symptomatic (Table 5). Of these four cases, three have already been reported in the medical literature.

Four cases were multiple ingestions, including a button cell and a penny (case 26), two identical calculator batteries (case 33), two batteries concealed in a taco (case 41), and eight hearing aid batteries by a deaf student (case 42). The prevalence of hearing aid batteries in this series is of note, accounting for 36.8% of 38 cases in which the battery's source was known. (Of known sources, camera accessories and watches each accounted for 23.7% of batteries, and calculators for 15.8%.)

Of the 37 cases in which the battery was allowed to pass through the gut spontaneously, transit time, documented in 25 cases, ranged from 14 hours to seven days (Fig 1). Battery passage was delayed beyond 48 hours in 36% of cases.

Four patients received syrup of ipecac (cases 4, 33, 44, and 52). Subsequent emesis did not expel the button cell in any of these cases. Endoscopic removal under general anesthesia was attempted in eight cases (39, 40, 43, 46, 47, 48, 50, and 52), but was successful in only three (40, 47, and 50). (Case 44 is excluded from this tabulation since endoscopy was performed only for visualization, not for battery retrieval.) Surgical intervention followed four of five unsuccessful endoscopic removal attempts. The remaining patient (case 43) was allowed to pass the cell spontaneously. Thus, in 62.5% of cases in which endoscopic retrieval was attempted, the button cell could not be removed by this technique despite direct visualization of the battery. Several endoscopists described grabbing or "lassoing" the cell only to lose hold of the slippery foreign body. Greater endoscopic success would have been expected with a "basket" for foreign-body retrieval through the endo-

Table 2.—Button Batteries Beyond Esophagus: Asymptomatic Cases, Without Intervention*

Case/Age/Sex	Source†	Description of Battery‡			Interval Between Ingestion and Passage in Stool, hr	Comment
		System	Diameter, mm	Use		
6/Child/. . .	Ray-O-Vac	Ag_2O	11.6	Hearing aid	96	. . .
7/2½ yr/F	Ray-O-Vac	Ag_2O	11.6	. . .	. . .	. . .
8/59 yr/F	Ray-O-Vac	Ag_2O	11.6	. . .	85	. . .
9/2 yr/	Ray-O-Vac	Ag_2O	11.6	Hearing aid	24	. . .
10/5 yr/M	Ray-O-Vac	HgO	. . .	. . .	144-168	. . .
11/3 yr/M	NBBIS	Ag_2O	11.6	Calculator	42	. . .
12/10 yr/M	NBBIS	. . .	. . .	Hearing aid	. . .	Actual battery passage not observed but absent on follow-up roentgenogram
13/11 mo/M	NBBIS	. . .	. . .	. . .	. . .	Parent found battery in child's diaper; moderate corrosion implicates prior ingestion
14/18 mo/M	Union Carbide	Ag_2O	7.9	Watch	. . .	. . .
15/7 yr/. . .	Union Carbide	HgO	15.6	. . .	60	. . .
16/Child/. . .	Union Carbide	Ag_2O	11.6	. . .	. . .	. . .
17/18 mo/. . .	Union Carbide	HgO	15.6	. . .	. . .	. . .
18/Child/. . .	Union Carbide	. . .	. . .	Hearing aid	. . .	. . .
19/22 mo/F	Union Carbide	Ag_2O	7.9	. . .	24	. . .
20/1 yr/F	NPCN	Ag_2O	11.6	Calculator	72	. . .
21/5 yr/F	NPCN	. . .	. . .	Hearing aid	72	. . .
22/3 yr/F	NPCN	. . .	. . .	Watch	96	. . .
23/3 yr/M	NPCN	HgO	11.6	Hearing aid	23	. . .
24/1 yr/M	NPCN	. . .	. . .	Hearing aid	. . .	. . .
25/15 mo/M	NPCN	HgO	11.6	Camera	. . .	. . .
26/18 mo/M	NPCN	. . .	. . .	Watch	48	Ingested penny simultaneously
27/1 yr/M	NPCN	. . .	. . .	. . .	. . .	. . .
28/10 yr/M	CPSC	. . .	. . .	Watch	. . .	Battery not documented in stool, but roentgenogram at 36 hr located battery 6 in from rectum; child asymptomatic 1 mo later
29/17 mo/F	NPCN	Ag_2O	11.6	Camera	24	. . .
30/90 yr/F	NPCN	. . .	. . .	Hearing aid	14	. . .
31/12 yr/M	NPCN	Ag_2O	7.9	Watch	24	. . .
32/4½ yr/F	NBBIS	. . .	. . .	Watch	. . .	Battery not documented in stool; 26-hr roentgenogram located battery at ileocecal valve; absent on 72-hr roentgenogram
33/15 mo/F	NPCN	Ag_2O	7.9	Calculator	24	2 identical batteries ingested; ipecac administered unsuccessfully
34/4 yr/F	NPCN	Ag_2O	11.6	Watch	24	. . .
35/4 yr/M	NPCN	HgO	11.6	Hearing aid	24	. . .
36/7 yr/M	NPCN	HgO	. . .	Hearing aid	72	. . .
37/6 yr/M	NPCN	. . .	. . .	Camera	48	. . .
38/7 yr/M	NPCN	. . .	. . .	Watch	24	. . .

*No surgical or endoscopic procedures.
†NBBIS indicates National Button Battery Ingestion Study; NPCN, National Poison Center Network.
‡Diameter and system known only where manufacturers' imprint code available. Ag_2O indicates silver oxide; HgO, mercuric oxide.

scope.

Seventeen of the batteries involved in this study were described as "corroded, pitted, or split." Twelve passed through the gastrointestinal tract spontaneously; five were surgically removed. The battery's can-grommet interface was frequently the site of the most severe corrosion. Four batteries split open. The presence of corrosion or evidence of leakage did not correlate with the magnitude of the patient's symptoms.

A large variety of sizes, shapes, and chemical systems are required to meet the varying demands of calculators, watches, hearing aids, and pacemakers. The choice of battery system is based on size, weight, and shelf-life constraints, as well as energy density, leakage, and corrosion characteristics. Three battery systems were encountered in this series: (1) the alkaline or manganese dioxide (MnO_2) system with its manganese dioxide cathode and zinc anode; (2) the silver oxide (Ag_2O or AgO) system with a silver oxide cathode and zinc anode; and (3) the mercuric oxide (HgO) system with a mercuric oxide cathode and zinc anode. All of these systems contain an alkaline electrolyte that is generally a 45% solution of potassium hydroxide but may be sodium hydroxide. The electrolyte constitutes a very small volume of each cell (1% to 12% by weight of the 1- to 10-g battery).

The unusual propensity of the larger (23-mm diameter) cells to become lodged in the esophagus was noted previously. The large size of these batteries is likely responsible for their increased toxicity rather than their manganese dioxide (alkaline) system, the other common feature of batteries associated with esophageal damage.

A useful division of cases in which the button battery passed beyond the esophagus separates those cases that were entirely benign (Tables 2 and 3)

Table 3.—Button Batteries Beyond Esophagus*

Case/Age/Sex	Source†	Description of Battery: System	Diameter, mm	Use	Clinical Course
39/13 mo/F	NPCN	. . .	. . .	Calculator	Unsuccessful endoscopy led to gastrotomy 5 hr after ingestion
40/15 mo/M	Union Carbide	HgO	15.6	. . .	Battery retrieved from stomach by endoscopy within 24 hr
41/12 yr/M	NPCN	. . .	. . .	. . .	Child ingested 2 batteries placed in a taco by his uncle; one passed in the stool; second was removed surgically as abdominal roentgenogram revealed it split in two parts
42/Child/. . .	Ray-O-Vac	HgO	11.6	Hearing aid	8 batteries ingested simultaneously by a student at a school for the hearing impaired; all removed surgically the same day
43/2 yr/M	Ray-O-Vac	HgO	11.6	Hearing aid	After 2 unsuccessful endoscopic retrieval attempts, surgery was scheduled; however, preoperative abdominal roentgenogram localized the battery in the ascending colon; battery passed within 48 hr
44/10 yr/M	NBBIS	HgO	7.9	Watch	Battery visualized in stomach by endoscopy shortly after ingestion; no retrieval attempt made; battery passed in stool within 48 hr
45/2½ yr/M	CPSC	. . .	. . .	Calculator	Battery removed by jejunotomy 6 hr after ingestion; a black precipitate surrounded the battery and adjacent mucosa, but no mucosal lesions were noted

*Asymptomatic cases where endoscopy or surgery was performed and no mucosal lesions noted.
†NPCN indicates National Poison Center Network; NBBIS, National Button Battery Ingestion Study; CPSC, Consumer Product Safety Commission.

from those with mucosal lesions or symptoms (Tables 4 and 5). An analysis of the 28 cases in which the manufacturers' identifying imprint code could be determined for the battery reveals the following: (1) Of the entirely benign ingestions, 22 button cells were completely identified, including 13 silver oxide cells and nine mercuric oxide cells. (2) Of those ingestions that became symptomatic or with mucosal lesions identified at the time of endoscopy or surgery, six button cells were completely identified, including five mercuric oxide cells and one manganese dioxide (alkaline) cell.

Although silver oxide and mercuric oxide systems each accounted for roughly half of the ingested cells, there was a statistically significant ($P<.05$ by χ^2 analysis) association of mercuric oxide cells with cases with symptoms or mucosal lesions, occurring in five of six such cases *v* only nine of 22 entirely benign ingestions. This association may be related to the known corrosive effect of mercuric oxide. It is also of note that no silver oxide button cells were implicated in cases with mucosal lesions or symptoms.

COMMENT

The data presented allow the formulation of a rational management protocol. All six cases with severe complications involved impaction of the foreign body with surrounding tissue necrosis. Furthermore, in five of these cases, the button battery lodged in the esophagus. It is imperative that every button battery ingestor have an emergent chest roentgenogram to exclude the possibility of an esophageal location. This exhortation follows the observation of esophageal injury in case 4, only four hours after ingestion. Esophageal lodgment is further implicated by the rapid development of symptoms—dysphagia, vomiting, anorexia, and fever. In the two toddlers who died, the parents were unaware of the battery ingestions.[3,4] In a third severely affected child (case 3), the mother ignored the child's persistent vomiting and refusal to eat for five days before seeking medical attention.

While button batteries located in the esophagus must be removed immediately, a considerably less invasive approach is indicated if the battery has passed beyond the esophagus. Ipecac administration is not advised because of the small but real risk of airway obstruction developing at the time of regurgitation of the foreign body. Furthermore, in the four cases in which it was used, ipecac-induced vomiting did not expel the battery.

Several authors recommend immediate surgical or endoscopic intervention to remove button batteries; however, endoscopy proved unsuccessful in a majority (62.5%) of cases in which it was used to retrieve the battery. Moreover, successful endoscopy in small children is likely to require general anesthesia and specialized equipment. This study does not support the need for urgent intervention in the 51 cases in which the battery passed beyond the level of the esophagus. Only one of these cases was associated with a severe complication: a perforated Meckel's diverticulum. The presence of this anatomic feature in less than 2% of the population provides scant justification for any invasive procedure in an asymptomatic ingestor when the battery has passed beyond the esophagus. Mucosal lesions, noted incidentally in seven asymptomatic patients, were not associated with complications or sequelae. Knowing that retrieval attempts were not precipitated by any signs or symptoms in these cases, it is highly unlikely that these patients would have suffered adverse consequence if left to pass the button cells through the gastrointestinal tract spontaneously. Indeed, symptoms developed in only four patients of the 51 whose batteries passed beyond the esophagus. Since three of these were previously reported as single case reports in the medical literature, the actual frequency of symptomatic patients has been distorted by the tendency to report only the complicated or unusual. In only one of these four patients were symptoms severe (case 53, with the perforated Meckel's diverticulum).

Recent statements in the literature

vigorously advocate surgical intervention if the battery does not pass within 48 hours.[8,10] However, these recommendations are anecdotal and are not supported by the clinical data presented herein. This, the largest clinical study of this problem to date, demonstrated no correlation between transit time and the presence of even minor symptoms. Daily roentgenograms to monitor battery progression are often espoused; however, careful observation (at home) for symptoms would seem sufficient. Cathartics are routinely advocated to hasten transit. In theory, cimetidine, antacids, or metoclopramide hydrochloride administration to minimize gastric acid-induced corrosion may be of some additional value, but these recommendations require confirmation in animal studies.

Button batteries are constructed with an electrolyte-soaked fabric inserted between a cathode can and anode top (Fig 2). Although the mechanism of injury from these batteries has yet to be fully elucidated, one or several factors are doubtlessly culpable:

1. A small percentage of button batteries spontaneously leak electrolyte. Leakage may occur circumferentially at the seal (see diagram in Fig 2). The alkaline electrolyte can cause liquefaction necrosis of tissue. This might represent a severe problem if the battery were lodged in a single area rather than free-floating in the gut where the potassium hydroxide would be diluted, producing a more diffuse but less intense effect. Impaction also allows a slow leak to have a cumulative effect on localized tissue.

2. Spontaneous leakage of electrolyte in some imperfectly sealed batteries may be limited to diffusion of fluid (in both directions) across the seal in a liquid medium. Impaction would again intensify the local effect.

3. Mercuric oxide has known corrosive effects but is poorly soluble and poorly absorbed. Even in cases of split batteries, mercury poisoning has only been documented in one case, and there it was of low level.[11] As cells discharge, the highly toxic mercuric oxide is converted to essentially nontoxic elemental mercury. Further-

Table 4.—Button Batteries Beyond the Esophagus*

Case/Age/Sex	Source†	Description of Battery‡: System	Diameter, mm	Use	Clinical Course
46/Child/. . .	Union Carbide	HgO	15.6	. . .	Unsuccessful gastroscopic retrieval attempts led to surgical removal; the battery was adherent to the greater curvature with underlying mucosal irritation; no sequelae
47/2½ yr/. . .	Union Carbide	HgO	15.6	. . .	Endoscopy 6 hr after ingestion; a few gastric erosions were noted and the button battery was successfully retrieved; no sequelae
48/3 yr/M	Union Carbide	MnO_2	23.0	. . .	Unsuccessful endoscopic retrieval attempts led to surgical removal; superficial mucosal burns in fundus and *antrum; no sequelae*
49/17 mo/F	Union-Carbide	HgO	. . .	Light meter	Gastrotomy performed 18 hr after ingestion; gastric erosions and minor bleeding; no sequelae
50/5 yr/M	CPSC	. . .	. . .	Camera	Endoscopy was performed 2 hr after ingestion with successful battery retrieval; superficial gastric erosions; no sequelae
51/21 mo/M	CPSC	. . .	. . .	Hearing aid (his own)	Operative retrieval 6 hr after ingestion; gastritis observed; no sequelae or lesions on follow-up studies
52/3 yr/M	CPSC	. . .	. . .	. . .	Ipecac administration did not expel battery; unsuccessful gastroscopy led to gastrotomy; mucosal erosions and a greenish-black precipitate noted; no sequelae

*Asymptomatic cases with unanticipated evidence of mucosal lesions.
†CPSC indicates Consumer Products Safety Commission.
‡HgO indicates mercuric oxide; MnO_2, manganese dioxide.

Table 5.—Button Batteries Beyond the Esophagus: Symptomatic Cases

Case/Age/Sex	Source	Description of Battery*: System	Diameter, mm	Use	Clinical Course
53/2½ yr/M	Willis & Ho[8]	HgO	11.6	Hearing aid	Intermittent abdominal pain, guarding, tenderness, and vomiting developed; button battery lodged in Meckel's diverticulum with necrosis and perforation; small-bowel resection required
54/2 yr/M	Reilly[6]	HgO	15.6	Camera	Child passed black stool; battery split in two parts during gastrotomy; mercury levels were within normal range
55/1 yr/F	Barros & Barros[5]	HgO	15.6	. . .	48-hr roentgenogram revealed split button cell (one half in stomach, other half in ascending colon); both passed in stool by 72 hr; child vomited and had dark-gray stools initially; anorexia and lassitude lasted 1 wk; no mercury levels were *assayed*
56/Child/F	Duracell	HgO		. . .	Roentgenogram demonstrated battery split in stomach; tarry stools and minor upper gastrointestinal tract bleeding; battery passed in stool within 24 hr

*HgO indicates mercuric oxide.

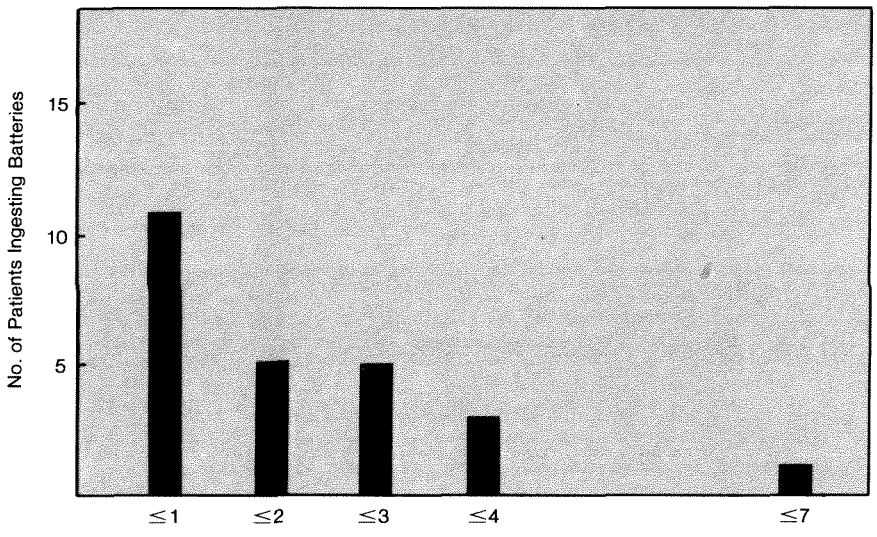

Fig 1.—Transit time of batteries allowed to pass through gut.

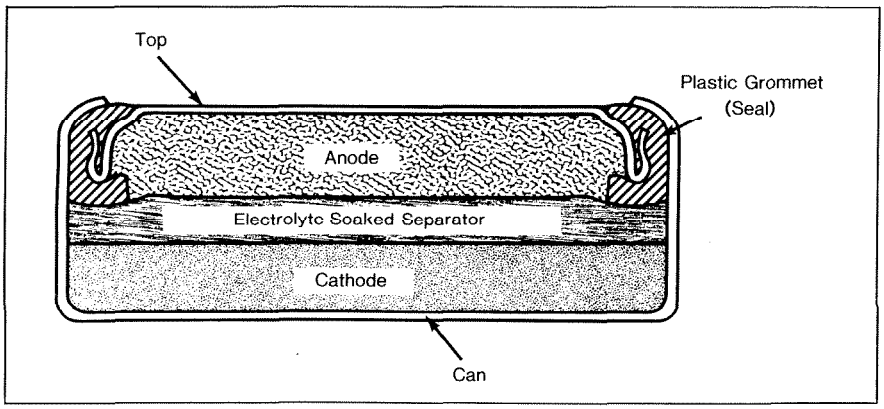

Fig 2.—Mercuric oxide button cell consists of an amalgamated powdered zinc anode, a highly compacted mercuric oxide and graphite cathode, an electrolyte-soaked felted fabric separator, a nylon grommet, a steel can coated with nickel, and a steel top coated internally with copper and externally with nickel and gold.

more, discharged button cells pose a lower risk of electrolyte leakage at the seal.

4. When placed in a conducting medium (such as the electrolyte-rich fluids of the gastrointestinal tract), the button battery may give rise to a current external to the battery, generated by the electrical potential between the cathode and anode (opposite sides of the can). It is possible that this current passes through body tissues, causing cumulative electrical injury in the setting of an impacted battery.

5. Pressure necrosis from any impacted foreign body may cause perforation of either the esophagus or Meckel's diverticulum. The medical literature contains reports of 86 cases in which nonbattery esophageal foreign bodies (including coins) caused aortoesophageal fistulae and deaths.[12,13] This may be a contributing factor but is not the sole mechanism, as liquefaction necrosis has been described at autopsy.

6. The most likely mechanism of injury is multifactorial. Peak external electrochemical current density would be expected at the point with the shortest distance between anode and cathode. This site is the plastic seal separating the anode and cathode. As an external electrochemical current develops, corrosion progresses, allowing increased electrolyte leakage at the seal. (The clinical observation of maximal corrosion at this site in batteries that have passed supports this theory.) The electrochemical current is maximal in the acid medium of the stomach, thus, maximal corrosion likely occurs here.

CONCLUSION

This study clearly demonstrates that most ingested button batteries can be safely allowed to pass through the gastrointestinal tract without invasive intervention. The patient at risk of severe complications is readily identified by an initial chest roentgenogram demonstrating an esophageal position or by the unlikely subsequent development of severe abdominal symptoms (pain, tenderness, guarding).

Case reports and anecdotes introduce a bias toward more sensational, and therefore severe, cases. While this retrospective study of patients ingesting button batteries produces a more balanced portrait of this subject, to further confirm these conclusions, a prospective investigation, the National Button Battery Ingestion Study, has been under way since March 1982. Please report all button battery ingestions to the National Capital Poison Center 24-hour emergency line at 202-625-3333. Call collect.

Case reports and technical information were provided by many sources. Raymond L. Balfour of Ray-O-Vac, Samuel R. Converse of Union Carbide, the Consumer Product Safety Commission, and many poison centers cooperated in this study. Kenneth L. Dretchen, PhD, provided statistical assistance.

References

1. National Poison Center Network Computer System for Documenting Data on Poison Exposures. Pittsburgh, 1981.
2. *Packaged Power.* Bethel, Conn, Duracell Products Co, 1979.
3. Blatnik BS, Toohill RJ, Lehman RH: Fatal complications from an alkaline battery foreign body in the esophagus. *Ann Otol* 1977;86:611-615.
4. Shabino CL, Feinberg AN: Esophageal perforation secondary to alkaline battery ingestion. *JACEP* 1979;8:360-362.
5. Barros EA, Barros AAB: Mercury battery ingestion. *Br Med J* 1979;1:1218.
6. Reilly DT: Mercury battery ingestion. *Br Med J* 1979;1:859.
7. Votteler TP: Warning: Ingested disc batteries. *Tex Med J* 1981;77:7.
8. Willis GA, Ho WC: Perforation of Meckel's diverticulum by an alkaline hearing aid battery. *Can Med Assoc J* 1982;126:497-498.
9. Temple AR, Veltri JC: One year's experience in a regional poison control center: The Intermountain Regional Poison Control Center. *Clin Toxicol* 1978;12:277-289.
10. Easom JM: Risks from swallowing small alkaline batteries. *Pediatric Alert* 1982;7:21-22.
11. Kulig K, Rumack CM, Rumack BH, et al: Disk battery ingestion: Elevated urine mercury levels and enema removal of battery fragments. *JAMA* 1983;249:2502-2504.
12. Nandi P, Ong GB: Foreign body in the esophagus: Review of 2,394 cases. *Br J Surg* 1978;65:5-9.
13. Vella EE, Booth PJ: Foreign body in the esophagus. *Br Med J* 1965;2:1042.