ABSTRACT
Corrosive alimentary tract injuries are a source of considerable morbidity all over the world. Despite this, actual data on the epidemiology of this problem are scarce mainly due to the lack of a well-established reporting system for poisoning in most countries. The burden of the disease is naturally more in countries such as India where the condition is common because of poor regulation of sale of corrosive substances. We analyse the available data on epidemiology of corrosive injuries, as well as patterns of involvement of the alimentary tract, with special reference to Indian data, and also provide an overview of the management options and long-term sequelae of this condition.

Natl Med J India 2013;26:31–6

INTRODUCTION
Both acids and alkalis when ingested act as corrosives and produce considerable and progressive injuries to the upper gastrointestinal tract. The magnitude of the injury depends upon several factors such as the nature of the caustic agent, volume ingested, concentration, duration of mucosal exposure, age of the patient and the intent (suicidal or accidental) with which the corrosive was consumed.1–3 The outcome of adverse events due to corrosive injury is compounded in those with a pre-existing comorbid condition. Corrosive injuries are a source of morbidity all over the world, but the burden is more in developing countries.4,5 This is because corrosive agents are easily available as items of household use and are not subject to any regulatory control. Human exposure to caustic substances is usually due to accidental or suicidal injuries. The circumstances of the corrosive injury are different in paediatric and adult populations; 80% of injuries in children are accidental whereas injuries in adults are more often the result of suicidal attempts.6,7 Even though corrosive injuries are routinely encountered in medical facilities all over the world, there is not much published data on the epidemiological factors associated with corrosive upper alimentary tract injury. We attempt to identify some of these epidemiological parameters that are unique to this condition and which have a bearing on the management. The data available for this purpose are only hospital-based. Epidemiological data from the field are conspicuous by their absence. Awareness of the epidemiological features may promote active campaigning for legislation to prevent or reduce accidental corrosive ingestion as has been done in many western countries.

NATURE OF CORROSIVES
As mentioned earlier, caustic injuries may be caused by acidic or alkaline agents. The term ‘lye’ is commonly used to denote a strong alkali. The usual incriminating substances are cleaning agents (which contain sodium hydroxide), drain openers, bleaches, toilet bowel cleaners, dishwashing agents and detergents.8 Alkaline agents are usually colourless, relatively tasteless, more viscous and have a less marked odour. Hence, the amount ingested is often more with such agents. The usual mode of injury with these agents is a liquefaction necrosis resulting in tissue damage.3,5 The combination of the viscous nature of these agents and the process of liquefaction implies that these agents remain in contact with the mucosa for a longer period of time and hence are more likely to produce deeper injuries. There is, therefore, a greater likelihood of transmural injuries with lye.

Some common acid-containing agents implicated in corrosive poisoning include toilet bowl cleaners (sulphuric or hydrochloric acid), antirust compound (hydrochloric, oxalic, hydrofluoric acid), swimming pool cleaners (hydrofluoric acid), vinegar (acetic acid), formic acid used in the rubber tanning industry and other similar acids. Due to their pungent odour and unpleasant taste, acids tend to be consumed in smaller amounts, are swallowed rapidly after ingestion and may cause more gastric than oesophageal injury. The mode of tissue injury with acids is a process of coagulation necrosis. The coagulum prevents the corrosive agent from spreading transmurally and hence reduces the incidence of full thickness or peri-alimentary injury.

Mahatma Gandhi Medical College and Research Institute, Puducherry 607402, India
N. ANANTHAKRISHNAN Department of Surgery and Surgical Gastroenterology
Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry 605006, India
C.P. LAKSHMI Department of Medical Gastroenterology
RANJIT VIJAYAHARI, VIKRAM KATE Department of Surgical Gastroenterology
Correspondence to N. ANANTHAKRISHNAN; n.ananthk@gmail.com
© The National Medical Journal of India 2013
The type of corrosive agent most commonly implicated in poisonings varies from country to country. In the annual report of the American Association of Poison Control Centers (AAPCC) in 2008, the most commonly implicated corrosive agent was the alkali—sodium hypochlorite, which is contained in bleach, toilet bowl cleaners, drain cleaners and household disinfectants. Reports from Denmark, Israel, the UK, Peru, Spain, Australia, Saudi Arabia and Turkey also showed that alkaline agents were more commonly involved in corrosive poisoning. In these studies, the most common substance ingested was sodium hypochlorite which was contained in bleach or as seen in Denmark, Australia and the UK, disinfectants and cleaning solutions containing sodium hydroxide. Most corrosive substances were ingested in the liquid form but in a few cases the granules or tablets were ingested. The ingestion occurred most commonly at home and the substance was kept within reach of children in a majority of accidental ingestions by children. Child-proof packaging was not always effective in preventing older children from accessing the product.

However, Indian data are strikingly different; the majority of ingestions are due to acids as these are more easily available. These are used as toilet cleaners more commonly than caustic soda, which is more expensive. In our experience, the predominant acidic agents involved are toilet cleaning fluid (hydrochloric acid) and aqua regia or goldsmith’s solvent which is a 3:1 mixture of hydrochloric acid and nitric acid used by goldsmiths as a gold solvent. Similar reports have been published from Nigeria, where acids were involved in about 65% of corrosive ingestions, the rest being due to alkalis.

NATIONAL POISONING STATISTICS
Accurate national data regarding poisoning are difficult to obtain due to under-reporting. However, data from the USA are available from the annual reports of the AAPCC. Their first annual report in 1983 reported 22,347 cases of corrosive ingestion to the centre, and accounted for 8.9% of all reported poisonings for that year. A large proportion (82.5%) of corrosive ingestions were in children; 96.6% of all exposures were accidental and four deaths were due to poisoning. The report with statistics for 2008 documents an increase in the total number of exposures to 191,397. However, the proportion of poisoning attributed to caustic substances remains steady at 8.6%. Of these exposures, only 62.9% were in children and accounted for 8.9% of all reported poisonings for that year. A large proportion (82.5%) of corrosive ingestions were in children; 96.6% of all exposures were accidental and four deaths were due to poisoning.

The report with statistics for 2008 documents an increase in the total number of exposures to 191,397. However, the proportion of poisoning attributed to caustic substances remains steady at 8.6%. Of these exposures, only 62.9% were in children and accounted for 8.9% of all reported poisonings for that year. A large proportion (82.5%) of corrosive ingestions were in children; 96.6% of all exposures were accidental and four deaths were due to poisoning.

Unfortunately, such data are not available from India or other developing countries. A report published in 2005 provided data for 1999–2002 from the National Poisons Information Centre, All India Institute of Medical Sciences, New Delhi. Data on a total of 2,494 cases reported to the centre indicate gross under-reporting. Of the cases reported, only 3.1% (n=76) were due to caustic agents.

TIME TRENDS
As mentioned previously, data from the USA show that the proportion of poisonings attributed to corrosive ingestions has remained steady 8%–9% over the years. There is also a relative decline in the proportion of accidental injuries in children from household caustic substances especially cleaning agents. The introduction of legislation to ensure adequate safety measures such as child-proof safety caps on bottles of household cleaning products has probably led to this decline in accidental ingestions in children. Such safety precautions are not in place in many developing countries and corrosive ingestion among children continues to be common. However, data from developing countries are sparse in this regard. An Indian study compared the time trends in acute poisonings in children over a 15-year period and showed that the percentage of all poisonings attributed to corrosives has been fluctuating: 4.3% in 1993–97, 12.3% in 1998–2002 and 7.1% in 2003–08. This study also showed that, during the same period, the percentage of poisonings attributed to kerosene ingestion declined significantly whereas those due to organophosphorus compounds increased significantly.

Epidemiological Data

Other countries
Small series are available from several countries which give us data on the common corrosives used, the circumstances of injury (accidental or suicidal) and the age distribution (Table 1). However, these series are limited to a few tertiary care institutions in these countries which cater to more serious injuries and most likely do not represent the actual national statistics which would involve a large number of unreported minor household accidental ingestions. Among children, corrosive ingestions were more common in boys, attributed to their more inquisitive nature; 100% were accidental involving predominantly alkalis. Data from Denmark also show a decline in the incidence of alkaline ingestions after implementation of legislation mandating child-proof containers and adequate safety precautions. However, in other countries where such legislations do not exist, alkaline ingestions continue to occur frequently in children.

In series with predominantly adult patients, ingestions were more common in women, except for reports from Africa where men predominated. Suicidal ingestions were more common among women whereas accidental ingestion was more common among men.

Table 1. Epidemiological data on corrosive poisoning from various countries

<table>
<thead>
<tr>
<th>Author, place (year)</th>
<th>Patients (n)</th>
<th>Alkali (%)</th>
<th>Men (%)</th>
<th>Children (%)</th>
<th>Accidental (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chien et al., Taiwan (1974)</td>
<td>60</td>
<td>66.7</td>
<td>46.7</td>
<td>13.3</td>
<td>38.3 (children 100)</td>
</tr>
<tr>
<td>Christensen et al., Denmark (1994)</td>
<td>102</td>
<td>86.3</td>
<td>60</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Christensen et al., Denmark (1994)</td>
<td>75</td>
<td>65</td>
<td>43</td>
<td>Nil</td>
<td>39</td>
</tr>
<tr>
<td>Bautistas Casanovas et al., Spain (1997)</td>
<td>743</td>
<td>73</td>
<td>66.7</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Rodriguez et al., Peru (2003)</td>
<td>45</td>
<td>68.9</td>
<td>35.6</td>
<td>Nil</td>
<td>8.9</td>
</tr>
<tr>
<td>Satar et al., Turkey (2004)</td>
<td>37</td>
<td>64.9</td>
<td>43.2</td>
<td>Nil</td>
<td>86.5</td>
</tr>
<tr>
<td>Zhou et al., China (2005)</td>
<td>149</td>
<td>81.2</td>
<td>65</td>
<td>29.5</td>
<td>75.2</td>
</tr>
<tr>
<td>Arevalo-Silva et al., Israel (2006)</td>
<td>50</td>
<td>68</td>
<td>54</td>
<td>50</td>
<td>66.7 (children 100)</td>
</tr>
<tr>
<td>Cibisev et al., Spain (2007)</td>
<td>517</td>
<td>38.7</td>
<td>28.8</td>
<td>Nil</td>
<td>5.2</td>
</tr>
<tr>
<td>Thomas et al., Nigeria (2009)</td>
<td>78</td>
<td>35.9</td>
<td>78.2</td>
<td>23.1</td>
<td>88.6 (children 100)</td>
</tr>
<tr>
<td>Al-Binali et al., Saudi Arabia (2009)</td>
<td>72</td>
<td>88.9</td>
<td>53.5</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>
In the vast majority of published series, except for Nigeria\textsuperscript{18} and one paper from Spain,\textsuperscript{25} injuries were mostly due to alkaline substances.

\textbf{Indian data}

There have been few published reports from India dealing with small cohorts of patients presenting to particular centres with corrosive injury mainly highlighting the therapeutic modalities used. These studies also throw some light on the demographic profile of these patients as well as provide data (Table II) on the circumstances of poisoning (suicidal or accidental). All these series show that acids were involved more commonly than alkalis. Men were affected more than women and suicidal injury was more common than in western series. Corrosive injuries are largely unreported in India and developing countries and their prevalence cannot be extrapolated from sporadic papers. Data on national poisoning statistics, as available from the USA, are lacking in India and hence we do not have authentic data on all cases of poisoning in the country.

\textbf{Paediatric and adult corrosive injuries}

In general, corrosive injuries are common in children, and are mostly accidental. Due to their inquisitive nature, young children tend to explore household items which may include corrosive cleaning products. They accidentally ingest these substances resulting in corrosive injuries. This is especially true in developing countries where overcrowding and insanitary living conditions combined with poor regulatory control expose children to such chemicals. A recent review of data on corrosive ingestion in children from Sierra Leone estimates that most frequently injured were children below 5 years of age (80%), and more boys were injured than girls (70%).\textsuperscript{4} The most reported corrosive agent was caustic soda, followed by kerosene, sodium hypochlorite and other alkaline household chemicals. The ingestion of acid was more common in India than in other countries.\textsuperscript{7} In sub-Saharan Africa, corrosive ingestion accounts for 0.3% of paediatric admissions in the Gambia and for 0.5% in Nigeria, thus amounting to 0.84% of total childhood mortality in these countries.\textsuperscript{4} Death rates, especially in underprivileged countries, may reach as high as 12%.\textsuperscript{4}

According to the 2008 annual report of the AAPCC, corrosive ingestion ranked third among causes of poisonings in children, with cosmetics and analgesics being the first and pesticides the second leading cause of poisoning.\textsuperscript{3} Corrosive ingestion accounted for 9.7% of all poisonings in children. However, there was no mortality among children in 2008 in the USA as a result of corrosive poisoning.\textsuperscript{8}

A large Indian study among children showed that corrosive ingestion accounts for 7.6% of all cases of acute poisoning admitted to a tertiary care centre, the leading agents being kerosene, drugs and pesticides.\textsuperscript{21} A multicentric study of children showed that corrosive poisoning accounted for 1.7%–9.3% of all cases of poisoning in various centres in India ranking behind kerosene, drug and pesticide poisonings.\textsuperscript{30}

\textbf{Suicidal versus accidental corrosive injuries}

A published study from Singapore in 1955 showed that corrosive ingestion was the second most common mode of suicide, accounting for 27% of all suicidal deaths.\textsuperscript{31} Later, legislation prohibiting over-the-counter sale of caustic soda was enforced, following which there was a steady decline in the number and proportion of suicidal deaths from corrosive ingestion. However, the number of suicides have steadily increased all over the world and people with suicidal intent resort to methods other than corrosives. The same trend is seen in most developed countries. As mentioned previously, of all corrosive poisonings in the USA, only 4.5% were suicidal.\textsuperscript{8}

Indian data on suicides are not systematic due to high rates of under-reporting of attempted suicides. It is estimated that the national suicide rate for India in 2005 was 10.3/100 000 population. Tamil Nadu has a rate higher than the national average with 18.6 suicides per 100 000 population, while Puducherry has the highest rate of suicides at 52.1/100 000 population.\textsuperscript{32} Data on attempted suicides are available from Tamil Nadu as verbal autopsy reports of large population cohorts.\textsuperscript{33} These data group corrosive ingestion along with other poisons and do not provide a split up of the data to ascertain the frequency of suicidal deaths attributable to corrosive ingestion. Hence, we have to rely on data of patients admitted with corrosive poisoning to tertiary care centres (Table II), to ascertain the proportion of corrosive poisonings which are suicidal.

Some authors have shown that mucosal injury to the oesophagus is much more serious and grades 3 and 4 injuries are more frequently seen in patients who attempted suicide as compared with accidental ingestion.\textsuperscript{3} In our experience, suicidal corrosive injuries are more often associated with marked oral, oropharyngeal and proximal oesophageal injuries because of hesitant sipping of the fluid whereas accidental injuries are usually associated with ingestion of larger volumes which are gulped down fast and are associated with a higher proportion of gastric injuries. No similar reports are available in the literature.

\textbf{Frequency of gastric versus oesophageal injuries}

‘Acid licks the oesophagus and bites the stomach’ was the dictum in the past. This principle has been challenged by many authors who have shown that even with acid ingestion oesophageal injuries are common.\textsuperscript{1} It is also believed that if the injury occurs in a fasting state, gastric injuries occur in the antrum whereas if they occur in the postprandial state, the body of the stomach is affected. The largest Indian experience, reported from our centre, had 82.6% of 109 injuries secondary to acid ingestion (Table II).

\begin{table}[h]
\caption{Epidemiological data on corrosive poisoning from India}
\centering
\begin{tabular}{|l|l|l|l|l|l|}
\hline
Author, place (year) & Patients & Mean age & Males & Acid ingestion & Suicidal purpose \ (n) & (years) & (\%) & (\%) & (\%) & (\%) \\
\hline
Rao et al., Puducherry (1988)\textsuperscript{26} & 50 & 2\% children & 46.0 & 68.0 & 54.2 \\
Zargar et al., Chandigarh (1989)\textsuperscript{2} & 41 & 26.0 & 66.7 & 100.0 & 39.0 \\
Lahoti et al., Delhi (1995)\textsuperscript{27} & 21 & 21.6 & 55.0 & 50.0 & na \\
Broor et al., Delhi (1996)\textsuperscript{28} & 24 & 8.8 & 62.5 & 61.5 & 0 \\
Poddar and Thapa, Chandigarh (2001)\textsuperscript{29} & 54 & 4.9 & 77.7 & 62.9 & 0 \\
Gupta and Gupta, Chandigarh (2004)\textsuperscript{30} & 51 & 26.5 & 66.6 & 83.4 & na \\
Ananthakrishnan et al., Puducherry (2010)\textsuperscript{31} & 109 & Range: 4–65 & 55.0 & 82.6 & na \\
\hline
\end{tabular}
\end{table}

\textsuperscript{na not available}
III). The majority had prepyloric strictures (83.5%) while the remaining strictures were located in the antrum, body, pyloro-duodenal area, or were diffuse. The surgical procedure most commonly done was a Billroth I gastrectomy (77.1%). Other procedures done included loop gastrojejunostomy, distal gastrectomy with Polya reconstruction, pyloroplasty and colonic conduit jejunal anastomosis. Other Indian centres have also reported a similar spectrum of gastric injuries.37,39

### SEQUELAE OF CORROSIVE INGESTION

#### Acute sequelae

Patients with acute corrosive ingestion may develop a variety of complications such as oesophageal perforation (seen in <2% of cases), aspiration pneumonia and respiratory failure. In patients with acute corrosive ingestion, after stabilization of the patient, upper gastrointestinal (GI) endoscopy is indicated to characterize the nature of injury, if it can be done between 48 and 72 hours of ingestion. However, due to a high risk of perforation, upper GI endoscopy is not recommended between 5 and 15 days after ingestion. The findings on endoscopy can be graded as per the classification system proposed by Zargar et al.42

Broadly, corrosive injuries are endoscopically classified as grade 1, if there is only erythema and oedema. Grade 2a involves haemorrhage, erosions, blisters and ulcers with exudate, while grade 2b refers to circumferential oesophageal ulceration. Grade 3 indicates deep ulcers with brown, black and grey discoloration (3a scattered, 3b extensive) and grade 4 oesophageal perforation.42 In series from tertiary care centres, more severe injuries such as grades 3b and 2b tend to predominate, whereas in most other centres, minor injuries are more common than the severe ones.43,44 In their study, Zargar et al. found that all patients with grades 0, 1 and 2a burns recovered without sequelae. The majority of patients (71.4%) with grade 2b injury and all survivors with grade 3 injury developed oesophageal or gastric cicatrization, or both, which needed endoscopic or surgical treatment.42 A few other authors have also shown that stricture formation is most often associated with grade 2b and 3 injuries.34,45

#### Long-term sequelae of corrosive poisoning

Large series from at least two centres show that up to 7% of oesophageal carcinomas arise as long-term sequelae of corrosive ingestion, especially ingestion of lye. In these series, the interval between the caustic burn and the diagnosis of scar carcinoma was up to 46.1 years and the survival rates of these groups of patients were better than those for patients with oesophageal carcinoma due to other causes. Indian data on this condition are limited to one case report and one small series of three patients by Kochhar et al. In our experience of over 500 corrosive injuries seen over a 30-year period, only one patient, who was a smoker, developed a cricopharyngeal carcinoma following oesophageal burns by a caustic agent and one case of peri-gastroenterostomy stomal carcinoma 17 years after ingestion. There is no long-term follow-up study to clarify the incidence of malignancy in corrosive oesophageal injury; such studies are difficult to do considering that the interval between caustic ingestion and malignancy may be 25–50 years. However, it is felt that the fear of malignancy in corrosive injury has been overemphasized particularly for acid injuries. Nearly all reported cases in the literature have occurred after ingestion of lye. It is possible that in India, as the overwhelmingly common corrosive ingestant is acid this may be the reason for the low incidence of oesophageal malignancy on follow-up.

An issue of concern is the risk of malignancy in a scarred oesophagus which has been left behind after bypass surgery. Even though there are inadequate data in this regard, it is felt that the chance of developing carcinoma in a scarred oesophagus left in situ is minimal because there is no further mechanical, chemical or thermal irritation of the damaged oesophagus. Routine resection in all cases is associated with increased operative risks and is not widely advocated.40

It is also speculated whether repeated dilatations perse increase the risk of malignancy. However, there are no data to support this either. The universal policy remains that dilatation, if feasible, forms the primary recommended method of treatment of corrosive oesophageal strictures and the fact that the overwhelming majority of patients with corrosive strictures managed with dilatation do not develop oesophageal cancer indicates that the fear of malignant change in corrosive oesophageal injury is overrated. In patients who fail dilatation, it is our policy to do a bypass without resection.

#### Management of corrosive strictures: Endoscopic or surgical

The management options in corrosive oesophageal strictures may be endoscopic or surgical. Endoscopic management is in the form of dilatation, which may be done with either bougie dilators (most commonly with Savary Gilliard dilators) or with balloon dilators. The accepted protocol is for dilatation at regular intervals until a lumen size of 15 mm is achieved with complete amelioration of dysphagia. Subsequently, dilatation is repeated whenever dysphagia recurs. Several Indian centres have reported good results with endoscopic dilatation (Table IV). These data show that dilatation is effective in achieving symptom-free status in a majority of patients in whom the stricture permits endoscopic dilatation as a treatment modality. The procedure is associated with acceptable morbidity. Only a few studies have assessed the long-term follow-up of these patients, and an important issue to be considered is the recurrence rates of dysphagia. Broor et al. have studied long-term success after adequate initial dilatation in 36 patients with corrosive strictures (mean [SD] follow-up 32.36

---

### Table III. Data from Indian studies on corrosive injuries of the stomach

<table>
<thead>
<tr>
<th>Author, place (year)</th>
<th>Patients (n)</th>
<th>Isolated gastric injury (%)</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subbarao et al., Puducherry (1988)44</td>
<td>16</td>
<td>37.5</td>
<td>Surgical</td>
</tr>
<tr>
<td>Chaudhary et al., Delhi (1996)35</td>
<td>34</td>
<td>47</td>
<td>Surgical</td>
</tr>
<tr>
<td>Kaushik et al., Chandigarh (2003)38</td>
<td>10</td>
<td>50</td>
<td>Surgical</td>
</tr>
<tr>
<td>Nagi et al., Chandigarh (2004)40</td>
<td>74</td>
<td>32.4</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Agarwal et al., Lucknow (2004)37</td>
<td>28</td>
<td>54</td>
<td>Surgical</td>
</tr>
<tr>
<td>Kochhar et al., Chandigarh (2004)41</td>
<td>8</td>
<td>100</td>
<td>Endoscopic dilatation</td>
</tr>
<tr>
<td>Gupta et al., Chandigarh (2009)49</td>
<td>44</td>
<td>59.1</td>
<td>Surgical</td>
</tr>
<tr>
<td>Ananthakrishnan et al., Puducherry (2010)17</td>
<td>109</td>
<td>33</td>
<td>Surgical</td>
</tr>
<tr>
<td>Rana et al., Chandigarh (2011)49</td>
<td>7</td>
<td>100</td>
<td>Endoscopic dilatation</td>
</tr>
</tbody>
</table>
33 patients. There was no mortality and there was no instance of oesophageal strictures from the mid-ascending to the mid-descending colon in mid-colon oesophagocoloplasty using an iso-peristaltic colonic segment from the mid-ascending to the mid-descending colon in 33 patients. There was no mortality and there was no instance of colonic necrosis. The procedure restored an ability to eat normal food in 93.9% of patients. It has now been done in over 125 patients with similar results.

The other line of surgical treatment is with oesophagotomy, either transhiatal or transthoracic. Data from Chandigarh, on 51 patients, show that transhiatal oesophagectomy in corrosive ingestion is an important cause of morbidity in developing countries, where a large proportion of ingestions are suicidal. The high frequency of this poisoning in children results in considerable long-term morbidity as a result of late sequelae of poisoning especially oesophago-gastric strictures. Parents should be made aware of the need to keep household corrosives safely away from children to prevent such debilitating injuries. Also, stringent legislation is necessary in developing countries to curtail unrestricted access of adults to dangerous corrosive chemicals.

There is need for systematic reporting to generate authentic data from developing countries to create a national database which will be useful to estimate the actual public health importance and burden of corrosive poisoning and which can serve as a motivator for legislation to tightly control the availability of these chemicals and to make the packing child-proof.

REFERENCES
