# Potentially lethal! Which of the mechanisms represented in the press is the cause of the damaging effects of swallowing button batteries?

*Edward Azurdia, Matthew O'Hare, William Reay and Daniel Walker*

ABSTRACT After seeing several reports in newspapers making claims about the cause of death after children had swallowed button batteries, a team of students decided to try to establish which (if any) claims were true. The findings were reported in the Schools Exhibition at the ASE Annual Conference 2018 in Liverpool and are shared in detail here.

## Context

Over Christmas in 2016, 2-year-old Brianna Florer died after she had swallowed a button battery. It was widely reported in the American press that she had developed a short-lived fever, vomited blood and turned blue. She was rushed to an Oklahoma hospital but, after 2 hours in the operating theatre, could not be saved. The cause of death was reported as a range of reasons including choking, burning, rupturing the carotid artery, release of the toxic battery contents, and electrolytic reactions. In the period from 2005 to 2014, 11940 children in the USA were reported to have been hurt by button batteries, with 15 dying and another 101 experiencing major medical problems.

Upon reading such articles, we realised that some of them were littered with scientific inaccuracies and contradictions, so we were keen to investigate further.

As indicated above, there is a difference of opinion as to what could have caused the deaths. The main factors reported by the newspapers included:

- death from choking;
- burning;
- a burst artery;
- capillary bleeding;
- poisoning;
- electrolytic effects.

We considered the possibility of death from choking, but as the battery was lodging in the oesophagus and not the trachea the possibility of choking was unlikely, especially given the time it took for the symptoms to be visible.

Our research started by looking into what type of battery was being ingested and considering the breakdown of the actual battery:

- We found that it was mainly lithium batteries that were causing the deaths.


**Figure 1** The presenting team: Edward Azurdia, Matthew O'Hare, William Reay and Daniel Walker of Birkenhead School on the Wirral Peninsula near Liverpool

- We also found that the battery had to get lodged to prove fatal, as otherwise the battery would pass through the body in a few days.

We first researched whether a burst artery was possible. The carotid artery is too far away from the oesophagus (Figure 2), and the battery would have to go through a large amount of body tissue to get to it, so this possibility was ruled out.

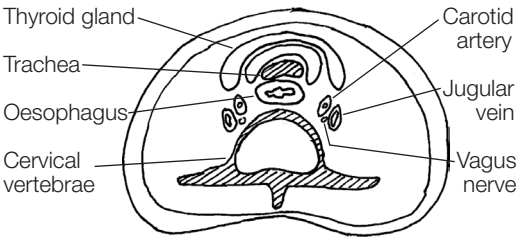


**Figure 2** The position of the carotid artery relative to the oesophagus

## Breakdown of the battery

Our first idea was that, following the battery becoming lodged in the child's throat, it would disintegrate into its constitutent parts (Figure 3) and perhaps these components would be the cause of damage in the oesophagus of the victims.

We then looked into whether there were chemicals in the battery that could have caused the deaths. Perhaps lithium in the batteries could have been absorbed by the body, leading to problems with the heart. A simple flame test on the batteries' contents produced a strong crimson flame, suggesting the presence of lithium, and the damage it may cause seemed worthy of investigation.

Our theory was that the lithium ions were interrupting nerve communication – as the vagus nerve is fairly close to the oesophagus, we suspected signals between the nerve and the heart may be interrupted.

When the nerve is resting, the axoplasm inside the axon has a high concentration of potassium ions and a low concentration of sodium ions, whereas the fluid outside the axon has a low concentration of potassium ions and a high concentration of sodium ions (Figure 4). Electrochemical gradients are maintained by active transport of ions against their concentration gradients, in specific regions known as sodium pumps.

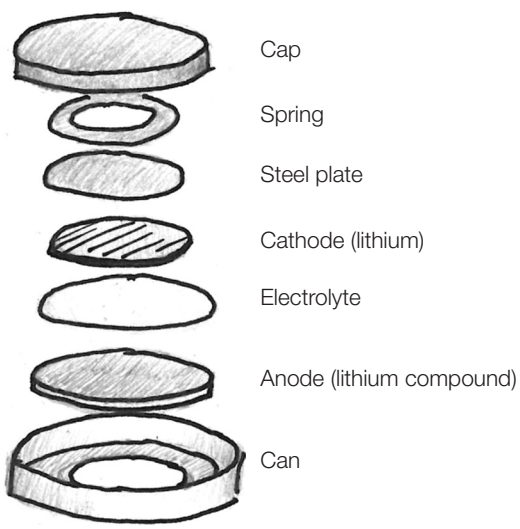


**Figure 3** An 'exploded' view of a button battery

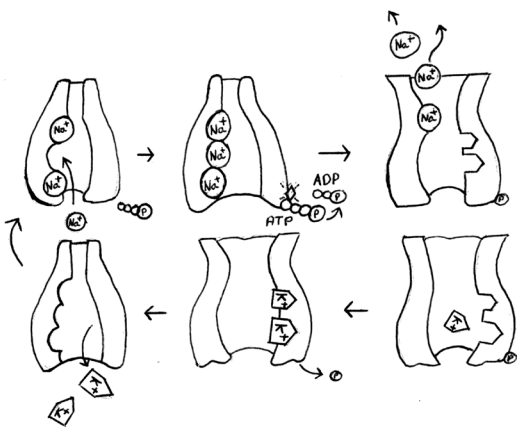


**Figure 4** The sodium/potassium pump, which may have been interupted by lithium ions

Nerve impulses are caused by a sudden momentary increase in the permeability of the axon membrane to sodium ions which enter the axon. This increase in sodium conductance increases the number of positive ions inside the axon, reversing the polarity between inside and outside the axon. This is known as an action potential.

Lithium ions replace sodium ions in the sodium/potassium pump mechanism. This was discovered by Keynes and Swan in 1959. It was also discovered in 1963 by Armett and Richie that lithium ions enter the nerve cell at approximately the same rate as sodium ions but are not efficiently pumped out. Therefore, restoring the resting potential is slow. This could affect the

vagus nerve and lead to greatly accelerated heart rates and heart attacks.

The lithium ions, which are smaller in size but are in the same periodic group as sodium and potassium and thus have similar chemical properties, could be drawn into the axon by facilitated diffusion as opposed to the sodium ions. This causes a disruption in the potential difference produced because of the action potential. The change in potential difference either sends the wrong nerve impulses, or none as the altered impulse does not cross a synapse.

Once we had proved the presence of lithium ions, we sought to test whether the lithium would actually affect the heart. To do this, we carried out tests on daphnia, a small river-based animal in which it is possible to view the heart as it beats using a microscope (Figure 5).

We altered the concentration of lithium ions in various solutions in which the daphnia were placed and measured their heart rates. The results are shown in Figure 6.

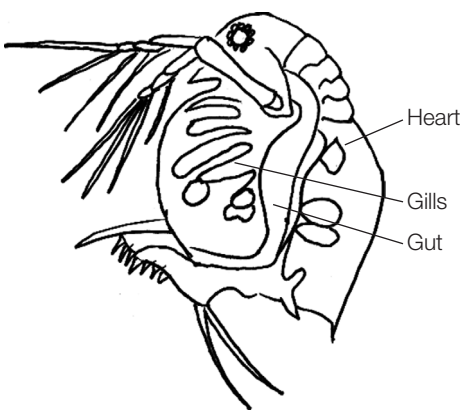


**Figure 5** A daphnia showing the position of the heart

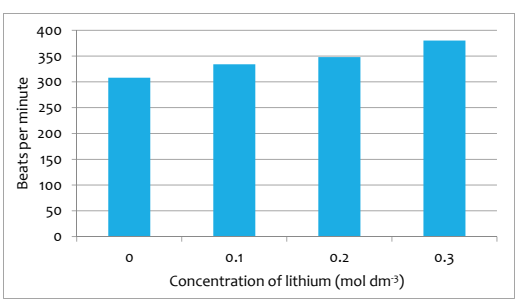


**Figure 6** The effect of lithium ion concentration on the heart rate of a daphnia

It could be concluded that an increase in the concentration of lithium ions can affect the functioning of the heart. Therefore, if they can be released, lithium ions may be the cause of death owing to cardiac arrest, but the affect is relatively small and is unlikely.

### Other substances within the battery

Inside the battery we also found a black powder. We tested the powder to see what it was and found that it catalysed the breakdown of hydrogen peroxide into water and oxygen (Figure 7). The equation for this reaction is:

$$2H_2O_2(l) \rightarrow 2H_2O(g) + O_2(g)$$

This experiment therefore suggests that the substance present was manganese dioxide.

Manganese dioxide and lithium are used in the battery with manganese dioxide as the positive terminal and lithium as the negative terminal. The equations for the reactions are as follows:

Positive reaction: $MnO_2 + Li^+ + e^- \rightarrow MnOOLi$

Negative reaction: $Li \rightarrow Li^+ + e^-$

Total reaction: $MnO_2 + Li \rightarrow MnOOLi$

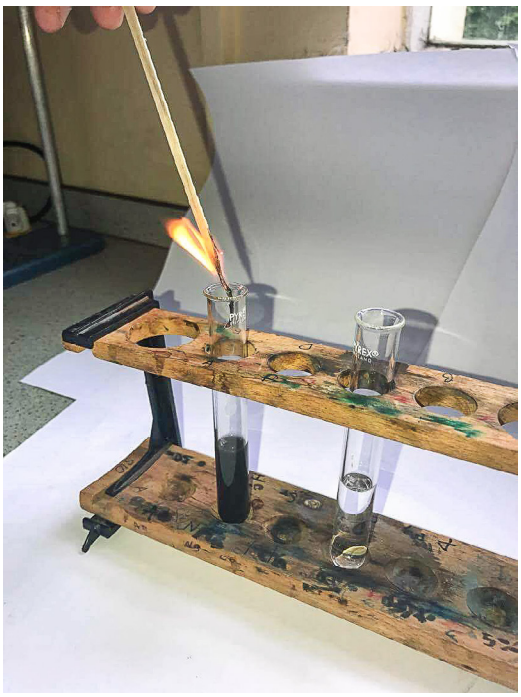

**Figure 7** Confirming the presence of oxygen after adding hydrogen peroxide to the black powder

Manganese dioxide contains manganese ions and manganese is a toxic trace element. This means that, while it is vital for humans to survive, if too high a concentration is present in the body it can be very toxic. For children, the maximum intake range is anywhere from 2 to 9 mg (see *Bibliography*). Yet even if these values are exceeded, the symptoms of toxicity do not explain the extreme bleeding.

However, for the lithium ions or the manganese oxide to be released from the battery, it must be broken down by some part of the body. The expected and most probable cause of the breakdown would be acid from the stomach meeting the battery and corroding it. To test this, we placed the whole battery in 2 mol $dm^{-3}$ hydrochloric acid – we observed no visible reaction. Very similar results were observed with concentrated hydrochloric acid though some very slight colouration of the acid was present after 24 hours (Figure 8). This was probably an electrolytic effect as the battery remained unscathed. To check this, the battery was manually broken apart and the casing only was placed in a solution of concentrated hydrochloric and concentrated nitric acid. This combination of acids is known to be highly corrosive to metals but again no visible reaction occurred (Figure 9).


**Figure 9** No damage was caused to the batteries even by concentrated acids

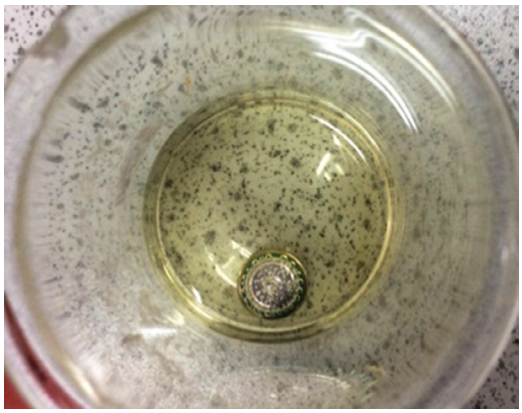

**Figure 8** Only slight colouration of the concentrated hydrochloric acid occurred after 24 hours

Therefore, we could conclude that the batteries' stainless steel casing would not break down in the lower concentrations of acid found in the stomach.

## Electrolytic effect

If the structure of the battery was not leading to the cause of death, then it could possibly be the current generated by the battery in the oesophagus that could be the cause of the symptoms. We were convinced that a current would be created as, in the oesophagus, as there is a saline-like solution that surrounds the battery within the mucus that lines the inside of the oesophagus.

The first effect of the formation of the current would be electrolysis of the electrolyte, forming products at the terminals that could be creating harmful effects. In the electrolysis of saline solution, the chloride ions and the hydroxide ions are attracted to the anode and the hydrogen and sodium ions are attracted to the cathode as shown in Figure 10. This electrolysis leaves an excess of hydroxide ions and sodium ions, creating a solution of sodium hydroxide.

This formation of sodium hydroxide (caustic soda) led us to the theory that the corrosive effects of caustic soda were destroying the cells of the oesophagus, allowing a bleed to occur. Furthermore, the collection of the chloride ions at the anode creates chlorine gas that is also very harmful to the body.

To prove the presence of chlorine gas, hydrogen and sodium hydroxide, we placed a battery into saline and collected the gases produced (Figure 11). The gas that was collected

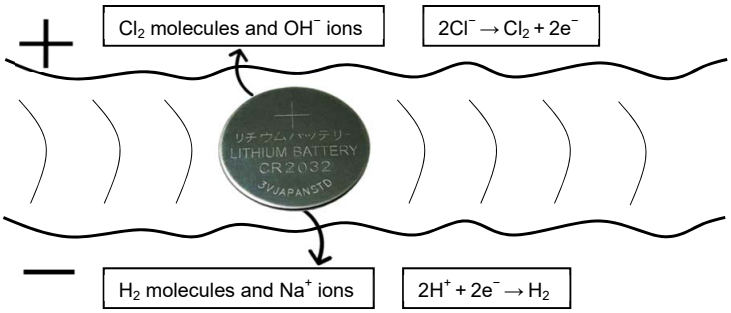


**Figure 10** Electrolytic reactions around the battery within the oesophagus

at the cathode gave a positive test for hydrogen as it exploded when we placed a naked flame near it. The gas that was collected at the anode was chlorine as it bleached damp litmus paper white.

Finally, we placed universal indicator in the solution that was left behind after the electrolysis, and it gave a blue colour (Figure 12), suggesting that an alkali (NaOH) had been produced.

**Figure 11** The electrolysis of saline to collect the products

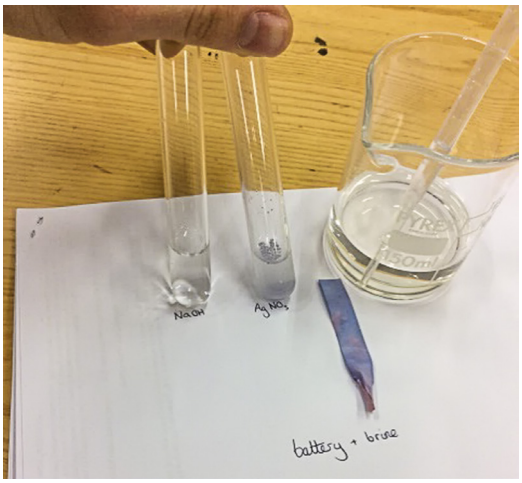


**Figure 12** The alkaline solution resulting from the electrolysis of saline

## Effect on pH

Our experiment run on the electrolysis of saline was to test how the pH around the battery changes over time using a pH meter in order to prove our previous suggestion of caustic soda formation. We ran the test at two different temperatures. The results are shown in Table 1 and Figure 13.

**Table 1** How the pH of saline changed around a battery over time

| Time (min) | pH at room temperature | pH at body temperature |
|---|---|---|
| 0 | 8.2 | 9.8 |
| 0.2 | 9.6 | 10.1 |
| 1.2 | 10.2 | 10.7 |
| 2 | 11.3 | 11.4 |
| 3 | 10.9 | 12.0 |
| 4 | 11.3 | 11.8 |

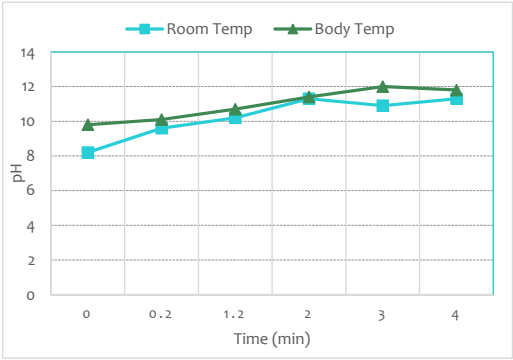


**Figure 13** Graph of pH against time for the saline solution around a battery

The experiment was left to run for several hours and pH values of 11–12 were maintained.

It should be noted, to fully comprehend the graph in Figure 13, that pH is a logarithmic scale such that a 1 unit increase in pH is a 10-fold increase in $OH^-$ concentration.

The results show that there is immediate electrolysis and production of corrosive sodium hydroxide. However, it must be recognised that the concentration of $OH^-$ is greater at body temperature than at room temperature. This suggests that an increased temperature will simply exacerbate the situation when a battery is swallowed. Furthermore, this experiment showed us that the battery in the body does not go flat quickly as we could take readings over many hours. Therefore, the battery has enough time to cause serious damage.

From here we began to investigate what exactly the formation of these chemicals did to the surrounding cells and tissues of the oesophagus.

The mucus, which is secreted by glands in the oesophagus for protection and lubrication along the oesophagus wall, is rich in glycoproteins and water. Therefore, we deduced that the battery may have the opportunity to hydrolyse the mucus owing to the creation of an excess of $OH^-$ ions from the caustic soda.

The hydrolysis of the bonds connecting the sugar molecules to the protein chain would result in the breakdown of the mucus layer, allowing the battery to damage the oesophageal wall.

We were keen to establish whether the $OH^-$ ions generated by the battery would be capable of hydrolysing the glycoprotein structure of mucus that protects the oesophagus and then go further to hydrolyse the phospholipids of the cells lining the oesophagus (Figure 14). The results in Table 2 show that the glycoprotein structure of mucus is destroyed by $OH^-$ ions.

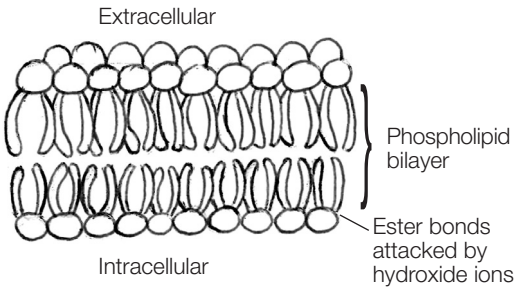


**Figure 14** The structure of the cell membrane and the bond between the phosphate, glycerol and fatty acids that is attacked by hydroxide ions

We were also aware that the button batteries would be in contact with tissue present in the oesophagus.

We tested the batteries directly on various samples of flesh, including preserved and unpreserved bacon as well as actual porcine oesophageal tissue from a slaughterhouse.

**Table 2** Tests on mucus

| Test | | Result | Inference |
|---|---|---|---|
| 1 | Benedict's test<br>2 $cm^3$ mucus + 2 $cm^3$ Benedict's solution in a water bath at 50 °C | No visible reaction | No reducing sugars released from glycoprotein chain |
| 2 | As (1) above but with the addition of 2 $cm^3$ of 0.1 mol $dm^{-3}$ NaOH(aq) | Blue solution formed a red/brown precipitate | Reducing sugar released from glycoprotein chain therefore mucus has been destroyed |
| 3 | Biuret test<br>2 $cm^3$ mucus + 2 $cm^3$ Biuret solution in water bath at 50 °C | Blue solution went to a purple solution | Little additional evidence as the test gave a positive with the intact glycoprotein |
| 4 | As (3) above but with the addition of 2 $cm^3$ 0.1 mol $dm^{-3}$ NaOH(aq) | Blue solution went to a purple solution | As above |
| 5 | Molisch test<br>sulfuric acid + phenol | No visible reaction | The test was used as a confirmation of Benedict's |
| 6 | As (5) above but with 2 $cm^3$ of 0.1 mol $dm^{-3}$ NaOH(aq) | Yellow darkening to red at interface | It works with non-reducing sugars in the unlikely event that the hydrolysis of mucus released these sugars only |

We were able to improve our experiments by enclosing the tissue around the battery as well as by adding solutions that would be present inside the oesophagus, such as mucus and saline.

We observed damage to the meat and carried out tests to see whether any of these potential products could have any excessively damaging effect on the person's oesophagus and surrounding tissue or organs.

The meat that we tested, in the presence of a battery submerged in saline solution, produced a variety of unexpected outcomes. The meat on both sides of the battery discoloured, as products were green, black and white. We discovered that the green colour was not reproduced when using unpreserved meats. We also observed that more damage was caused to the meat in contact with the positive side of the battery. This could have been due to the chlorine gas formation due to the electrolysis of the saline solution. The chlorine gas is very damaging to cell membranes and can cause oxidation. Within the oesophagus, the cells will be ruptured and leak their contents. Again, the chlorine could be contributing to the bleeds that may occur in the oesophagus.

Additionally, we suspected the sodium hydroxide was causing further problems with regard to the cells in the oesophagus. Sodium hydroxide has the effect of breaking up the cell membrane of cells within the oesophagus. The phospholipids that exist in the cell membrane are the molecules that are attacked by the sodium hydroxide. A reaction called alkaline hydrolysis occurs whereby the carbonyl carbon (carbon with a double oxygen bond) undergoes a nucleophilic acyl substitution reaction, which specifically involves the hydroxide ions – the mechanism is shown in Figure 15.

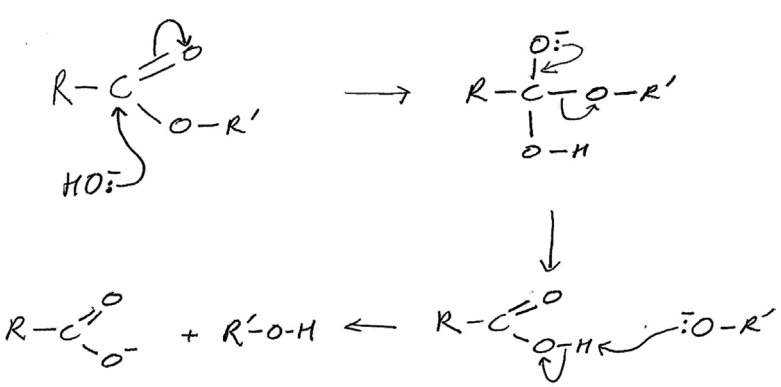

**Figure 15** The mechanism for the alkali attack on an ester bond

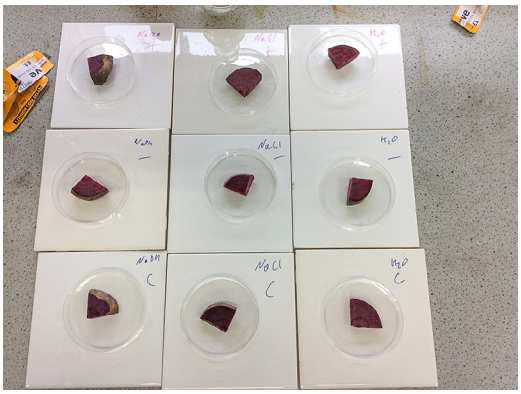

**Figure 16** Beetroot in contact with test solutions and batteries

As a result, the phospholipids are broken apart to form glycerol plus a phosphate group and two fatty acids. The cell membrane is therefore disintegrated and all the contents of the cell is released. This reaction can also break open capillaries and larger blood vessels to cause bleeding in the oesophagus.

Another consideration is that the charged phosphate head ($PO_4^{3-}$) may be attracted or repelled, depending on the terminal of the battery. This could potentially rupture further cell membranes.

Our first experiment to explore this theory involved placing pieces of beetroot into Petri dishes (Figure 16) and adding substances to observe whether any pigment was leaked (cell membranes were broken). The beetroot piece after being cut was dried using paper towels to avoid any pigment being leaked into the solution that wasn't actually as a result of the chemical being tested. We tested sodium hydroxide in the presence of beetroot. The pigment leaked out in each case. Using a colorimeter, we quantified the darkness of the solution produced by pigment leakage, as shown in Table 3. Initially, we tested sodium hydroxide, sodium chloride and water with the beetroot, with both positive and negative sides of the battery. We also carried

**Table 3** Colorimeter results for the damage done to beetroot cell membranes

| Substance added to beetroot | % transmittance* |
|---|---|
| NaOH (positive terminal contact) | 57.8 |
| NaOH (negative terminal contact) | 62.5 |
| NaOH (no battery present) | 50.1 |
| NaCl (positive terminal contact) | 58.0 |
| NaCl (negative terminal contact) | 68.0 |
| NaCl (no battery present) | 87.0 |
| $H_2O$ (positive terminal contact) | 94.0 |
| $H_2O$ (negative terminal contact) | 96.5 |
| $H_2O$ (no battery present) | 93.5 |

Note: A lower % transmittance means a darker colour (due to pigment leakage).
*A few drops of concentrated HCl were added in each case to prevent any pH-related colour changes.

out control tests for each solution without the battery at all, to observe what pigment leakage occurred without the battery, as well as a control test with water. We also considered the fact that beetroot cells have cells walls; however, they are very permeable and wouldn't affect the results.

A preliminary experiment was carried out and we realised that the beetroot pigment was an indicator and the alkaline solutions turned it green as the pigment was released, somewhat distorting the results. We then decided it would be appropriate to add an acid to the alkaline solution and decided to add dilute HCl that neutralised the NaOH, preventing the colour change associated with the indicator pigment. This led to dilution factors, making comparisons difficult. Instead, we decided to add a few drops of concentrated HCl, which didn't significantly affect the dilution of the resulting purple colouration.

This experiment demonstrated that sodium hydroxide did break cell membranes because the purple red pigment inside the beetroot cells was released. This would also occur to the cells inside the oesophagus.

We found that, in general, the positive side of the battery did more damage than the negative side of the battery, as less light passed through the solution – it was darker as more pigment had been released as more cells were damaged to release the beetroot pigment. We also found that the damage was caused mainly by the sodium hydroxide because less light was transmitted when sodium hydroxide was used in comparison with sodium chloride without the battery.

At this point, we were confident that capillary bed bleed was the cause of death, as the cells lining the oesophagus where being ripped apart, and this was why it was not possible to stitch up the patients to stop the bleeding. To make our final judgement valid, we carried out a set of experiments to prove that the experiments that we carried out were valid under the conditions in the oesophagus.

## Tests on preserved meat

Tests on preserved meat samples showed a green precipitate deposited around the battery site after a few minutes of the battery being in contact with the meat (Figure 17). The test we carried out involved placing batteries on unpreserved meat with 2 $cm^3$ of saline solution.

We hypothesised that the green substance around the battery site was perhaps the green precipitate formed when ions reacts with hydroxide ions. This would therefore seem to suggest that the battery actually damages the cells and reacts with the $Fe^{2+}$ in the haem group of the myoglobin in the cells.

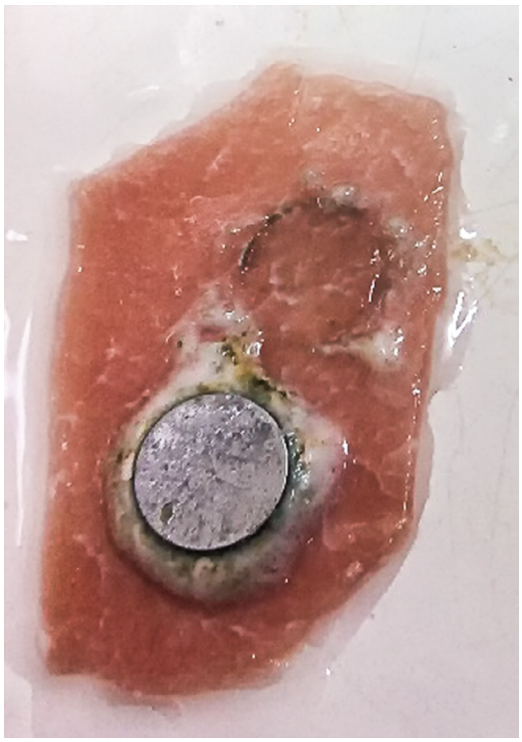

**Figure 17** The damage caused to preserved meat by the battery

## Tests on unpreserved meat

In order to see whether this was true, we placed batteries in 2 $cm^3$ of saline solution on unpreserved meat. The same green colour was not formed; therefore, we can conclude that the preservatives were probably the cause of the formation of the green precipitate. We also got a coloured solution when the battery was placed in saline, suggesting that some of the transition elements alloyed in the stainless steel casing may be released during the electrolytic process.

However, a black colour was achieved around the rim of the batteries. This could be charring and therefore carbon or, sticking with the hypothesis that the battery damages haem subgroups, could be iron(II) oxide. The black deposit was collected and added to hydrochloric acid.

$$FeO + 2HCl \rightarrow FeCl_2 + H_2O$$

NaOH was then added; a positive result would have been a green precipitate indicating that the acid had broken down FeO into $Fe^{2+}$ ions. However, the test result was negative and therefore we can conclude that the damage to the meat, was caused by heat created by the battery and the black substance around the battery is indeed carbon charring.

## Validity of our theory

One of our concerns was focused around whether our results would be valid at the temperature of the body, yet we were cautious that heating up a button battery cell too rapidly may cause it to explode. Therefore, we used a Daniell cell to investigate the effect of temperature on cell potential.

In the Daniell cell, a piece of zinc metal is placed in a solution of zinc sulphate (0.1 mol $dm^{-3}$) in one container acting as the anode, and a piece of copper metal is placed in a solution of copper(II) sulphate (1.0 mol $dm^{-3}$) in another container as the cathode. The salt bridge contains a concentrated solution of KCl that allows the ions to flow, allowing the battery to function. We tested this cell by altering the temperature of the solutions and recording the voltage of the cell using a voltmeter (Figure 18).

At room temperature the voltage of the battery was 1.01 V but at 35 °C the voltage was 1.03 V. We felt that such a small change in voltage was not sufficient in being able to come to any conclusion of the effect of temperature on the cell potential of the battery. However, it did suggest that there is no such significant change to the potential of the battery at the required temperature that would make us question our findings elsewhere at room temperature.

At room temperature the theoretical potential of the Daniell cell is given by:

$$Cu^{2+} + 2e^- \rightarrow Cu \qquad E = +0.34\,V$$

$$Zn^{2+} + 2e^- \rightarrow Zn \qquad E = -0.76\,V$$

$$\text{Difference} = 1.10\,V$$

In order to support this proposition, we looked to investigate the Nernst equation. The Nernst equation allowed us to determine the potential of a cell (the amount of voltage that exists between two half cells of a battery) without being under standard conditions. The equation is given as

$$E = E^{\ominus} - (RT/nF) \ln Q_r$$

where:

- $E$ is the cell potential (V);
- $E^{\ominus}$ is the standard cell potential;
- $R$ is the universal gas constant, $R = 8.31\,J\,K^{-1}\,mol^{-1}$;

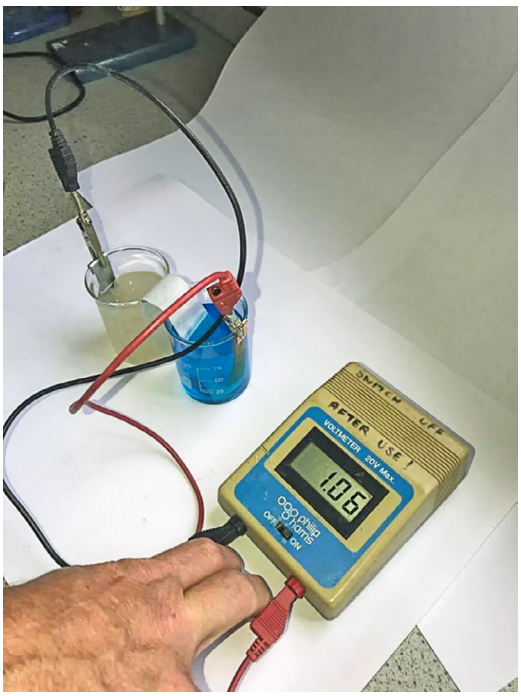


**Figure 18** Voltage created by a Daniell cell

- $T$ is the temperature in kelvin;
- $F$ is the Faraday constant, the number of coulombs per mole of electrons, $F = 700496485339900000♠9.65 \times 10^4\,\mathrm{C\,mol^{-1}}$;
- $n$ is the number of electrons transferred in the cell reaction;
- $Q_r$ is the reaction quotient.

At 25 °C, then, the equation produces:

$$E = 1.1 - \frac{8.31 \times 298}{2 \times 9.65 \times 10^4} \ln \frac{0.1}{10} = 1.159\,\mathrm{V}$$

and at 37 °C:

$$E = 1.1 - \frac{8.31 \times 310}{2 \times 9.65 \times 10^4} \ln \frac{0.1}{10} = 1.161\,\mathrm{V}$$

These results support the idea that, although one may conceive a change in temperature to decrease the cell potential, the change that occurs due to the rise in temperature is far too subtle to allow any contradictions in our findings at room temperature, as the potential of the battery will have only changed so little with an increase in temperature by 12 °C. This allowed us to confidently say that the harmful effects caused by the electrolysis in the oesophagus when swallowed, and the biological effects this may have, will be as serious when in the body as it is outside the body.

## Conclusion

Throughout our project we have attempted to either prove or disprove theories put forward by the media as to the cause of death of the victims. The first and most unscientifically based theory was that children were dying due to suffocation – this was quickly disproven as it was discovered that the battery was lodged in the oesophagus for a considerable time and could not have compromised the airways. Another theory was that the battery was able to break open in the stomach, yet we were able to disprove this theory by placing the battery in concentrated acids, which did not lead to the battery opening; it was not ruptured at all, so we could therefore conclude that that the cause of death must have originated from something else.

As we reconsidered the causes, it became clear that the effects of electrolysis may have been the precursor to death of the children. We began to investigate whether the sodium hydroxide produced could have reached the arteries present in the neck. This is incorrect as the arteries are simply too far away from the oesophageal wall even in an infant's body.

Multiple victims were said to have lost their lives by heart attack. However, electrolysis by the button battery was the most serious damage caused by the battery in the oesophagus. Therefore, the heart attacks may have resulted from shock and blood loss, and if this was not by an artery burst it must have been from elsewhere. After testing how the electrolysis affected the tissue and mucus in the oesophagus, we came to our final conclusion that electrolysis damages the near-lying capillary bed (which is impossible to stitch), leading to fatal levels of blood loss that were responsible for the shock and heart attack.

### Acknowledgements

The artwork was done by Archie Finter and Annie Robertson, year 10 students at Birkenhead School. The supporting teachers were Mike Hayward, Sharon Tharme and Kelvin Britton and the supporting technician was Barbara Moore.

### Bibliography

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