Disintegration of mercury disc cells in simulated gastric juice: implications for management of disc cell ingestion

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SUMMARY

The disintegration of charged alkaline mercury button cells in simulated gastric fluid over a 24 h period has been studied. The integrity of the cells and the amount of mercury that can leak out of them were assessed. The cells raised the pH of the incubating solutions. Disruption was seen in five out of 18 cells tested in 0.1 mol/l hydrochloric acid and one out of nine in 0.9% saline. Five of the six disrupted cells were made by the same manufacturer. Major leakage of mercury only occurred after complete disintegration of the cells.

The implications of these findings for the management of patients who have ingested mercury-containing button cells are discussed.

INTRODUCTION

The incidence of button or disc cell ingestion is quite common and probably increasing; a significant number of cases involve children with hearing impairments (Litovitz, 1985). The U.K. Department of Health issue 16 million hearing aid calls per year, of which 8–10 million contain mercury (Department of Health, personal communication, 1987). Moreover, a large number of consumer electrical products also contain this type of cell. In the follow-up of 100 out of 180 cases of disc cell ingestion reported to the U.K. National Poisons Information Unit in one year, the type of cell ingested was known in only 49% of cases, and the state of charge was largely unknown (Mant, personal communication, 1987). The incidence of such cases in any one hospital in the

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UK is, however, relatively low so that likelihood of any one surgical team managing such a problem more than once is uncommon.

A single cell may contain from 0.09 to 21 g of mercuric oxide depending on manufacture and type (Temple & McNeese, 1983). The reported fatal oral dose of mercuric oxide is 1 g (Aronow, 1983), but there is evidence that gastrointestinal absorption is incomplete (Ellenhorn & Barceloux, 1988). Moreover, it is uncertain what proportion of mercuric oxide is converted to the more soluble and toxic form (mercuric, Hg^{2+}) or to less toxic elemental mercury.

There are a large number of case reports of the morbidity and mortality of mercury disc cell ingestion. Liquefaction necrosis of oesophageal tissue by these cells with a fatal outcome, has been reported (Reilly, 1979; Votteler *et al.*, 1983); and it is widely agreed that cells that are lodged in the oesophagus should be removed urgently. If a cell has passed beyond the oesophagus, a 12–24 h period of close observation is advised (Mant *et al.*, 1987). The consensus of opinion is that a conservative approach to management is preferable and indeed there have been warnings against 'alarming and inappropriate surgical intervention' (Litovitz, 1985; David & Ferguson, 1986). However, removal of a cell should be considered after 24 h if it remains in the stomach.

The effect of gastric acidity on cells available in the USA has been studied with *in vitro* models (Votteler *et al.*, 1983 and Litovitz *et al.*, 1984), and in dogs (Litovitz *et al.*, 1984). Litovitz *et al.* (1984) also demonstrated an absence of 'crimp area dissolution' (i.e. disruption of the metal can) in discharged cells and studied the beneficial effects of using antacids on the degree of 'crimp area dissolution'. The state of charge of ingested cells appears to have an important influence on the degree of damage caused, fully charged cells having the greatest effect. It is still not clear, however, what local caustic effect the cells can have. Fusion to the gastric mucosa with acute ulceration has been reported (Votteler *et al.*, 1983) and is probably related to the inherent electrical charge. In addition, the integrity of the cells in relation to the rate of mercury release during a non-interventional phase of management has not previously been investigated.

MATERIALS AND METHODS

An *in vitro* model was established to simulate the gastric environment in order to investigate changes in pH and mercury released from charged mercury-containing button-cells commonly available in the UK. The cells that were tested were unused and taken directly from the suppliers' packaging, thus representing the potential for greatest harm. A simulated gastric acid concentration of 0.1 mol/L HCl (pH 1–2) (Ganong, 1985) was used as a model of a resting gastric state without the bulk and buffering power of food residue.

Random samples of three of the most common brands of CP1 mercury-containing hearing-aid cells which are issued by the U.K. Department of Health (Duracell^R, Rayovac^R and Ucar^R) were used. Three cells of each brand were tested in each of the following three solutions:- (1) 0.9% saline, (2) 0.1 mol/L HCl, pH 1.5, (3) 0.1 mol/L HCl, pH 1.5, back-titrated with 1 mol/L HCl to pH 1.5 if hourly pH measurement revealed a rise in pH over the first 6 h. Thus, a total of 27 cells were tested.

Each cell was immersed in 75 mL of dilute acid or saline solution in an individual glass beaker and continually stirred at room temperature. The pH of each solution was measured at hourly intervals over a period of 6 h using a portable probe electrode pH-meter. Visible disintegration of the cell casing (resembling that which may be seen radiographically and endoscopically *in vivo*) was recorded. After the first 6 h, for practical reasons, the solutions were left unstirred to react with the cells for a further 10 h. The cells were removed after a total exposure of 24 h.

Aliquots (2 mL) of the incubation medium were taken from each of the beakers at 0, 2, 4, 6 and 24 h and stored at 4°C for subsequent determination of mercury concentration. These aliquots were fully dissolved in 5 mL concentrated hydrochloric acid (AristaR; BDH Ltd., Poole, Dorset). Further dilutions were required for some of the samples that were found to contain high concentrations of mercury. The assays were carried out using atomic absorption spectrometry (IL 751, Thermoelectron, Ltd. Warrington, UK) with a mercury 'cold vapour' technique. This technique consisted of a continuous flow hydride generator (PS Analytical, Orpington, Kent) using a 1% (W/V) solution of sodium borohydride (GPR grade; BDH Ltd) dissolved in 0.1 M sodium hydroxide (AnalaR; BDH Ltd.) and 5 mol/L hydrochloric acid. Calibration of the technique for mercury analysis was achieved using mercuric nitrate (Spectrosol; BDH Ltd.). The total weight of mercury released from each cell was calculated from the concentration of mercury found in each of the solutions.

RESULTS

The pH measurements and state of disintegration of the cells in the various solutions are recorded in Table 1. When the cells were incubated in either 0.9% saline or in 0.1 mol/L HCl, the pH increased over the course of the 24 h in each case, although the changes were minimized where back-titration was performed. In the latter case, only the 24 h samples exhibited an appreciable rise in pH.

Five out of 27 cells had released mercury into solution by 6 h, of which four had disrupted. The fifth cell had disintegrated at 24 h. One cell disintegrated at the moment of removal but no mercury was detected in the sample of incubating solution.

Six of the 27 cells (22%) had disrupted by 24 h, of which five had been immersed in acid and one in 0.9% saline. Five of the six cells carried the same brand label (Duracell®). The results of the mercury analysis of the supernatants are shown in Table 1.

DISCUSSION

When the problem of disc cell ingestion was first recognised, an aggressive approach to management was advocated (Reilly, 1979; Votteler *et al.*, 1983). The adoption of a more conservative policy was suggested by David & Ferguson (1986) but reports in the literature indicate that aggressive policies continue to be pursued (Fernando, 1987).

Table 1 Change in mean pH, cell disruption frequency and mercury release following immersion of mercury cells in simulated gastric juice

Cell	1	2	3	4	5	6	24 hours
<u>(0.9% saline pH 7.2)</u>							
Duracell	7.4	7.4	7.7	7.8	8.5	8.3*	11.4*** ^{1/3}
Rayovac	6.9	8.4	8.9	8.9	8.1	9.2	10.5
Ucar	6.8	8.4	7.7	8.3	8.3	8.2	7.8
<u>(0.1M HCl-back titrated to pH 1.0-1.5)</u>							
Duracell	1.5	1.6	1.7	1.7*	1.9** ^{1/3}	1.6** ^{1/3}	2.2** ^{1/3}
Rayovac	1.5	1.5	1.6	1.7	1.7	1.6	2.0
Ucar	1.4	1.6	1.7	1.8	1.6	1.6	2.3 ^{1/3}
<u>(0.1M HCl pH 1.0)</u>							
Duracell	1.4	1.4	1.6	1.9** ^{1/3}	2.2** ^{1/3}	2.5** ^{3/3}	5.0** ^{3/3}
Rayovac	1.4	1.6	1.6	1.8	1.9	1.9	2.7
Ucar	1.4	1.5	1.6	1.8	1.9	1.9	2.5

Mercury release

* = < 1 mg Hg

** = < 100 mg Hg

*** = > 100 mg Hg

■ = Cell disruption

2/3 = Two of three cells disrupted.

The U.K. National Poisons Information Service currently advises non-intervantional observation for up to 24 h. This advice is based on the follow-up data in 100 cases (Mant, personal communication, 1987) and other reports in the literature where, in most cases, cell ingestions resulted in minimal morbidity (Litovitz, 1985).

During the period after ingestion of an alkaline mercury-containing disc cell, the cell will tend to raise the pH in its locality. A high concentration of hydroxide ions from the cell and an electrolytic current between the terminals can be corrosive and cause liquefaction necrosis of tissue (Blatnik *et al.*, 1977; Shabino & Feinberg, 1979; Ito *et al.*, 1985; Fernando, 1989). Therefore, cells known to be lodged in the oesophagus should be removed. Fortunately, reports of visceral perforation by cells beyond the oesophagus are quite rare (Litovitz, 1985), probably because of the protective actions of mucus, acidity and fluid food contents in the stomach. However, over a prolonged time period (e.g. 24 h), gastric acidity may cause disruption of the cell if it remains in the stomach.

The management of some ingested North American brands of mercury disc cells located in the stomach has been studied (Litovitz *et al.*, 1984; Litovitz, 1985). It seems that ipecac or gag-induced emesis is not effective and whole-gut lavage is probably unnecessary. Indeed, the results of the present study suggest that lavage is contraindicated since cell disruption was seen in 0.9% saline. Furthermore, the electrolytic flux around a cell in 0.9% saline may be increased and hence lead to further tissue damage.

Litovitz *et al.*, (1984) reported that *in vitro* and *in vivo* (in dogs), eight out of nine commonly used antacids reduced the degree of cell disruption. However, the administration of a 'neutralizing' dose of antacid may prove impracticable in clinical practice, particularly in children. The use of H₂-blockers was reported not to be effective in dogs and indeed the marked reduction in hydrogen ion production may promote caustic injury by the cell. Increased gastric emptying with metoclopramide was also ineffective in dogs (Litovitz *et al.*, 1984).

In the present study, incubation with hydrochloric acid at a concentration which simulated the gastric pH, caused the disruption of five out of 18 cells, with subsequent leakage of mercury. Furthermore, it was found that one out of nine cells in 0.9% saline also underwent disintegration. It appears that if a cell is liable to disintegrate, then this may occur between 4 to 6 h after ingestion.

The results did not indicate a gradual leakage of mercury over the time course of the experiment, but rather a sudden release following cell disruption. There is good evidence that the cell casing itself may confer some protection from the formation of the more toxic, soluble mercuric chloride (Barber & Menke, 1984). Iron (Fe^{2+}), dissolved from the casing, may reduce the mercuric oxide content of the cell to elemental mercury in the presence of gastric acid. However, the case for therapeutic administration of ferrous preparations is uncertain.

The mercury content of these cells is high enough to give cause for concern if it is released into the upper gastrointestinal tract (Mant *et al.*, 1987). The present study demonstrated an overall 22% disruption rate, with inter-product variation, which increased to 28% in an acid environment. It is therefore impending cell disruption (Figure 1) that has to be monitored using a combination of clinical observation, radiography and endoscopy. The U.K. National Poisons Unit recommends intervention if there are significant symptoms (Mant *et al.*, 1987). But reported symptoms following button cell ingestion are generally mild (Table 2) and not necessarily indicative of mercury toxicity. If the cell remains in the stomach for over 12 h, removal using a magnet and balloon catheter under fluoroscopy may be considered (Ito *et al.*, 1985; Jaffe & Corneli, 1984). Endoscopic retrieval was unsuccessful in 10 of a series of 15 patients reported by Litovitz *et al.* (1984). Surgical intervention should be deferred to 24 h unless symptoms develop. Similarly, if there are signs of corrosion (haematemesis and melaena), or there is radiographic or endoscopic evidence of cell disruption, prompt removal is indicated.

Table 2 Reported signs and symptoms following disc cell ingestion (Adapted from Mant, personal communication)

(1) Anorexia	(6) Mild pyrexia
(2) Vomiting	(7) Stool discoloration
(3) Diarrhoea	(8) Melaena
(4) Abdominal pain	(9) Haematemesis
(5) Lassitude	

The U.K. Department of Trade and Industry (Consumer Safety Unit) has encouraged the battery industry to improve battery packaging and include warnings on the packets (DTI, 1987 personal communication). Even in the present simple study, the surprising variability in reaction to gastric-type acidity suggests that greater attention to design and construction is indicated and that cells could be made more robust. One particular brand of cell (Duracell[®]) performed poorly, but the present sample size is too small to draw firm conclusions regarding interbatch and interbrand variability in disintegration.

Fig. 1 Mercury button cells showing relative size and stages of disruption;
(a) new cell and 5 pence coin (23 mm diameter)
(b) early cell disruption
(c) advanced cell disruption with contents

Fig. 2 Flow-chart for the recommended management of disc cell ingestion.

Our recommendations for the management of mercury cell ingestion are summarized in Figure 2. If the mercury disc cell is in the oesophagus, it should be removed immediately. If the cell has reached the stomach, the management is conservative but the patient should be observed closely as an in-patient. Blood or urine measurements may be used to monitor mercury absorption and excretion, but blood mercury is a better index of acute exposure. We emphasize the importance of taking urine and blood samples at the time of presentation. These should be stored in case there is a subsequent need to monitor mercury absorption and excretion, particularly if there is evidence of

mercury leakage at surgical intervention. Close observation for up to 24 h will confirm whether safe passage of the cell beyond the pylorus or lodgement in the stomach has occurred. In the latter situation, removal should be considered, since fusion of the cell to the gastric mucosa may have taken place. Impending disruption may become evident as early as 4 h after ingestion which might necessitate urgent removal.

Once the cell is beyond the stomach safe passage through the gastrointestinal tract can be expected. However, cathartics may be useful at this stage. If mercury-cell disruption occurs at these levels, close monitoring of symptoms and of blood and urinary mercury concentrations will give indications for intervention.

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