

Button battery induced oesophageal lesions: how and when?

Presented to the Faculty of
Mathematics and Life Sciences
Department of Biomedical Engineering
and
University Medical Centre Groningen
Division of Paediatric Cardiac Surgery
and
Department of Otorhinolaryngology

University of Groningen

In partial fulfilment of the
requirement for the degree of the
Bachelor Life, Science and Technology: Biomedical Technology

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Groningen, The Netherlands
10th July 2015

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INTRODUCTION

Foreign body ingestion is a common problem mostly occurring in children from 6 months to 3 years of age^{1,2}. Although button battery ingestion accounts for less than 2% of all foreign body ingestions², they are known to cause severe tissue damage. In the period 1982-2009, 6.3 to 15.1 cases per million button battery ingestions were reported in the US¹. Nowadays, on average 10.8 per million button batteries are ingested per year^{3,4}, although many cases are never reported⁴. However, only a minimum amount of button batteries lodge in the oesophagus, this group forms the greatest risk^{1,2}. There were 40,400 battery related injuries treated in 1997-2010, in children under 13 years of age⁵. Children aged 1 – 2 years old have largest chance of battery ingestion with oesophageal impaction, due to a smaller diameter oesophagus than in older children⁵.

Over the last 30 years the NBIHⁱ has analysed more than 14,000 button battery ingestions to identify factors contributing to severe outcomes and refining clinical protocols⁴. The NBIH registered a rise in severe cases around 2006, which continued to grow for the next 7 years⁴. The number of major injury or fatal outcomes increased fivefold in 2006-2012 compared to two decades before in 1986-1992⁴.

In households, button batteries are increasingly used for all kinds of gadgets. Gadgets are made more and more attractive to people, so also to children, and as most gadgets are easily accessible, an increasing amount of button battery cases is not a strange consequence⁶. Ingested batteries are usually retrieved from hearing aids (44.6%), watches, games and toys, calculators, cameras, key chains, clocks and remote controls^{5,7}.

Oesophageal lesions are particularly dangerous because of the proximity to major vessels and organs including the heart³. One in 1000 button battery ingestions can cause serious injuries, complications and even death^{2,3,8}. Especially in the oesophagus severe injuries can be formed. 12.9% of button battery ingested cases in 1992⁹ and in 2013 8% of 3366 battery ingestions lead to adverse events¹⁰. It is recognized in the past 30 years that oesophageal impaction can be associated with most severe morbidity¹⁰. Between March 2008 and March 2011, 13 deaths and 73 major outcomes were reported^{2,10}. However, to date 30 fatalities are registered; all occurring in children aged 4 years or younger⁴.

SIZE

The chance of severe injury of an ingested button battery is very dependent on the size. Most severe injuries are caused by 15 mm – 23 mm button batteries⁷. Although they account for 3% of all ingested batteries⁵, usually 20 mm button batteries tend to lodge in children's oesophagus^{1,5,8,11}. The CR2032 and CR2025 button batteries are specifically related to major and fatal ingestions because of their common household use^{8,10,12}. These button batteries have local impact on the oesophagus^{8,13}.

LITHIUM-ION BUTTON BATTERIES

In 1982-1992, only 0.4% of all button battery cases were lithium-ion batteries⁷, but in 2008 lithium batteries involved in 24% of the cases⁸. Already then it was determined that lithium and manganese dioxide button batteries >15mm lodged themselves in the oesophagus and could cause severe damage⁷. Increasingly

ⁱ National Battery Ingestion Hotline

frequent, devastating complications resulting from button battery ingestions are associated with 20 mm lithium batteries, which are related to clinically more significant outcomes than batteries with other chemistries⁸. Mercuric and manganese dioxide batteries dropped from common use after 1992⁸. Virtually all currently marketed large diameter button batteries in households are lithium cells⁸. Lithium batteries are determined to contain mildly irritating organic electrolyte instead of alkaline electrolyte, so leakage does not play a large role in production of the injuries in these cases⁸. Lithium batteries have 3V instead of 1.5V which means they have a higher capacitance and generate more current resulting in more generation of hydroxide, in a more rapid pace, which makes them more dangerous than alkali batteries as necrosis develops sooner^{5, 6, 10, 14}. The most frequently used button batteries in this category are, in descending order, CR2032, CR2025 and CR2016⁴. Nonetheless, the mucosal damage caused by lithium batteries is similar to alkaline damage¹⁴.

SYMPTOMS

Usually batteries lodge in the upper/cervical oesophagus, 90%, similar to the location of coin ingestions^{1, 2, 5}. Symptoms to determine battery ingestion in the oesophagus are variable². Children can be asymptomatic, 36%, or sustained damage with fatal outcome^{2, 6, 8}. Most cases, 56%, present with dysphagia or odynophagia as main symptom^{1, 5}. There are more non-specific symptoms like irritability, vomiting and coughing¹. Damage such as oesophageal burns, oesophageal stenosis, oesophageal perforation, mediastinitis, oesophageal oedema, aortic oesophageal perforation, spondylodiscitis, tracheo-oesophageal fistula and bilateral vocal cord paralyses can be caused by button battery ingestion when ingested or after removal^{1, 2, 9, 10}. When vascular injury leads to uncontrollable haemorrhage it results in death⁵.

CLASSIFICATION OF OESPHAGEAL INJURY

The oesophagus is a 10 - 12 cm tube at birth and can grow up to 30 cm in an adult. It consists of three layers: mucosa, submucosa and a muscular layer¹⁵. The mucosa, the innermost layer, is made up of squamous epithelium, lamina propria, and muscularis mucosa¹⁵. Fibrous connective tissue with blood vessels, nerves, and numerous mucous glands are found in the submucosa¹⁵. The muscular layer has two layers; an inner and outer¹⁵. The mucosal layers are not strongly attached to each other; a lot of movement is possible between them. The anatomy of the oesophagus is important because the staging system for injury is based on what is seen with endoscopy which correlates with the depth of injury^{15, 16}. The system is used for acidic and alkali lesions^{15, 16}.

Injury staging in the oesophagus^{15, 16}:

Grade 0	Normal mucosa
Grade 1 (mucosa)	Oedema, hyperaemia of mucosa
Grade 2a (transmucosal)	Blisters, haemorrhages, erosions, whitish membranes, exudates
Grade 2b	Grade 2a plus deep or circumferential ulceration
Grade 3a (transmural)	Small scattered area of ulceration and areas of necrosis
Grade 3b	Extensive necrosis

The oesophageal mucosa is bathed by swallowed saliva and submucosal gland mucins¹⁷. These mucins mainly include acidic mucin and bicarbonate¹⁷. Saliva is made up of 99% water¹⁸. The other 1% consists of electrolytes, mucus, glycoproteins, enzymes and antibacterial substances¹⁸. The electrolytes are sodium,

bicarbonate, chloride and potassium¹⁸. These concentrations of electrolytes in saliva can increase up to 135 mEq/L, 80 mEq/L, 70 mEq/L and 40 mEq/L respectively¹⁸.

CLINICAL TIMEPATH

Severe injuries are found even in the early diagnosed cases, although injuries tend to worsen as time goes by¹. Time until diagnosis can vary from 2 hours to 29 days¹. But even a few hours of ingestion can result into a major complication². Within 2-2.5 hours after ingestion significant injury in mucosa can be determined in the form of severe burns, oesophageal corrosive injury, stenosis, or bilateral vocal cord paralysis^{2,8,9,10}. Oesophageal perforation can occur within 5 - 6 hours in some cases^{2,12} but can also occur after 20 hours¹¹. After 9.5 hours the burn noted to be around 1 cm², when it is not removed and spreads during 24 hours of ingestion⁷. In 33% of the patient's dysphagia were not determined until more than 1 week after ingestion⁵. Tracheo-oesophageal fistulas become symptomatic within 9 days after removal of the button battery and fistulisation in an artery took 18 days after removal⁸. Tracheo-oesophageal fistula, mediastinitis, perforation of the aorta, oesophageal oedema and oesophageal stenosis can still occur a few months after removal². Oesophageal lesions can result into oesophageal dilations for up to 5 years⁵. Unfortunately, only a little more than 66% of children are diagnosed correctly. Reasons for delay include: history ingestion unclear, symptoms not specific or level of awareness low⁵.

THE FIVE MECHANISMS

The level of button battery injury depends on several factors such as location, duration exposure, remaining voltage and chemical composition⁶. Four mechanisms, occurring after button battery ingestion, are distinguished known to cause lesions; direct pressure, toxic substance absorption, leakage and electrolyte circuit^{1,2,5,19}.

PRESSURE

Direct pressure alone, which causes necrosis of various tissues, in an oesophageal button battery case proved to not be a factor responsible for the necrosis in the tissue⁶.

TOXIC SUBSTANCE ABSORPTION

A important factor which tends to burn tissues is toxic substance absorption¹⁹. This happens mostly in mercury battery cases when mercury is released^{1,2} in the form of mercuric oxide⁵ which is absorbed by the tissue. However, lithium-ion batteries do not cause toxicity, according to Yardeni D. et al, because lithium is a heavy metal⁵. Nevertheless, this argument is dubious as, for example, mercury is also a heavy metal and very toxic as mentioned before. Lithium toxicity from battery leakage has not widely been reported, although there are a few cases concerning this problem. The reason that not much is known about lithium toxicity leakage from batteries is that toxic effects can reveal themselves after many years.

LEAKAGE

In alkali batteries, leakage⁵ of alkaline electrolyte solution tends to be the main cause for penetrating tissues and producing liquefying necrosis which resembles skin burn, due to a strong exothermal reaction^{1, 2, 13, 19}. Although to cause a burn in the oesophagus, leakage of active ingredients is not a necessity⁵. In lithium button batteries leakage is not a great threat, probably because the quality of the button batteries is better.

ELECTROLYTE CIRCUIT

MECHANISM OF A BUTTON BATTERY

A lithium button battery requires three main components: a positive and negative electrode and a substance separating them, the electrolyte. When a battery is connected to a circuit, a chemical reaction takes place causing ions to go one way and the electrons the other, into the outer circuit, producing a current. In batteries, paradoxically, the positive terminal is called the cathode and the negative terminal is called the anode. However, in electrolysis and with X-rays, electricity passes through a chemical to split it up. Here cathode and anode are named the other way around. In a battery the mechanism is backwards electrolysis, which is why the names are switched. The electrons in the circuit move from the negative terminal to the positive terminal. The electrolyte works like an insulator so that the electrons do not take the short cut through the battery to the other pole.

CIRCUIT IN THE OESOPHAGUS

When a button battery is lodged in the oesophagus an electrolyte circuit is formed by the current in the battery^{1, 2}. The maximum current is determined by the (remaining) voltage¹⁹. The external current discharges the battery and causes electrolysis of the tissue or mucosal fluids, generating hydroxide locally at the anode/negative pole^{5, 6, 86, 19}. The DC current also liberates potassium in the tissues which causes cell death¹². Generation of hydroxide leads to significant tissue injury and seems to be the most important mechanism causing mucosal injury^{10, 20}. To remember the dangerous side of a battery the 3 N's mnemonic is created: negative, narrow and necrotic⁸. The electrolyte circuit is formed before any possible alkali leakage starts^{5, 12}.

Most ingested batteries are partially spent or functionally dead⁷. These spent cells still have sufficient residual voltage and capacitance to generate a current and so hydroxide⁸. Nevertheless, new batteries are also ingested which are 3.2 times more likely to cause significant damage than spent batteries^{4, 8}. Completely discharged batteries however often cause no mucosal damage at all⁶.

When a button battery is short circuited a two-slope low-voltage curve is observed with a faster decrease at the beginning^{12, 19}.

THERMIC LESION

Resulting from this external current is a rise in temperature which may be involved in tissue burn¹⁹. Commercial batteries, CR2025 in DMEM, showed consistent heat production with a temperature rise to 44.3 degrees Celsius and a 2.22 K rise in 100 minutes, which is a 0.1 K/min slope^{13, 19}. When a button battery is

lodged in very restricted part of oesophagus in close contact with a small part of epithelium, in absence of heat transfer by blood vessels temperature may reach this 44.3 degrees Celsius with heavy consequences on cell functions¹⁹. pH measurements showed values up to 9.7-12.3 which means generation and release of hydroxide anions to the surroundings^{13,19}.

IN VIVO RESEARCH

Animal researches⁸, as well as human histopathological findings, reveal transmural oesophageal necrosis¹⁹. These studies suggest that duration of contact is more important than the size of impaction⁹.

In rabbit tissues button batteries were implanted in vivo⁸, here tissues, in contact with the negative pole, showed a value of pH 10.79¹⁹. In vivo rat stomach implantation showed an increase of mucosal pH which caused ulcerations and perforation of the digestive tract¹⁹. CR2032 implanted in dogs showed superficial necrosis, from the lamina propria mucosa to the inner muscular layer¹⁴ after 15 minutes¹⁹. In 30 minutes, epithelium disappeared to form black-brown area which grew larger with time¹⁴. Furthermore, within one hour necrosis extended to trachea while the button battery remained intact¹². Dog and cat studies showed necrosis of inner muscularis layer after 1-2 hours progressing to full thickness necrosis in 2-4 hours⁶ caused by electrolyte leakage from the grommet of a button battery¹⁴.

CLINICAL CASES

Many cases result in major complications because of delayed identification of the problem. For instance, a 6-year-old girl was admitted due to respiratory distress and feeding problems²¹. A radiograph was taken and she was given antibiotics²¹. Yet 24 hours later she presented with respiratory failure²¹. A second radiograph was taken which revealed two foreign bodies, i.e. button batteries. These had been mistaken for electrocardiography stickers in the first radiograph²¹. Extensive tracheo-oesophageal lacerations and fistulas were found on endoscopy²¹. The child spent a year in hospital before recovering fully²¹.

But even cases which are identified immediately damage can already be caused¹. A seven-year-old girl had ingested a 20 mm button battery and presented with sialorrhoea and vomiting¹. The battery was covered in debris and had caused a 2-cm burn along 2/3 of the oesophageal circumference¹. On the whole, 6 hours had passed before endoscopy¹. Four weeks after the event oesophageal stenosis was found. Balloon dilation had to be performed four times every four weeks¹.

CLINICAL PROTOCOL AND DETECTION

These studies confirm that early detection is important². Now it is advised to remove button batteries within 2 hours of ingestion because then injury free removal is possible⁸. However, misdiagnosis and delays still cause various severe injuries and deaths⁸. Injury can continue days or weeks after removal because of residual alkali or weakened tissues, in the case of 20 mm lithium button batteries⁸.

To determine button battery ingestion protocol states to make a radiograph. Button batteries show double-ring shadow or double density on PA view and 'step-off' on lateral view⁵, which is slightly more translucent¹, on X-ray imaging. Coin-like foreign bodies should be assumed to be a button battery until proven otherwise¹¹.

Removal of an oesophageal button battery is done by endoscopic removal with optical graspers⁴ and examination². Main procedure is via rigid oesophagoscopy¹³. Other (post-) operative treatment options such as anti-reflux therapy, fluoroscopic removal with a magnet⁴, antibiotics, steroids and oesophageal stenting has

proven to be controversial¹³. Surgical intervention is required if the button battery remains fixed to mucosa for prolonged period of time³. It is important to know the extent of the injury, location and the direction of the negative pole of the button battery to anticipate acute and delayed complications. When the negative pole is facing anteriorly in the oesophagus, it is advised to consider airway evaluation with laryngoscopy and bronchoscopy, even in the absence of airway symptoms⁴.

CURRENT SAFETY

Current safety methods include warnings on packaging and making battery compartments of gadgets less accessible³. Another insight is to create a safer battery. Different from all previous ways of preventing ingestion, this safety measurement is focused on the batteries themselves by using a coating. QTCC is an inexpensive waterproof coating that disallows current flow in gastrointestinal environment, through being insulating under a small amount of compression. However, in battery holders, the coating works as a conductor when the compression is greater than the threshold stress level. The conduction is created when the metal micro particles are compressed close enough to each other. These QTCC button batteries can even conduct their full potential when immersed in conductive fluid environments where normally current would leak¹⁰. QTCC batteries cause no mucosal damage and have no battery content leakage¹⁰. Unfortunately, this is a very new development and it will take quite some time before all button batteries are QTCC's.

OBJECTIVES OF RESEARCH

Goals of this research are to investigate the discharge characteristics and discharge rate of button batteries when short circuited and the time vs. effect of these characteristics on the porcine oesophagus in a laboratory setting. Furthermore, we investigate the exact mechanism of both battery poles on the formation of acidosis or alkalosis, the effect of these pH values in the oesophagus, and the histology of the damage in relation to exposure time and the compounds produced by the short circuit current that may be relevant to the damage produced.

Furthermore, we investigate where the injury begins and which form it takes. The brand and battery thickness might also be a contributing factor to the characteristics of the lesion. Only the size 20 mm, 3V lithium ion, most frequently sold batteries, which tend to lodge in the oesophagus, will be assessed in this research. Smaller batteries continue to pass through the intestinal tract and bigger batteries cannot be swallowed. Smaller batteries, 7.9 mm and 5.8 mm, are found located in the nasal cavity or ear canal.

METHODS

To begin with the frequently used types of 20 mm button batteries were found, varying in thickness of the button battery; CR2032, CR2025 and CR2016. To understand the influence of various brands, three different CR2032 brands were compared; Duracell, Varta and L.Drake & Zn. (Blokker own label).

According to the VARTA Microbattery GmbH Safety Data Sheet MSDS 2.001.005 (2014), the Lithium button batteries used in this research are made of 0.07 gr. lithium metal (anode, negative), organic electrolyte (consisting of lithium perchlorate, organic carbonates and ethers) and manganese dioxide (cathode, positive). The casing is made of steel, nickel and plastic.

In the oesophagus, the battery is situated in wet surroundings causing an external short-circuit. So, these different types and brands of button batteries were short-circuited. Two polished metal components were used to short-circuit the button battery. A M3500 Picotest multimeter was used to determine the current curve, range 1 A. Components were soldered to wires which linked up to the meter. Resistance of the wire was calculated to be 0.08211 Ohms. A current curve was determined for each button battery to identify the discharge rate of the short-circuited button battery until 20 mA was left and once for 23 hours.

Batteries are sometimes replaced before they are completely empty; they are considered to be functionally empty or spent. Therefore, it was also important to determine the curve of these functionally empty batteries. AR12 and AAA LR03 were measured at 1.5V, 0.9 V (functionally empty) and < 0.7 V (empty) to determine their discharge characteristics. The voltages of the batteries were measured by a battery tester; BT20.

The laboratory set-up for the porcine oesophagus – button battery experiments was included an experiment basin attached to a Julabo M water bath, with silicone tubing, which warmed up the medium to 310K and made a small fluid current through the basin. To fill the basin and water bath 9 litres of medium were used.

Once the discharge characteristics were known and the significant differences were calculated with statistics, the button batteries were tested in two porcine oesophaguses of 30 cm. The oesophagus was placed in a medium of NaCl with Ringer's lactate, ratio 2:6, at 310 K on the first day and pure NaCl on the second and third day. Ringer lactate consists of NaCl, KCl, CaCl, Na lactate with bicarbonate as lactate.

The oesophagus was cut open length wise and sewn shut in width wise every 3 cm. This created slots for the button batteries to fit in. Next 10 new VARTA CR2032 button batteries were put into the created envelopes per oesophagus, with the negative pole of the battery facing upwards. Subsequently, the oesophagus was sewn shut with one or two stiches. The VARTA CR2032 was chosen for the oesophagus experiment because they are currently the most frequently sold CR2032 batteries in the Netherlands.

On the first day ten button batteries were sewn into one complete oesophagus and placed into the 310 K medium. One envelope with one button battery was cut off from the whole oesophagus at ten-minute time intervals to be inspected up to 70 minutes of exposure. All effects of the exposure to the button battery were noted and photographed.

On the second day an envelope was opened with one hour interval including an extra timeslot at 90 minutes. The longest period a button battery remained in the oesophagus was for eight hours. However, three button batteries were exposed for 18 hours overnight to register the impact of the button batteries for a longer period.

The parts of oesophagus inspected after various exposure times were sent to the pathology lab for histology to examine the impact at cellular level.

After ten, 30 and 90 minutes the pH values of the tissue at the positive and negative poles were determined with MColorpHast non-bleeding pH 5-10 pH indicator strips.

After ten, 70 minutes and 18 hours the remaining current was measured in the button battery.

To investigate the effect of a button battery in different mediums, four button batteries were placed in each their own medium; 150 ml of NaCl, NaCl and Ringer 2:6, Ringer lactate and saliva. Reaction product components were inspected to determine the reaction between the button battery and the medium. Mass spectrum chromatography was used to define the contents on the button battery and the products in the various mediums.

To understand the specific effect of alkalosis on the lesion of the tissue, one porcine oesophagus without a button battery was placed into an alkali medium of a pH value of 10, with an osmolality 300 mmol/L.

RESULTS

The button batteries were short-circuited in air surroundings and were found not to produce a rise in temperature through all periods of measurement, including the 23 hour measurement.

Differences in button battery discharge rate between CR2032, CR2025 and CR2016 were not significantly different with a standard deviation of 0.002143. Furthermore, all batteries produced the same two slope diagram seen in earlier research for voltages¹⁹. The first ten minutes showed a steep discharge rate which levelled out to a slow discharge rate, continuing in a decreasing exponential manner.

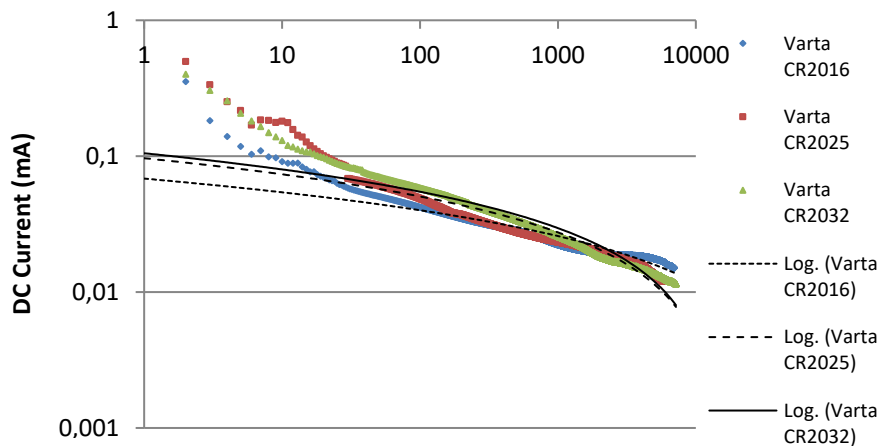
The differences in discharge rate between Duracell, VARTA and Blokker brands were insignificant with a standard deviation of 0.002166. These diagrams had the same discharge characteristics as all the batteries mentioned above. However, the VARTA button batteries had a small 2 minute bump during the initial fast discharge, the other brands did not have this characteristic.

Maximum current created in the initial short-circuit was found to be between 354 mA and 627 mA. After ten minutes the current was around 30 mA and then dropped to 20 mA in 30 minutes. Around 2.5 hours the current was approached 10 mA. In the 23-hour measurement, the last measured current was 3 mA.

Fig. 1: Current curves batteries differ in thickness

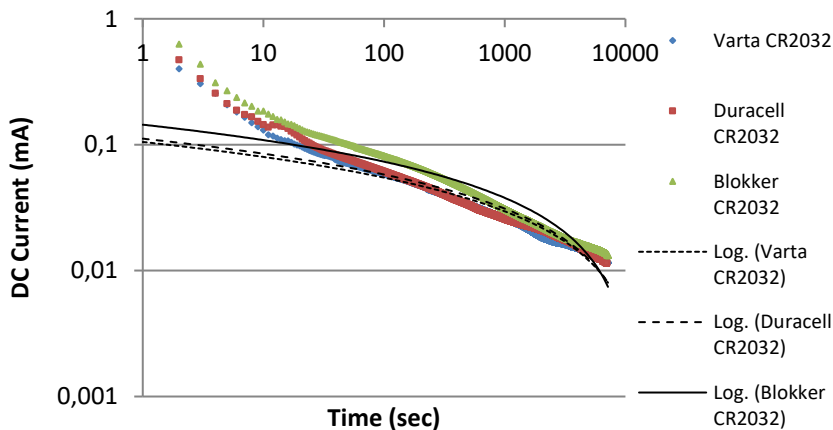
Fig. 2: Current curves batteries differ in brand.

Comparison different thickness 20 mm button batteries



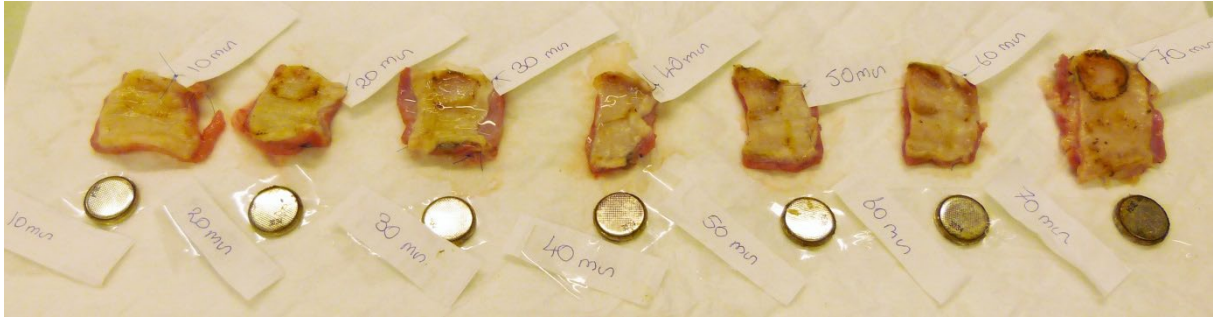
Short-circuiting the full, functionally empty and dead button batteries showed that the functionally empty batteries also had a two slope curve, comparable to the full batteries. However, the initial current was substantially lower, 35 mA, and it took two to three minutes to transfer to the second slower slope. This second slope was equal to the full batteries discharging

Comparison brands of CR2032 buttonbatteries



characteristics after a longer period of discharging. The empty batteries kept fluctuating between very low currents, around 0.0001 mA, which after some time resulted into a steady line.

Fig. 3: Oesophageal lesion with 10 minute interval



After ten minutes of button battery exposure to the oesophagus, only small patches of mucosa turned brownish on the tissue side related to negative pole of the button battery. The patches occurred at the grommet of the button battery which is where the anode and cathode join. On the tissue related to the positive terminal no damage seen on the tissue.

20 minutes of exposure showed a much clearer ring-shaped lesion with a deeper brown colouring and black spots. At this point the mucosa was much thinner, for an area of 5 mm along the ring lesion, showing the underlying muscle. This was the point where the tissue had been sewn together with one stitch.

Proceeding to 30 minutes, damaged mucosa had grown to a 15 mm lesion following the ring shape, but lesion had also grown width wise to the centre of where the button battery was situated.

At 40 minutes necrosis had formed around the whole place of the grommet, accompanied with thin mucosa which showed the underlying muscle.

Within 50 minutes a lesion had also begun at the opposite side of where the initial damage had been but also at the tissue which had been in contact with the positive pole of the button battery. A part of the muscle was also visible here. The initial damage had penetrated the muscle tissue deeper and the black necrotic spots were larger and began to clearly form the ring shape. The damage had also spread to the centre of ring-shaped lesion and all muscle was visible there too.

After 60 minutes the lesion had the same characteristics as the 50 minutes. However the lesions were less aggressive than the 50 minutes tissue.

Fig. 4: Oesophageal lesion with 1 hour interval



The oesophagus with 70 minutes exposure time had a complete ring shape of necrotic tissue. Within this ring the mucosa had disappeared nearly completely. One spot had a yellow ulceration. The tissue related to the positive pole of the button battery also had a few black-brownish spots.

The oesophagus with the remaining three button batteries was left in the set-up overnight, which resulted in a period of 18 hours. After the 18-hour exposure the mucosa had completely vanished on the tissue where the button battery was situated. The tissue on the outside was bright red and looked like it could burst. However, the muscle had not been perforated.

After placing the oesophagus with button batteries in NaCl on the second day, the tissue presented necrosis, after 60 minutes of exposure, along the whole edge where the oesophagus had been sewn shut after insertion of the button battery. The tissue related to the positive pole showed small parts of necrosis.

The 90 minutes exposed oesophagus showed a likewise result. Additionally, the ring shape displayed clearly on both sides of the oesophagus.

After 2 hours the mucosa had been thinned out, leaving a pinkish muscle visible at the sides. In the middle the most mucosa remained. Remarkable was that the tissue had seemed to contract itself around the button battery.

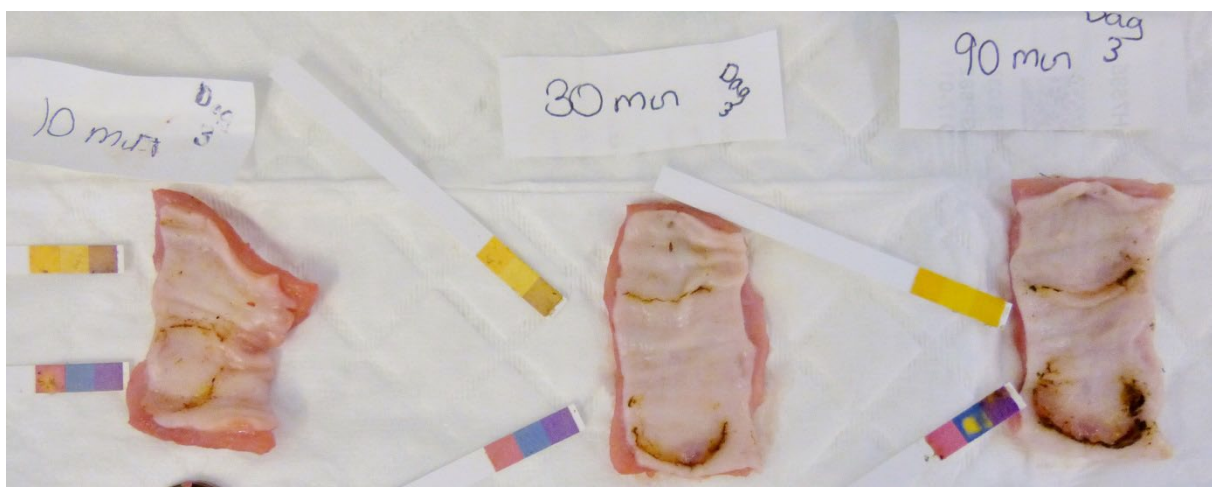
The 3 hours tissue again looked much like the 2 hours tissue, although the necrosis was much more severe. Only a tiny part of the mucosa in the centre of the tissue was still intact. The ring shape around the positive side was also completely necrotic. The edge of the oesophagus where the battery had been pushed into had been shaped around the battery and the muscle was clear to be seen.

During the next 4 hours, 4, 5, 6 and 7 hours, the necrosis only worsened on both sides of the oesophagus and more mucosa disappeared revealing the muscle, which was bright red. However, the mucosa exposed to the positive terminal stayed intact within the necrotic ring.

After 8 hours, necrosis also started within the ring of the negative side of the button battery. No necrosis was seen on the positive pole side and no perforation of the tissue occurred within the 8 hours.

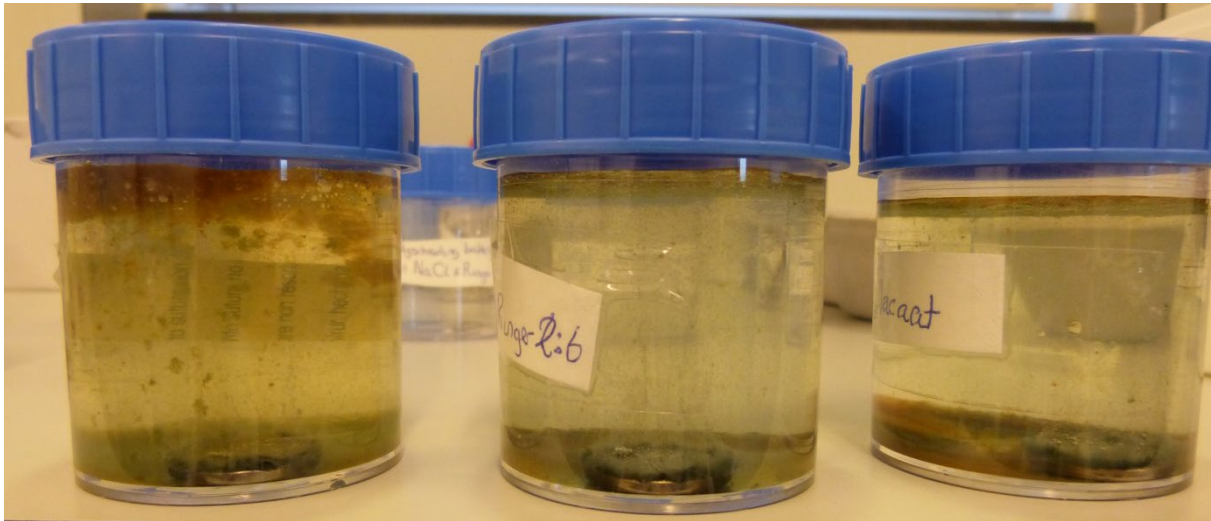
All through the experiments, corrosion on the positive terminal of the button batteries was seen. Particles, products of the reaction, oozed out of the oesophagus when incubated. The corrosion worsened on the button batteries which were exposed for a longer time period in the oesophagus, although the difference was not very big. Another remarkable observation was that gasses escaped out of the oesophagus independent of the mediums. This production of gas remained through the whole period of exposure to the button battery in the medium.

Fig. 5: Oesophageal lesion with pH values on pH indicator strips



The pH value was determined at three points in time. After 10 minutes the pH was determined to be 6 at the positive pole and 9 at the negative pole. Then at 30 minutes it was 6 and 9.5. After 90 minutes the pH values were lower than 5 and higher than 10.

Fig. 6: Button batteries in different media. From left to right: NaCl, Mix NaCl and Ringer lactate, Ringer lactate.



The investigated batteries without oesophagus in different mediums presented the following observations.

The battery in NaCl began with creating small bubbles immediately, simultaneously the grommet of the button battery became black. After 8 minutes the bubbles continued to form only on the grommet, accompanied by black greenish debris. In 25 minutes the 1 mm debris turned orange at the top. Shortly afterwards the orange debris drifted to the surface. Slowly, after another 35 minutes, the debris partially sank to the bottom of the container. The debris which sank to the bottom had a brownish orange colour.

The mix of NaCl and Ringer lactate created much larger bubbles which, after a while, got replaced by orange particles. Some floated to the surface and some sank to the bottom. Most bubbles clearly came from the grommet of the button battery, which also turned black in this case. Most spectacular was the way the bubbles and particles swirled to the surface of the medium from the battery.

The Ringer solution turned yellow immediately after the button battery was placed into the solution. Large bubbles were produced from the button battery and the debris became greenish yellow nearly instantly. This debris turned orange after a while and sank to the bottom, creating a thick, 1- 2 mm, carpet on the bottom of the container. Some orange particles stayed afloat.

Lastly, a button battery was put in 1 cm of saliva. This created black, white, green and red debris on and around the battery. Gas formation could not be identified.

The currents were measured of button batteries after being placed in the oesophagus at time points of 10, 70 minutes and 18 hours. The current was 480 mA after 10 minutes in the oesophagus. After 70 minutes the current was 340 mA and after 18 hours around 40 mA.

Unfortunately, the results regarding histology investigation, effects on the oesophagus in an alkalotic environment and mass spectrum analysis could not be acquired in time for this paper.

DISCUSSION

The objective of the study was to more specifically understand the mechanism, characteristics and time path of the damage caused by button batteries in the oesophagus. The characteristics and discharge rate of a short-circuited button battery was unknown and therefore the exact relation to the oesophageal damage.

When short-circuited the button battery shows a steep slope of discharge, followed after 10 minutes by a slower discharge rate, gradually approaching to zero. This relates to the earlier researched two-slope voltage curve. A battery is 'empty' when the current is 20mA or lower. This takes around 2 hours for all 20mm Lithium batteries¹⁹. After 6 hours the current is around 7 mA and after 23 hours 3 mA. The discharge pattern has no significant difference between different brands or thickness of the 20 mm Lithium batteries. However, the CR2016 batteries resulted in having a slightly flatter slope after the 10 minutes of fast discharging, although this was not significantly different. The VARTA button batteries had a small bump during the fast discharge, where the other brands did not have this characteristic. Being that the bump was so small, 2 minutes it was chosen to neglect this characteristic as it would not show in the experiments on swine oesophagus. According to these results it can be said that the battery type does not have to be taken into account when handling a stuck button battery in the oesophagus.

Current of button batteries that was measured after having spent time in the oesophagus showed that the discharge characteristics were very different than in dry surroundings. After 10 minutes the current was still 480 mA, compared to dry surroundings where it was 31 mA on average. The highest initial current measured in a VARTA button battery was 490 mA.

70 minutes showed a current of 330 mA, as to 15 mA. In dry surroundings the current was 3.5 mA after 18 hours of short-circuit, in the oesophagus current remained to be 20 mA.

However, the data corresponding to 18 hours of exposure in the oesophagus is not completely reliable as the water bath shut itself off during the night leaving the oesophagus in room temperature medium instead of 310 K environments.

When functionally spent cells are ingested, they are still able to create a high enough current, starting from between 30-20 mA, which was measured in spent cells of 0.9-1 V, to cause damage. This damage will be less severe and the process will be slower.

Expected was that the damage in the oesophagus would start heavily in the first 10 minutes due to high current. This was not the case as the lesion started out mildly at first and gradually became worse. Of course, the dry discharge rate does not relate to the oesophageal discharge rate where exposure to high currents lasts much longer.

Results showed that after 4 hours the damage tended to worsen in a slower rate than before. This might be related to the current having passed its peak of high currents, larger than 100 mA, which cause most damage. In dry surroundings the steep discharge slope stopped at around 300 mA and it took 2.5 hours to reach 100 mA.

The damage on the mucosa of the oesophagus in the first 10 minutes is minor but not to be neglected. In the first time-interval of an hour the damage worsens severely. The mucosa started disappearing after 20 minutes; this can be classified as a grade 1 oesophageal burn. The following hour a necrotic ring shape took place of where the battery had been and the mucosa disappeared within this ring, resulting in a grade 2 burn. After 3 hours only a tiny part of mucosa was still intact. The hours after this the damage tended to deepen into the muscle and more necrosis was seen around the edge of the place of the battery; grade 3 burn. At the opposite side of the initial lesion mild necrosis started after 8 hours.

No perforation was seen after 8 hours. Expected was, due to clinical cases, to see perforation after 18 hours. However, as the set-up could not maintain a temperature of 310 K all through the night, suspected is that the lower temperature slowed down the intensity of the lesion. The fact that literature states that no real harmful damage is done before 2 hours of ingestion is an understatement. After 20 minutes the lesion begins and after 70 minutes serious mucosal destruction has occurred.

Typical of the damage done by the button battery was the necrotic ring made evolving from the attachment of anode and cathode. This is related to the debris created on this attachment. The damage was focused on the negative side of button battery, where the mucosa disappeared. The pH values were indeed very alkaline, greater than 10. The positive side became acidic but in a much slower rate than that the negative side turned alkaline. The lesion is definitely connected to the high pH value. The high pH is caused by a hydrolytic reaction and helps the worsening of the damage. However, as the pH rises so quickly and the pH would be the only factor causing damage, the damage would continue to worsen as seen in the first hour, because that is the time needed for the pH to rise to above 10. The same results are found in two different studies showing 10.74 in rabbits at the negative pole and 9.7-12.3 in a vitro study^{13,19}. However, after 4 hours a decrease of the rate of damage is seen, which means a different mechanism, which decreases after 4 hours, must also have effect on the lesion. However, this theory needs to be tested by analysing the effect of a 10 pH 300 mmol/L solution on a piece of oesophagus without a battery.

Another surprising finding was the production of gas created by a reaction between the medium and button battery. Once this characteristic was not discovered when using DMEM medium and in another research chlorine gas was discovered²². For this research it is still unknown what kind of gas was produced in the experiment. Results of this will follow soon, subsequently leading to more knowledge about the reaction and mechanism of the button battery in oesophageal environment.

A type of corrosion was seen on the anode (+) closest to grommet of the button battery as product of the reaction. This was seen before when button batteries were tested in different mediums without esophagus.¹⁹

Certain aspects of this research are not entirely according to the mechanism in the human body. For instance, the oesophagus in the medium was place horizontally on the bottom of the experimenting basin, in the body the oesophagus is mostly situated in an upright position. This means that the produced gas would not be stuck between the battery and the tissue, as seen in this research but come up. However belching symptoms have not been reported, as this would occur with the amount of gas produced. A reason could be that the medium is not adequate to simulate the surroundings of the oesophagus in the body.

Furthermore, on two days NaCl was used and on one day NaCl and Ringer mix. The mix caused a faster severe damage of the oesophagus. This was also seen when only battery was placed in Ringer solution, the reaction occurred much quicker and more intense than in the NaCl solution or mix. Important to know is which medium best simulates the actual surroundings. As the Ringer solution contains more electrolytes that match the components of saliva and the oesophageal fluid than NaCl, the mixture medium would be preferable to simulate the human environment of the oesophagus. This means that the results gained at one-hour intervals are milder than they would be in clinical cases.

Also, the batteries which stayed in the oesophagus during 18 hours did not have a temperature of 37 degrees Celsius all night. This could be a reason why the oesophagus looked so much like the 8-hour oesophagus, as the raised temperature might speed up the reaction.

Further research could be done to determine the effect of the battery when it is placed vertically. Also, the effect of all the components of the reaction of medium with battery should be explored further to gain more understanding of the mechanism. To complete this research the results of the histology and mass spectrum analysis have to be taken in consideration. These results might change some of the conclusions made at this

point. Especially the components of the reaction between mediums and button battery are important to fully understand the mechanisms we are dealing with. In earlier research it was claimed that the discharging of the button battery produces OH^- and H_2 at the negative anode side and FeOH_3 at the positive cathode side whatever the medium¹⁹. In vitro studies showed that $2 \text{H}_2\text{O} + 2 \text{e}^- \rightarrow 2 \text{OH}^- + \text{H}_2$, which is a hydrolytic reaction, correlated clinically to substantial damage at the negative anode side of the button battery¹⁹. Maybe results of this research will conclude the same oxides, but such conclusions cannot be drawn at this time.

Other follow-up experiments would include: the characteristics of alkali lesion, the effect of pressure on the oesophagus with different alloys in coins, the effects of the thermic reaction in an oesophagus which might create a specific type of lesion and do these results also account for batteries with a different diameter and chemical composition.

Lastly it is important which measures must be made to decrease lesion and slow down the mechanisms causing damage. Most importantly regarding public awareness, what can a parent do at home to help their child?

With this research we now understand better the development of the damage caused by the 20 mm Lithium button battery in the oesophagus and hopefully, when completed, which mechanisms help such an injury evolve. The time between ingestion and damage to the oesophagus is apparently smaller than expected. The lesion will continue to grow, worsen and deepen over time.

The battery is able to create a great amount of current when ingested and the current does not discharge so easily. However, the pattern seen in dry surroundings when discharging button batteries relates to the initial fast development of lesion followed by a steady slower course of worsening lesion.

Lastly the shape of the lesion, following the ring structure in the button battery can conclude that the most dangerous part of the battery is the attachment of the anode and cathode, the grommet. Here the brownish debris causes necrosis and the mucosa is deteriorated from this edge inwards to the centre of where the battery had been.

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