T he common krait (Bungarus caeruleus, Schneider 1801) is a proteroglyphous elapid snake commonly found in Sri Lanka, Pakistan, Bangladesh, and India. The highest incidence of bites in Sri Lanka was reported from the North Central Province, where the vegetation and climate provide an ideal habitat for snakes. Common victims of B caeruleus are farmers who live in open wattle-and-daub houses and farmers sleeping in watch huts in agricultural fields. A significant number of patients die before reaching hospital. The approximate incidence of common krait bite in the late 1970s was 8.6% in Sri Lanka; the remainder were bitten by the hump nosed viper (27%), Russell's viper (17.5%), and cobra (12%).

METHODS

The prospective study included all the admissions with common krait bite to the General Hospital, Anuradhapura, Sri Lanka, from 1 January 1996 to the end of 1998. This institution is the main referral centre situated in the central dry zone of the island and has facilities for assisted ventilation and intensive care. The offending snakes were identified by either studying characteristics of the dead snake if it was produced, or showing the patient specimens of formalin preserved snakes. If both failed, clinical features and circumstantial evidences were used for arriving at the diagnosis of common krait bite. Patients were assessed at the time of admission and periodically, until the final outcome.

Clinical assessment included history and examination for neurotoxicity: ptosis, eye movements, papillary size and reaction to light, power of neck flexors and limb muscles, respiratory rate, tidal volume, chest expansion, strength of speech, level of consciousness, brain stem reflexes and cardiac status: pulse rate, blood pressure; muscle tenderness, and local effects. Further assessments included measurement of urine output, whole blood clotting test at 20 minutes, serum electrolytes, blood urea, serum creatinine, blood gases, and a 12 lead electrocardiogram. Limited necropsies were done on death. Neurophysiological tests (nerve conduction velocity of median, ulnar, sural, common peroneal, and posterior tibial nerves) were done as a routine for management of patients with late onset neuropathy. Their was regular follow up of the survivors.

Severity of neuromuscular paralysis (degree of envenom ing) was graded as mild: ptosis and external ophthalmoplegia; moderate: weakness of bulbar and neck muscles but not requiring assisted ventilation; and severe: those requiring assisted ventilation due to paralysis of respiratory muscles. Grading of muscle weakness was from grade 0–5 on the scale of the British Medical Research Council.

Alterations in level of consciousness was arbitrarily classified as normal, drowsy, semicoma, and deep coma. The grade of deep coma consisted of patients who went into a state of total unresponsiveness to deep pain and to loud command, having fixed dilated pupils and absent brain stem reflexes. The grade drowsy consisted of patients having rational motor and

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Alterations in level of consciousness was arbitrarily classified as normal, drowsy, semicoma, and deep coma. The grade of deep coma consisted of patients who went into a state of total unresponsiveness to deep pain and to loud command, having fixed dilated pupils and absent brain stem reflexes. The grade drowsy consisted of patients having rational motor and
verbal response, who remained sleepy with intermittent disorientation but were arousable. The balance belonged to the second group.

Autonomic dysfunction parameters included chemosis, lacrimation, salivation, sweating, rate and fluctuation of heart beat, and fluctuation of systolic and diastolic blood pressure. Monitoring pulse rate according to the respiratory cycle, changing position, and pharyngeal stimulation was possible. After reasonable recovery of patients, memory and higher functions were tested according to the standard recommendation.

The routine management protocol of common krait bite in hospital was adopted without modification. Initial resuscitation was the cornerstone of management and patients in severe respiratory paralysis were offered assisted ventilation. Patients with stable respiration were monitored for early neuromuscular respiratory paralysis.

Initiation of assisted ventilation was considered when tidal volume was below 200 ml, the power of neck flexors was below grade 3, there was central cyanosis, mental confusion, and failing speech. With the earliest sign of systemic envenoming, all the patients were treated with Indian polyvalent antivenom (Haffkine Bio-Pharmaceutical Corporation Ltd, Bombay, India) infusion, which is raised against four snakes including the common krait. The standard dose of 10 vials was given to all the patients and it was repeated in 36 cases. Vital parameters such as heart rate and rhythm, peripheral oxygen saturation, maintenance of fluid balance, physiotherapy, adjustment of assisted ventilation to maintain normal blood gases, early detection, and treatment of complications either by a result of envenoming or mechanical ventilation were closely monitored. Details including age, sex, socioeconomic status, time, place and site of bite, and clinical assessment, investigations, complications, outcome, and treatment were documented in a well designed pro forma. Recovered patients were followed up for late sequelae.

RESULTS
There were 210 patients with B. caeruleus bites, accounting for 9% of all snake bite admissions to the General Hospital, Anuradhapura. Envenoming was severe in 101 (48%), moderate in 38 (42%), and mild in 49 (23%) patients. Interestingly, in 22 (10%) patients there were no signs of envenoming, in spite of proved bites with definitive fang marks. Overall, in 99 (47%) situations, the offending snakes were killed and brought along with the patients and identified as common krait by the author. In the rest, other diagnostic criteria were adopted.

Socioeconomic status, time of bite, and seasonal distribution
All the patients were from poor farming families living in villages, 202 (96%) of them in cadjan thatched, wattle-and-daub houses where individuals sleep on the floor. These houses were surrounded by uncleared vegetation. Most of the bites occurred at night while the victims were sleeping on the floor except in three cases where the bite took place during the daytime on footpaths and in another in a watch hut situated in a treetop. In two thirds, the bite took place between 2200 to 0400 hours, indicating midnight preponderance.

A significant number of patients 65 (31%) had not been aware of the bite but had woken up with colicky abdominal pain. In 35 (17%) patients the site of the bite was undetectable and they presented with abdominal pain, dyspnoea, dysphagia, and signs of neuromuscular paralysis. All detectable bites were seen on exposed parts of the body: 50 (30%) on the hand, 27 (16%) foot, 20 (12%) upper arm, five neck, and three earlobe (out of 168 patients).

The maximum number of krait bites 103 (49%) occurred during the rainy season, especially in the months of September to December, during the North and East monsoon. It was our experience that most admissions of krait bites follow rainfall, even after an isolated shower during the drier months of the year. However, there were 19 admissions during the severe drought in July to August 1997.

Age and sex distribution
The sex ratio was equal and the commonly affected age group was between 10 and 30 years (52%). Envenoming was extremely severe in children below 10 years of age (12 patients). Nine patients were above the age of 60 years.

Clinical features
Abdominal pain was the first symptom to manifest with a range of minutes to a few hours. Other common clinical features were weakness of limbs, inability to stand up, drooping of eye lids, double vision, difficulty in breathing, and changing sensorium; all progressed rapidly to severe neuromuscular paralysis. Less commonly, myalgia, paraesthesia at the site of bite, decreased hearing and vision, and faintness were observed. Very often the site of the bite and fang marks were indistinct and the local reaction was faint. Bites on fingers and hands invariably produced a significant local reaction with swelling and pain (table 1). The time taken for hospital admission from the time of bite ranged from one to 20 hours; 164 patients were admitted to hospital within seven hours of the bite.

Respiratory paralysis
Of the 210 patients, 101 were ventilated, and the time lag to initiation of ventilation was 30 minutes to 50 hours with a mode of six hours. Seventy five (74%) patients needed ventilation within 10 hours of the bite. Fifty five (54%) patients developed respiratory arrest with no recordable tidal volume during ventilation and 13 (12%) of them did so within one hour of admission. Duration of ventilation ranged from 12 hours to 29 days (mode two days).

Recovery of neuromuscular paralysis was assessed by examination of muscle power and measuring tidal volume at regular intervals. Recovery of neck flexion to power grade 2–3 had significant correlation to the onset of stable recovery of respiration (r = 0.491, p<0.05); this was used as a parameter to wean patients off ventilation.

Level of consciousness
The level of consciousness was normal in 60 (29%), drowsy in 91 (43%), semi-conscious in 24 (11%), and deep coma in 35 (17%) patients. Patients in deep coma had absent brainstem and spinal reflexes; pupils remained fully dilated and light reflexes were absent. The onset of deep coma ranged from two

<table>
<thead>
<tr>
<th>Symptom/sign</th>
<th>All patients (%)</th>
<th>Severe patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnoea</td>
<td>143 (68)</td>
<td>91 (90)</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>143 (68)</td>
<td>83 (82)</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>134 (64)</td>
<td>73 (73)</td>
</tr>
<tr>
<td>Chest pain</td>
<td>109 (52)</td>
<td>60 (60)</td>
</tr>
<tr>
<td>Faintness</td>
<td>96 (46)</td>
<td>46 (46)</td>
</tr>
<tr>
<td>Giddiness</td>
<td>67 (32)</td>
<td>32 (32)</td>
</tr>
<tr>
<td>Myalgia</td>
<td>63 (30)</td>
<td>36 (36)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>33 (16)</td>
<td>18 (18)</td>
</tr>
<tr>
<td>Phosis</td>
<td>147 (70)</td>
<td>83 (82)</td>
</tr>
<tr>
<td>Weakness of limbs</td>
<td>134 (64)</td>
<td>78 (78)</td>
</tr>
<tr>
<td>Decreased consciousness</td>
<td>134 (64)</td>
<td>78 (78)</td>
</tr>
<tr>
<td>Weakness of neck flexors</td>
<td>126 (60)</td>
<td>68 (68)</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>111 (53)</td>
<td>65 (65)</td>
</tr>
<tr>
<td>Decreased respiration</td>
<td>94 (45)</td>
<td>77 (77)</td>
</tr>
<tr>
<td>Local reaction</td>
<td>63 (30)</td>
<td>18 (18)</td>
</tr>
</tbody>
</table>

*Ventilated patients.
two. Three patients developed ARDS while recovering from arrhythmia in one, and poor resuscitation before admission in seven (17%) patients died in spite of intensive care. The highest mortality rate was seen in the year 1996, during which time 7 of 184 (3.8%) patients died. Outcome in 1998 had improved: three (3.7%) died (ARDS in one and delay in resuscitation in two patients). There were no deaths in children after the availability of a paediatric intensive care unit in close proximity to the children’s ward. Overall mortality rate was 7.6%. The recovery rate of physiological functions in severely envenomed patients (n = 101) was assessed (table 3).

Eight necropsies were done on three clinical categories of cases: four cases of ARDS, one case of hypoxic brain death ventilated for more than two days, and three cases of acute death within 17 hours of the bite. In ARDS patients the lungs were congested and haemorrhagic and histology confirmed ARDS; erosions and bleeding points were seen in gastric mucosa, one case showed mottling haemorrhages in the renal cortex and bleeding in both adrenal glands. In the acute deaths large submucosal haemorrhagic patches (5–9 cm in diameter) in the gastric mucosa were seen in all the three cases; mottling haemorrhages in myocardium, adrenal glands, and kidneys were seen in one case. Congestion of brain, lungs and severe gastric erosions, were seen in the patients with hypoxic brain death.

**Anterograde memory loss**
Eighty four (40%) patients who recovered had a variable duration of memory loss. The range was 12 hours to eight days (mode three days). Duration of anterograde memory loss had no significant correlation to the lowest level of consciousness (r = 0.403, p = 0.000) or the duration of respiratory paralysis (r = 0.6386, p < 0.01) as patients in deep coma needed longer duration of ventilation.

**Delayed neuropathy**
Thirty eight patients had delayed neurological deficits. Fourteen of them had nerve conduction defects in the ulnar, median, and common peroneal nerves that lasted for two weeks to six months before complete recovery. Sensory loss at the site of bite was observed in 34 patients; this lasted for two weeks to six months. One patient had bilateral ulnar nerve palsy with wasting of small muscles of the hands, four patients had glove type sensory motor neuropathy, and one patient developed cerebellar ataxia which persisted for two years.

**DISCUSSION**
The medical importance of the genus *Bungarus*, its member species, details of physical characterisation, and distribution

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**Table 2** Details of autonomic dysfunction (188 patients)

<table>
<thead>
<tr>
<th>Dysfunction</th>
<th>Severe group (%) (n=101)</th>
<th>Mild-moderate (%) (n=87)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemosis, lacrimation, sweating</td>
<td>101 (100)</td>
<td>38 (43)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>90–120</td>
<td>45 (44)</td>
<td>46 (52)</td>
</tr>
<tr>
<td>120–150</td>
<td>38 (37)</td>
<td>4</td>
</tr>
<tr>
<td>&gt;150</td>
<td>18 (18)</td>
<td>0</td>
</tr>
<tr>
<td>Paralytic ileus</td>
<td>42 (41)</td>
<td>0</td>
</tr>
<tr>
<td>Blood pressure (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>130/90–150/100</td>
<td>68 (67)</td>
<td>42 (48)</td>
</tr>
<tr>
<td>100/110–190/130</td>
<td>24 (23)</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dysfunction</th>
<th>Severe group (%) (n=101)</th>
<th>Mild-moderate (%) (n=87)</th>
</tr>
</thead>
</table>

**Table 3** Recovery of functions in severely envenomed patients (n=101)

<table>
<thead>
<tr>
<th>Recovered function</th>
<th>No of patients</th>
<th>Mean (days)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough reflex</td>
<td>53</td>
<td>2.6</td>
</tr>
<tr>
<td>Gag reflex</td>
<td>54</td>
<td>2.8</td>
</tr>
<tr>
<td>Normal consciousness</td>
<td>90</td>
<td>2.8</td>
</tr>
<tr>
<td>Ophthalmoplegia</td>
<td>101</td>
<td>3.6</td>
</tr>
<tr>
<td>Memory</td>
<td>78</td>
<td>4.0</td>
</tr>
<tr>
<td>Neck to power grade 2–3</td>
<td>96</td>
<td>4.0</td>
</tr>
<tr>
<td>Normal respiration</td>
<td>101</td>
<td>3.0</td>
</tr>
<tr>
<td>Facial muscles</td>
<td>101</td>
<td>5.0</td>
</tr>
<tr>
<td>Phasis</td>
<td>101</td>
<td>5.1</td>
</tr>
<tr>
<td>Distal muscles (hand grip/foot)</td>
<td>101</td>
<td>6.0</td>
</tr>
<tr>
<td>Proximal muscles (hip/shoulder)</td>
<td>101</td>
<td>7.5</td>
</tr>
<tr>
<td>Sitting up, unsupported</td>
<td>96</td>
<td>7.0</td>
</tr>
<tr>
<td>Neck to full strength</td>
<td>101</td>
<td>8.7</td>
</tr>
</tbody>
</table>

*Mean number of days taken for full recovery of function.
in South East Asia was described in the early 19th century. It was believed that the venom of the common krait (B. caeruleus) produces depression of vital centres in the brainstem and causes respiratory paralysis and hypotension.

Currently it is understood that bungarotoxins block the transmission at the neuromuscular junction. The mortality rate of 77% among 35 proved common krait bites in India and deaths of 27 patients who received only traditional treatment in Sri Lanka have been reported in the year 1954 and 1987 respectively. Field surveys and case studies done in the past have described the ecology, epidemiology, and some clinical aspects of common krait bite in Sri Lanka. Some studies concluded that 97% of snake bite deaths in Sri Lanka are due to common krait and Russell's viper bites. The mortality rate of 77% among 35 proved common krait bites in India and deaths of 27 patients who received only traditional treatment in Sri Lanka have been reported in the year 1954 and 1987 respectively. Field surveys and case studies done in the past have described the ecology, epidemiology, and some clinical aspects of common krait bite in Sri Lanka. Nevertheless, the latest statistics of the national statistics. Nevertheless, the latest statistics of the General Hospital, Anuradhapura, showed the incidence of species specific snake bite as Russell's viper 48%, non-venomous snakes 33%, common krait 9%, hump nosed viper 7%, and cobra 2%. The present study is the largest study of common krait envenoming in Sri Lanka that has unearthed significant findings worth further discussion and debate.

The common krait is a nocturnally active, terrestrial snake which lives close to human dwellings, but is not vicious by nature. It creeps into houses over the ground or through the roof and exhibits arboreal tendencies. The present study testifies to these facts but also found the occasional daytime bite and biting in the tops of trees. It has also been observed by others that the bites occur at night while the victims are asleep. Why the snake prefers to creep into human dwellings and how man becomes the victim is yet to be documented. The common krait normally prefers to feed on small snakes. However, the krait is attracted by mice, rats, and geckos that are abundant in houses. While asleep, humans may be bitten either due to accidental handling or rolling over the snake, or exposed parts of the human body might be misidentified as prey. These may be possible provocative factors for a krait bite even though the common krait remains naturally indolent. The seasonal pattern of the common krait bite has been explained with mating behaviour, but the present study has shown the influence of changing environment like rain and severe drought as contributing factors.

Abdominal pain is an important and unexplained symptom observed in the present study and mentioned by other authors. When a patient is unaware of the bite, and wakes in the night with colicky abdominal pain, mainly in the epigastrium, they may be misdiagnosed due to lack of awareness on the part of medical practitioners. The finding of submucosal haemorrhages in the stomach in acute death may be responsible for this symptom. However, the finding of gastric erosions in many necropsies could be related to acute stress.

Mottling haemorrhages in the other organs and adrenal bleeding in a patient who developed shock before death could be directly related to envenoming or a secondary manifestation. Nevertheless, there are a few cases of bleeding tendency reported in the past. A significant number of patients had myalgia but the lack of facilities prevented us testing for myoglobin in plasma and urine. A former study of five common krait bites at Anuradhapura had demonstrated myoglobinemia in one patient who had myalgia. One of the patients envenomed by the Malayan krait (B.arendsi candidus) had generalised muscle pain and tenderness. These suggest that presynaptic phospholipase A2 could cause rhadomyolysis. However, none of the present patients had renal consequences of rhadomyolysis. The question is: Does a similar process affect the smooth muscles in the stomach and gut to produce abdominal pain and minute bleeding?

Severe respiratory paralysis was seen in the half the number of patients and they would have died without assisted ventilation. Similar results have been reported in a previous study in which two patients out of five needed assisted ventilation, and it gives us an understanding about survival chance in natural evolution of envenoming in common krait bite. Soon after establishing the intensive care unit at Anuradhapura, a study was done in a series of 30 patients who were managed with assisted ventilation in the year 1990 and the duration of ventilation ranged from eight hours to 10 days (median 33 hours), which is comparable with the duration of the present series. Rapidity of the development of respiratory paralysis in the present study showed a wide range from 30 minutes to 50 hours and another study has shown time range from seven to 12 hours.

On comparison with cobra bite, which can kill a man in 30 minutes, the progression of envenoming may be slow in an occasional case of krait bite.

The phenomenon of hypokalaemia needs explanation. A previous study has observed hypokalaemia in three patients and it was thought to be due to respiratory alkalosis as a result of hyperventilation. Hypokalaemia was considered as an indicator to adjust the rate and volume of respiration in assisted ventilation. However, the incidence of hypokalaemia was very high in the present study and it was associated with metabolic acidosis even though there were normal blood gases. The most likely explanation is the internal shift of potassium into cells due to β-adrenergic stimulation as a result of autonomic dysfunction. However, external losses and internal shift of K+ and H+ due to hormones such as insulin, renin, and aldosterone should be excluded.

Delayed neuropathy is different from acute neuromuscular paralysis and needs structural damage to nerve fibre, nerve ending, or demyelination. A case report has highlighted symmetrical distal motor neuropathy after Ceylon krait bite in 1988. The neurotoxins probably cause ultrastructural damage to motor nerve endings. Excitation of both parasympathetic and sympathetic autonomic systems explains most of the clinical manifestations like increased secretions, dilated pupils, tachycardia, and hypertension. A patient who had a Malayan krait bite had fixed dilated pupil, sweating, tachycardia, and hypertension due to parasympathetic abnormalities.

Alteration of the sensorium and progression to a deeply comatose state is not simply explained by cerebral hypoxaemia and the locked-in position due to severe neuromuscular paralysis. These patients were well oxygenated and the brainstem function tests were negative. Furthermore, associated anterograde memory loss is strongly suggestive of widespread depression of cerebral functions. Documentation of these observations are few in the literature. One patient with Ceylon krait bite remained deeply unconscious until death.

The polyvalent antivenom, which is manufactured in India, has doubtful efficacy in reversing established neuromuscular paralysis and needs structural damage to nerve fibre, nerve ending, or demyelination. A case report has highlighted symmetrical distal motor neuropathy after Ceylon krait bite in 1988. The neurotoxins probably cause ultrastructural damage to motor nerve endings. Excitation of both parasympathetic and sympathetic autonomic systems explains most of the clinical manifestations like increased secretions, dilated pupils, tachycardia, and hypertension. A patient who had a Malayan krait bite had fixed dilated pupil, sweating, tachycardia, and hypertension due to parasympathetic abnormalities.

The polyvalent antivenom, which is manufactured in India, has doubtful efficacy in reversing established neuromuscular paralysis and respiratory paralysis. Thekston et al clearly show the clearance of venom antigenaemia with intravenous polyvalent antivenom. It had no effect on bound antigen in neuromuscular junctions, which occurs quite rapidly with envenoming, but it neutralises the unbound venom in the blood. This concept is supported by the absence of correlation between recovery of respiration and dose of administered antiserum observed in the study. Development of highly penetrable monospecific antivenom against the local species to counter bound venom might reverse prolonged neuromuscular paralysis. Finding a suitable drug to displace venom tagged to the neuromuscular junction has been considered by many workers. Anti-cholinesterase had been tested and no benefit was found in reversing paralysis in two common krait bites and it was further confirmed by neuropathological tests in six patients in a recent study. However, anticholinesterase had produced dramatic improvement in one patient after Malayan krait bite.
Neurotoxins of the Asian cobra are predominantly postsynaptic in action and their effect can be reversed by the administration of anticholinesterase. Therefore, development of a new drug with presynaptic action might be useful in common krait bites, rather than further testing anticholinesterase.

The overall picture so far discussed is beyond the action of presynaptic neuromuscular block by β-bungarotoxin, phospholipase A2. Studies report the existence of 16 isotoxins in the β-bungarotoxin family. Anticoagulant and neurotoxic activities were found in the protein isolated from common krait venom which was subjected to sequence and crystal structure determination. The enzyme phospholipase A2 is shared by many snakes and it has a wide array of effects including haemolysis, vasodilatation, rhabdomyolysis, and release of endogenous autotoxins in addition to neuromuscular blocking. Further studies into venom toxicology would facilitate the understanding of the overall picture of krait bite.

This study identified factors such as poor resuscitation, delay in hospital admission, and complications contributing to deaths in common krait bite in Sri Lanka. Improvement of intensive care facilities in hospitals, awareness of the clinical course, and anticipation and management of complications can overcome the morbidity. Educating high risk populations about the biting pattern of the common krait and on preventive measures is likely to reduce the incidence of snake bite.

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