Indian National Snakebite Conference

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Draft Summary Protocols

Snakebite Prevention
Snakebite First Aid and Treatment
Support Concepts

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First Aid Treatment Protocol

Of primary importance is the need to recommend the most effective first aid for victims, to enable them to reach the nearest medical facility, in the best possible condition. Much of the first aid currently carried out is ineffective and dangerous (Simpson, 2006). The Conference has agreed on the following recommended method having viewed and considered the available research and concluded that other methods are not appropriate for the conditions in India.

Recommended Method for India

The first aid being currently recommended is based around the mnemonic:

"Do it R.I.G.H.T."

It consists of the following:

- ☑ **R =** **Reassure the patient. 70% of all snakebites are from non-venomous species. Only 50% of bites by venomous species actually envenomate the patient**
- ☑ **I =** **Immobilise in the same way as a fractured limb. Use bandages or cloth to hold the splints, not to block the blood supply or apply pressure. Do not apply any compression in the form of tight ligatures; they don't work and can be dangerous!**
- ☑ **G H =** **Get to Hospital immediately. Traditional remedies have NO PROVEN benefit in treating snakebite.**
- ☑ **T =** **Tell the doctor of any systemic symptoms such as ptosis that manifest on the way to hospital.**

This method will get the victim to the hospital quickly, without recourse to traditional medical approaches which can dangerously delay effective treatment (Sharma et al, 2004), and will supply the doctor with the best information on arrival.

Traditional Methods to Be Discarded

Tourniquets

The use of tight tourniquets made of rope, string, belt, or cloth have been traditionally used to stop venom flow into the body following snakebite. However, they have the following drawbacks and problems:

- Risk of ischemia and loss of the limb (Warrell, 1999).
- Increased risk of necrosis with 4/5 of the medically significant snakes of India (Fairly, 1929) (Pugh et al, 1987) (Warrell, 1995).
- Increased risk of massive neurotoxic blockade when tourniquet is released (Watt, 1988)
- Risk of embolism if used in Viper bites. Pro-coagulant enzymes will cause clotting in distal blood. In addition, the effect of the venom in causing vasodilation presents the danger of massive hypotension when the tourniquet is released.
- They do not work! (Tun Pe 1987) (Khin-Ohn Lwin 1984). Venom was not slowed by the tourniquet in several experimental studies, as well as in field conditions. Often this is because they are tied on the lower limb, or are incorrectly tied (Watt, 2003) (Amaral, 1998) (Nishioka, 2000).
- They give patients a false sense of security, which encourages them to delay their journey to hospital.

For the above reasons, Tourniquet use is contra-indicated for use in India.

Incision and Suction

- Incision (cutting) a victim with incoagulable blood increases the risk of severe bleeding as the clotting mechanism is no longer effective, and also increases the risk of infection. No venom is removed by this method.
- Suction devices have been conclusively proven not to reduce the amount of circulating venom (Bush, 2000) (Bush, 2004). On the other hand, there has been some evidence that these devices increase envenomation as they inhibit natural oozing of venom from the wound (Alberts et al, 2004). In addition, they have been shown to increase the local effects of necrosis (Alberts et al, 2004).

Electrical Therapy and Cryotherapy

Electric shock therapy for snakebite received a significant amount of press in the 1980's. The theory behind it stated that applying an electric current to the wound denatures the venom (Guderian et al, 1986). Much of the support for this method came from letters to journals, and not scientific papers (Bucknall, 1991) (Kroegal et al, 1986).

Meticulous research however showed that the venom is not denatured (Davis et al, 1992). In addition, it has been demonstrated that the electric shock has no beneficial effect at all

(Dart et al, 1991) (Howe et al, 1988) (Russell, 1987) (Russell, 1987a) (Snyder et al, 1987) (Hardy, 1992). It has now been abandoned as a method of first aid.

Cryotherapy involving the application of ice to the bite was proposed in the 1950's (Stanke 1951) (Glass, 1981). It was subsequently shown that this method had no benefit, and merely increased the necrotic effect of the venom.

Newer Methods Considered Inapplicable in the Indian Context

Pressure Immobilisation Method (PIM)

Pressure Immobilisation has gained some supporters on TV and in the herpetology literature. Some medical textbooks have referred to it. They have not however, reviewed the research, nor considered PIM's applicability in the Indian context!

- PIM was developed in Australia in 1974 by Sutherland (Sutherland 1981). His research involved tying monkeys to wooden frames and injecting venom, then seeing if a pressure bandage would slow the absorption. He achieved some good results, but there were mixed findings. He only used 13 monkeys which is not an adequate sample. He argued that a crepe bandage AND an integral splint be applied over the wound to a pressure of 55mm of mercury. The version used in India of a bandage alone, Sutherland argued would be ineffective.
- Further work done by Howarth (Howarth 1994) demonstrated that the pressure, to be effective, should be different in the lower and upper limbs. The recommended upper limb pressure was 45-75mm of mercury; the lower limb was 50-75mm of mercury.
- Howarth's work also showed that full immobilisation was crucial. If the victim walked for 10 minutes after application, the PIM would be ineffective (Currie, 1993). He also stated that pressures above the ranges specified would INCREASE the flow of venom. Gray (Gray 2003) argued that pressures under the recommended range may also increase venom flow.
- Work carried out by Norris (Norris 2005) showed that only 5% of lay people and 13% of doctors were able to correctly apply the technique!
- Further studies have demonstrated that improvised splints are ineffective (Davidson, 2001).
- In addition, pressure bandages should not be used where there is a risk of local necrosis, that is in 4/5 of the medically significant snakes of India (Bush, 2004).
- Therefore, Indian rural workers would need:
    1) To be in possession of crepe bandages and splints,
    2) For the victim to immediately drop to the ground when bitten
    3) To have to be in pairs as the bystander must tie the bandage and splint, while the victim remains immobile.
    4) To be able to tie the bandage to the correct level of pressure depending on whether an upper or lower limb was involved, when only 17% of emergency room doctors could achieve this.
    5) And not to have to walk for more than 10 minutes.

For the above reasons, Pressure Immobilisation is not recommended for use in India.

Washing the Wound

Victims and bystanders often want to wash the wound to remove any venom on the surface. This should not be done as the action of washing increases the flow of venom into the system by stimulating the lymphatic system.

Recent First Aid Research

There has been some initial research that has suggested that a 'Pressure Pad or Monash Technique' may have some benefit in the first aid treatment of snakebite (Anker et al, 1982) (Tun Pe et al, 1995) (Tun Pe et al, 2000). In this method, a hard pad of rubber or cloth is applied directly to the would in an attempt to reduce venom entering the system.

This method should be subjected to further research in India to assess its efficacy. It may have particular relevance to the Indian Armed Forces who carry Shell Dressings as part of their normal equipment, and would thus be ideally equipped to apply effective first aid in difficult geographic settings where the need is great.

Snakebite Prevention

The normal perception is that rural agricultural workers are most at risk, and that the bites occur first thing in the morning and last thing at night. However, this information is of very little practical use to rural workers in preventing snakebite, since it ignores the fact that often snakebites cluster around certain bio-mechanical activities, in certain geographic areas, at certain times of the day.

• Grass-cutting remains a major situational source of bites.
• In rubber, coconut, and areca nut plantations, clearing the base of the tree to place manure causes significant numbers of bites.
• Rubber tapping in the early hours (03:00-06:00).
• Vegetable harvesting/ fruit picking.
• Tea and coffee plantation workers face the risk of disturbing arboreal and terrestrial vipers when picking or tending bushes.
• Clearing weeds exposes workers to the same danger as their grass-cutting colleagues.
• Walking at night without a torch (flashlight) barefooted, or wearing open sandals accounts for a significant number of bites.
• Bathing in ponds, streams, or rivers in the evening. It should not be assumed that because the victim is bitten in water that the species is non-venomous. Cobras and other venomous species are good swimmers and may enter the water to hunt for prey.
• Walking along the edge of waterways.

Preventive Measures

- ☑ Walk at night with closed-type footwear (e.g., shoes or boots), and a flashlight that is switched on!
- ☑ Carry a stick when grass cutting or picking fruit or vegetables, or clearing the base of trees. Use the stick to move the grass or leaves first. Give the snake a chance to move away!
- ☑ Pay close attention to the leaves and sticks on the ground when collecting wood.
- ☑ Keep animal feed and rubbish away from your house. They attract rats, and snakes will eventually follow.
- ☑ Try to avoid sleeping on the ground.
- ☑ Keep plants away from your doors and windows. Snakes like cover, and plants help them climb up and into windows.

Snake Bite Treatment Protocol

Patient Assessment Phase: On Arrival

Deal with any life threatening symptoms on presentation, i.e., attend to Airway, Breathing and Circulation.
If there is evidence of a bite, where the skin has been broken, give Tetanus Toxoid
Routine use of antibiotic is not necessary.

Diagnosis Phase: General Principles

- ☑ Whenever possible, identify the snake responsible. However, it is important to remember that snake colouration is a very unreliable means of determining species.
- ☑ All patients should be kept under observation for a minimum period of 24 hours.
- ☑ According to some investigators abroad, bite marks have a limited use in determining species (Nishioka et al, 1995) (Norris, 1995). However, in India bite marks are of no use in identifying whether a species is venomous or not. Many non-venomous species leave just two fang-like marks, e.g., Wolf Snake. Some species like the Krait may leave no bite mark at all. Many venomous species have more than two fangs, as they grow reserve fangs in case the main ones break off.
- ☑ Determine if any traditional medicines have been used; they can sometimes cause confusing symptoms.
- ☑ Determine the exact time of the bite. This can give an indication as to the progression of any symptoms.
- ☑ Ask the victim as to what he was doing at the time of the bite. Some activities such as grass cutting or feeding stock animals in the evening can be indicative of the possibility of snakebite.

Pain

Snakebite can often cause severe pain at the bite site. This can normally be treated with painkillers such as paracetamol. Aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) should not be used, as they can exacerbate bleeding. This can be particularly dangerous in a patient already having coagulopathy. Mild opiates such as tramadol 50 mg can be used for good pain relief.

Handling Tourniquets

Care must be taken when removing tight tourniquets tied by the victim. Sudden removal can lead to a massive surge of venom leading to neurological paralysis, hypotension due to vasodilation, etc.

- Before removal of the tourniquet, test for the presence of a pulse distal to the tourniquet. If the pulse is absent, ensure a doctor is present before removal.

- Be prepared to handle complications such as sudden respiratory distress or hypotension. If the tourniquet has occluded the distal pulse, then a blood pressure cuff can be applied to reduce the pressure slowly.

Diagnosis Phase: Investigations

20 Minute Whole Blood Clotting Test (20WBCT)

This is generally considered to be the most reliable test of coagulation, and has the advantage of being able to be done at the bedside without specialist training. It can also be carried out in the most basic settings.

A few millilitres of fresh venous blood is placed in a new, clean and dry glass vessel and left at ambient temperature for 20 minutes. The glass vessel should be left undisturbed for 20 minutes and then gently tilted (not shaken!). If the blood is still liquid then the patient has incoagulable blood. The vessel must not have been washed with detergent, as this will inhibit the contact element of the clotting mechanism.

Other Useful Tests depending on availability:

- ☑ Haemoglobin/ PCV/ Platelet Count/ PT/ APTT/ FDP/ D-Dimer
- ☑ Peripheral Smear
- ☑ Urine Tests for Proteinuria/ RBC/ Haemoglobinuria/ Myoglobinuria
- ☑ Biochemistry for Serum Creatinine/ Urea/ Potassium
- ☑ Oxygen Saturation/ PR/BP/ RR/ Postural Blood Pressure
- ☑ ECG/ X-Ray/ Ultrasound (The use of X-Ray and Ultrasound are however of unproven benefit, apart from identification of bleeding in Viperine bites).

Diagnosis Phase: Symptoms

General

There are a great many myths surrounding snake symptoms. The table below summarises the evidence based situation. Haemostatic abnormalities are prima facie evidence of a Viper bite. Cobras and Kraits do not cause haemostatic disturbances.

Saw Scaled Viper does not generally cause renal failure, whereas Russells Viper and Hump-nosed Pitviper do.

Russells Viper can also manifest neurotoxic symptoms, and these have been observed by a number of investigators in India. This can sometimes cause confusion, and further work is necessary to establish how commonly it occurs. The neurotoxic symptoms in Russells Viper are believed to be pre-synaptic or Krait-like in nature. It is for this reason that doubts have been expressed over the response of both species to neostigmine.

Feature	Cobra	Krait	Russells Viper	Saw Scaled Viper	Hump Nosed Viper
Local Pain/ Tissue Damage	**YES**	**NO**	**YES**	**YES**	**YES**
Ptosis/ Neurological Signs	**YES**	**YES**	**YES!**	**NO**	**NO**
Haemostatic abnormalities	**NO**	**NO!**	**YES**	**YES**	**YES**
Renal Complications	**NO**	**NO**	**YES**	**NO**	**YES**
Response to Neostigmine	**YES**	**NO?**	**NO?**	**NO**	**NO**
Response to ASV	**YES**	**YES**	**YES**	**YES**	**NO**

General signs and symptoms of Viperine envenomation –

- Swelling and local pain
- Tender enlargement of local lymph nodes as large molecular weight Viper venom molecules enter the system via the lymphatics
- Bleeding from the oral cavity (gingival sulci) and other orifices
- Epistaxis
- Vomiting (Kalantri SP et al. 2006)
- Abdominal tenderness which may suggest gastro-intestinal or retro peritoneal bleeding.
- Hypotension resulting from hypovolaemia or direct vasodilation.
- Low back pain, indicative of early renal failure, although this must be carefully investigated, as many rural workers involved in picking activities complain of back pain generally
- The skin and mucous membranes may show evidence of petechiae, purpura, and ecchymoses
- The passing of reddish or dark-brown urine, or diminishing/ nil urine output.
- Lateralising neurological symptoms such as asymmetrical pupils may be indicative of intra-cranial bleeding
- Muscle pain indicating rhabdomyolysis
- Parotid swelling, conjunctival oedema, sub-conjunctival haemorrhage

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General signs and symptoms of Elapid envenomation –

- Swelling and local pain (Cobra)
- Local necrosis (Cobra) and/or blistering
- Descending paralysis, initially of muscles innervated by the cranial nerves, commencing with ptosis, diplopia, or ophthalmoplegia. There may be some involvement of the senses of taste and smell, but these need further research.
- Numbness around the lips and mouth, progressing to pooling of secretions, bulbar paralysis, and respiratory failure.

- Paradoxical respiration, as a result of the intercostal muscles becoming paralysed is a frequent sign.
- Stomach pain (Krait) which may suggest submucosal haemorrhages in the stomach (Kularatne 2002)
- Krait bites often present in the early morning with paralysis that can be mistaken for a stroke.

Late-onset envenoming –
Patient should be kept under close observation for at least 24 hours. Many species, particularly the Krait and the Hump-nosed Pitviper are known for substantial delays in the onset of manifestations (Joseph et al, 2006). Often this can take between 6 to 12 hours or more. Late onset envenoming is a well documented occurrence as reported by several other investigators also (Ho et al, 1986) (Warrell et al, 1977) (Reitz, 1989).

This is also particularly pertinent at the start of the rainy season when snakes generally give birth to their young. Juvenile snakes, 8-10 inches long, tend to bite the victim lower down on the foot in the hard tissue area, and thus any signs of envenomation can take much longer to appear.

ASV Administration Criteria

ASV is a scarce, costly commodity and should only be administered when there are definite signs of envenomation. Unbound, free flowing venom, can only be neutralised when it is in the bloodstream or tissue fluid. In addition, Anti-Snake Venom carries risks of anaphylactic reactions and should not therefore be used unnecessarily.

As per the W.H.O. SEARO Guidelines **ONLY** if a Patient develops one or more of the following signs/symptoms should ASV be administered:

Systemic envenoming -

- Evidence of coagulopathy: Primarily detected by (20WBCT) or visible spontaneous systemic bleeding, or gingival bleeding, etc. Further laboratory tests for thrombocytopenia, Hb abnormalities, PCV, Peripheral smear etc.
- Evidence of Neurotoxicity: ptosis, external ophthalmoplegia, paralysis, etc
- Cardiovascular abnormalities: hypotension, shock, cardiac arrhythmias, abnormal ECG
- Acute renal failure: oliguria/ anuria, rising blood creatinine/ urea
- Haemoglobinuria-/myoglobinuria: dark brown urine, urine dipsticks, other evidence of intravascular haemolysis or generalised rhabdomyolysis (muscle aches and pains, hyperkalaemia)
- Persistent and severe vomiting or abdominal pain

Local envenoming -

- Local swelling involving more than half of the bitten limb (in the absence of a tourniquet). In the case of severe swelling after bites on the digits (toes and especially fingers) after a bite from a known necrotic species.

- Rapid extension of swelling (for example beyond the wrist or ankle within a few hours of bites on the hands or feet)
- Development of enlarged tender lymph nodes draining the bitten limb

N.B.
If a tourniquet or tourniquets have been applied, once they have been removed for 1 hour and the swelling continues, this is unlikely to be as a result of the tourniquet, and ASV may be applicable.

Prevention of ASV Reactions – Prophylactic Regimes:

There is no statistical, trial evidence of sufficient statistical power to show that prophylactic regimes are effective in the prevention of ASV Reactions.

Of the three published studies on the efficacy of prophylactic regimens for prevention of reactions to ASV, one (Wen Fan et al) showed no benefit and the other two (Premawardenha et al, 1999; Gawarammana et al, 2003) showed only modest benefit. However, because these studies were underpowered to detect the true outcome effect, we need a large clinical trial to conclude that the prophylactic treatment helps.

Two regimes are normally recommended:

- 100mg of hydrocortisone and 10mg of H_1 antihistamine to be administered IV 1-2 minutes before ASV administration.

The dose for children is 0.2mg/kg of antihistamine IV, and 2mg/kg of hydrocortisone IV.

- 0.25-0.3mg adrenaline 1:1000 to be given subcutaneously.

The conclusion in respect of prophylactic regimes to prevent anaphylactic reactions, is that there is no evidence at present from good quality randomized clinical trials to support their use. If they are used, then the decision must rest on other grounds, such as political policy in the case of Government hospitals, which may opt for a maximum safety policy, irrespective of the lack of definitive trial evidence.

ASV Administration – Dosage:

In the absence of definitive data on the level of envenomation, such as provided by ELISA testing (Greenwood et al, 1974) (Theakston et al, 1977) (Ho et al, 1986), symptomology is not a useful guide to the level of envenomation. Any ASV regimen adopted is only a best estimate. What is important is that a single protocol is established and adhered to, in order to enable us to gauge results.

The dosage level detailed here is based on published research that Russells Viper injects on average 63mg SD 7 mg of venom (Tun Pe 1986). Logic suggests that our initial dose should be calculated to neutralise the average dose of venom injected. This ensures that

the majority of victims should be covered by the initial dose and keeps the cost of ASV to acceptable levels. The range of venom injected is 5mg – 147 mg.

This would mean that the total required dose will be between 10 vials to 25 vials as each vial neutralises 6mg of Russells Viper venom. Not all victims will require 10 vials as some may be injected with less that 63mg. Similarly, not all victims will require 25 vials. However, starting with 10 vials ensures we start by neutralising the average amount of venom injected, and during the next 12 hours have time to neutralise any remaining free flowing venom.

There is no convincing evidence that shows that Low Dose Strategies (Paul et al, 2004) (Srimannanarayana et al, 2004) (Agrawal et al 2005) have any validity in India. These studies have serious methodological flaws: the randomization is not proper, the allocation sequence was not concealed, the evaluators were not blinded to the outcome; there was no *a priori* sample size estimation, and the studies were underpowered to detect the principal outcome.

The same problem relates to high dose regimes (Wallace, 2004), often based on Harrison's textbook of medicine, which was written specifically for snakes encountered in the US, and not intended for use in the developing world.

NO ASV TEST DOSE MUST BE ADMINISTERED!
Test doses have shown to have no predictive value in detecting anaphylactoid or late serum reactions and should not be used (Warrell et al 1999). These reactions are not IgE mediated but complement-activated. They may also pre-sensitise the patient, and thereby create greater risk.

ASV is recommended to be administered in the following initial dose:

Neurotoxic/ Anti Haemostatic: 8-10 Vials

N.B. Children should receive the same ASV dosage as adults. The ASV is targeted at neutralising the venom. Snakes inject the same amount of venom into adults and children.

ASV can be administered in two ways:

1. Intravenous Injection: reconstituted or liquid ASV is administered by slow intravenous injection (2ml/ minute).
2. Infusion: liquid or reconstituted ASV is diluted in 5-10ml/kg body weight of isotonic saline or glucose

All ASV is to be administered over 1 hour.

The patient should be closely monitored for 2 hours.

Local administration of ASV, near the bite site, has been proven to be ineffective, painful, and raises the intracompartmental pressure, particularly in the digits. It should not be done.

Victims who arrive late:

A frequent problem is victims who arrive late after the bite, often after several days, usually with acute renal failure. Should the clinician administer ASV? The key determining factor is: are there any signs of current venom activity? Venom can only be neutralised if it is unattached! Perform a 20WBCT and determine if any coagulopathy is present. If coagulopathy is present, administer ASV. If no coagulopathy is evident treat the renal failure by referral to a nephrologist and subsequent dialysis.

In the case of neurotoxic envenoming where the victim is evidencing symptoms such as ptosis, respiratory failure, etc, it is probably wise to administer 1 dose of ASV to ensure that no unbound venom is present. At this stage, it is likely that all the venom is bound, and respiratory support will be the required treatment.

ASV Reactions:

Anaphylaxis is life-threatening, but despite the reluctance in giving ASV due to reactions (Kalantri et al, 2005), if the correct protocol is followed, it can be effectively treated and dealt with. Anaphylaxis can be of rapid onset, and can deteriorate into a life-threatening emergency very quickly. The patient should be monitored closely (Peshin et al, 1997) and at the first sign of any of the following, ASV should be discontinued, and 0.5mg of 1:1000 adrenaline given IM: urticaria, itching, fever, shaking chills, nausea, vomiting, diarrhoea, abdominal cramps, tachycardia, hypotension, bronchospasm, and angioedema.

Children must be given 0.01mg/kg body weight of adrenaline IM.

In addition, to provide longer term protection against anaphylactoid reaction, 100mg of hydrocortisone and 10mg of H_1 antihistamine should be administered IV.

The dose for children is 0.2mg/kg of antihistamine IV and 2mg/kg of hydrocortisone IV.

If after 10 to 15 minutes, the patient's condition has not improved or is worsening, a second dose of 0.5 mg of adrenaline 1:1000 IM is given. This can be repeated for a third and final occasion, but in the vast majority of reactions, 2 doses of adrenaline will be sufficient. If there is hypotension or haemodynamic instability, IV fluids should be given.

Once the patient has recovered, the ASV can be restarted slowly for 10-15 minutes, keeping the patient under close observation. Then the normal drip rate should be resumed.

The IM route is the option selected, due to the rapidity of development in anaphylaxis. Studies have shown that adrenaline reaches necessary blood plasma levels in 8 minutes by the IM route, but up to 34 minutes by the subcutaneous route (American Society,

2003) (Simons, 1998). The early use of adrenaline is being recommended due to study evidence suggesting better patient outcome if adrenaline is used early (Sampson et al, 1992).

In extremely rare, severe life threatening situations, 0.5mg of 1:10,000 adrenaline can be given IV. However this must be undertaken only if IM adrenaline has been tried without success, and the administration of IV adrenaline is in the presence of ventilatory equipment and ICU trained staff.

It is widely believed that anaphylactoid reactions are under reported (McLean-Tooke et al, 2003)

Late serum sickness reactions can be easily treated with oral antihistamines and corticosteroids (for e.g., prednisolone - adults 5mg 6 hourly; child 0.7mg/kg/day)

Neurotoxic Envenomation:

Neostigmine is an anticholinesterase and prolongs the life of acetylcholine which can reverse respiratory failure and neurotoxic symptoms. It is particularly effective in post-synaptic neurotoxins such as those of the Cobra (Watt et al, 1986). There is some doubt over its usefulness against the pre-synaptic neurotoxin as encountered in the venom of the Krait and the southern Russells Viper (Warrell et al, 1983) (Theakston et al, 1990). However it is worth trying in these cases.

In the case of neurotoxic envenomation, the 'Neostigmine Test' can be administered: 1.5-2.0 mg of neostigmine IM, preceded by 0.6mg of atropine IV.
The paediatric neostigmine dose is 0.04mg/kg IM.
The patient should be closely observed for 1 hour to determine if the neostigmine is effective.

The following measures are useful objective methods to assess this:
a) Number of millimetres of iris uncovered
b) Inter incisor distance
c) Length of time upward gaze can be maintained
d) Single breath count
e) FEV-1 or FVC (If available)

If the victim responds to the Neostigmine Test then continue with 0.5mg of neostigmine IM half hourly, together with 0.6mg of atropine IV over an 8 hour period by continuous infusion. If there is no improvement in symptoms after one hour, the neostigmine should be stopped.
Some authors have suggested that it may be possible to treat patients with anticholinesterase drugs solely, in the case of elapid bites (Bomb et al, 1996). However this approach ignores the value of neutralising the free flowing venom before it can attach and require reversing.

Recovery Phase

If an adequate dose of appropriate antivenom has been administered, the following responses may be seen:
a) Spontaneous systemic bleeding such as gum bleeding usually stops within 15-30 minutes.
b) Blood coagulability is usually restored in 6 hours. Principal test is 20WBCT
c) Post synaptic neurotoxic envenoming such as in the case of Cobra may begin to improve as early as 30 minutes after antivenom, but can take several hours.
d) Presynaptic neurotoxic envenoming such as in the case of Krait usually takes a considerable time to improve reflecting the need for the body to generate new acetylcholine emitters.
e) Active haemolysis and rhabdomyolysis may cease within a few hours and the urine returns to its normal colour.
f) In shocked patients, blood pressure may improve after 30 minutes.

Repeat Doses of ASV: Anti Haemostatic:
In the case of anti haemostatic envenomation, (in line with the W.H.O/S.E.A.R.O. Guidelines) the ASV strategy must be based around a six hour time period. When the initial blood test reveals a coagulation abnormality, the initial ASV amount can be given over 1 hour.

No additional ASV must be given until the next Clotting Test is carried out. This is due to the inability of the liver to replace clotting factors in under 6 hrs.

After 6 hours, a further coagulation test should be performed, and a further dose should be administered in the event of continued coagulation disturbance. This dose should also be given over 1 hour. CT tests and repeat doses of ASV should continue on a 6 hourly pattern until coagulation is restored.

The repeat dose should be 5-10 vials of ASV, i.e., half to one full dose of the original amount. The most logical approach is to administer the same dose again, as was administered initially. Some Indian doctors however argue that since the amount of unbound venom is declining, due to its continued binding to tissue, and due to the wish to conserve scarce supplies of ASV, there may be a case for administering a smaller second dose. In the absence of good trial evidence to determine the objective position, a range of vials in the second dose has been adopted.

Recurrent Envenomation:
When coagulation has been restored, no further ASV need to be administered, unless a proven recurrence of a coagulation abnormality is established. There is no need to give prophylactic ASV to prevent recurrence (Srimannarayana et al, 2004). Recurrence has been a mainly U.S. phenomenon, due to the short half-life of Crofab ASV. Indian ASV has a half-life of over 90 hours, and therefore is not required in a prophylactic dose to prevent re-envenomation.

Anti Haemostatic Maximum ASV Dosage Guidance:
The normal guidelines are to administer ASV every 6 hours until coagulation has been restored. However, what should the clinician do after say, 30 vials have been administered and the coagulation abnormality persists?

There are a number of questions that should be considered. Firstly, is the envenoming species one for which polyvalent ASV is effective? For example, it has been established that envenomation by the Hump-nosed Pitviper (*Hypnale hypnale*) does not respond to normal ASV. This may be the reason why in the case of Hypnale, as is well known, coagulopathy can continue for up to 3 weeks!

The next point to consider is whether the coagulopathy is resulting from the action of the venom. Published evidence suggests that the maximum venom yield from say a Russells Viper is 147 mg, which will get reduced the moment the venom enters the system and starts binding to tissues. If 30 vials of ASV have been administered, it accounts for 180 mg of neutralising capacity. This should certainly be enough to neutralise free flowing venom. At this point the clinician should consider whether the continued administration of ASV is serving any purpose, particularly in the absence of proven systemic bleeding.

Repeat Doses of ASV: Neurotoxic:
The ASV regime relating to neurotoxic envenomation has caused considerable confusion. If the initial dose has been unsuccessful in reducing the symptoms, or if the symptoms have worsened, or if the patient has gone into respiratory failure then a further dose should be administered. After 1-2 hours the patient should be re-assessed. If the symptoms have worsened, or have not improved, a second dose of ASV should be given.

This dose should be the same as the initial dose, i.e., if 10 vials were given initially, then 10 vials should be repeated for a second dose, and then ASV is discontinued.

Once the patient is in respiratory failure, has received 2 doses of ASV, and is supported on a ventilator, ASV therapy can be stopped. This recommendation is due to the assumption that all circulating venom would have been neutralised by this point. Therefore further ASV serves no useful purpose.

Evidence suggests that ‘reversibility’ of post-synaptic neurotoxic envenoming is only possible in the first few hours. After that, the body recovers by using its own mechanisms. Large doses of ASV, over long periods, have no benefit in reversing envenomation.

Confusion has arisen due to some medical textbooks suggesting that ‘massive doses’ of ASV can be administered, and that there need not necessarily be a clear-cut upper limit (Pillay,2005). This may be true with reference to snakes which inject massive amounts of venom, such as the King Cobra or Australian Elapids, but there appears to be no justification for massive doses of 50+ vials in India (Agrawal et al, 2001), which usually result from the continued use of ASV whilst the victim is on a ventilator.

No further doses of ASV are required; unless a proven recurrence of envenomation is established, additional vials to prevent recurrence is not necessary.

Hypotension

Hypotension can have a number of causes, particularly loss of circulating volume due to haemorrhaging, vasodilation due to the action of the venom, or direct effects on the heart. Test for hypovolaemia by examining the blood pressure in the lying down position, as well as in the reclining state, to establish a postural drop.

Treatment is by means of plasma expanders. There is no conclusive trial evidence to support a preference for colloids or crystalloids.

In cases where generalised capillary permeability has been established, a vasoconstrictor such as dopamine can be used. Dosing is 2.5- 5μg/kg/minute.

Russells Viper bites are known to cause acute pituitary adrenal insufficiency (Tun Pe et al, 1987) (Eapen et al 1976). This condition may contribute to shock. Follow-up checks on known Russells Viper victims needs to ensure that no long-term pituitary sequelae are evident.

Surgical Intervention

Whilst there is undoubtedly a place for surgical debridement of necrotic tissue, the use of fasciotomy is highly questionable. The appearance of

- Pain on passive stretching
- Pain out of proportion
- Pulselessness
- Pallor
- Parasthesia
- Paralysis

with significant swelling in the limb, can lead to the conclusion that the intracompartmental pressure is above 40 mm of mercury and thus requires a fasciotomy (Joseph, 2003). Fasciotomy is required if the intracompartmental pressure is sufficiently high to cause blood vessels to collapse and lead to ischemia. Fasciotomy does not remove or reduce any envenomation.

There is little objective evidence that the intracompartmental pressure due to snakebite in India, ever reaches the prescribed limit for a fasciotomy. Very limited trial data has tended to confirm this.

What is important is that the intracompartmental pressure should be measured objectively using saline manometers or newer specialised equipment such as the Stryker

Intracompatmental Pressure Monitoring Equipment. Visual impression is a highly unreliable guide to estimating intracompartmental pressure.

The limb can be raised in the initial stages to see if swelling is reduced. This is however not reliable, as there is no trial evidence to support its effectiveness.

Persistent or Severe Bleeding

In the majority of cases, the timely use of ASV will stop systemic bleeding. However in some cases, the bleeding may continue to a point when further treatment should be considered.

The first point to note is that clotting must have been re-established before additional measures are taken. Adding clotting factors, FFP, Cryoprecipitate or whole blood in the presence of un-neutralised venom will increase the amount of degradation products with the accompanying risk to the renal function.

Renal Failure and ASV

Renal failure is a common complication of Russells Viper and Hump-nosed Pitviper bites (Tin-Nu-Swe et al, 1993; Joseph et al, 2006). The contributory factors are intravascular hemolysis, DIC, direct nephrotoxicity, and hypotension (Chugh et al, 1975).

Renal damage can develop very early in cases of Russells Viper bite and even when the patient arrives at hospital soon after the bite, the damage may already have been done. Studies have shown that even when ASV is administered within 1-2 hours after the bite, it was incapable of preventing ARF (Myint-Lwin et al, 1985).

The following indicate the onset of renal failure:
Declining or no urine output, although not all cases of renal failure exhibit oliguria (Anderson et al, 1977).
Blood Testing

- Serum Creatinine > 5mg/dL or rise of > 1mg / day.
- Urea > 200mg/dL
- Potassium > 5.6 mmol/L

Evidence of uraemia:
Declining renal parameters require the referral to a specialist nephrologist with access to dialysis equipment. Peritoneal dialysis could be performed in secondary care centres. Haemodialysis is preferable in cases of hypotension or hyperkalaemia.

Use of Heparin and Botropase in Viper Bites

Heparin has been proposed as a means of reducing fibrin deposits in DIC (Paul et al, 2003). However, heparin is contraindicated in Viper bites. Venom induced thrombin is

resistant to heparin; the effects of heparin on Antithrombin III are negated due to the elimination of ATIII by the time heparin is administered, and heparin can cause bleeding by its own action. Trial evidence has shown it has no beneficial effect (Tin Na Swe, 1992)

Botropase is a coagulant compound derived from the venom of one of two South American Pitvipers. It should not be used as a coagulant in Viper bites as it simply prolongs the coagulation abnormality by causing consumption coagulopathy in the same way as the Indian Viper venom currently affecting the victim.

Appendix 1
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Appendix 2

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Appendix 3
Support Factors to Enhance Snakebite Protocols

Snakes of Medical Significance
Indian venomous snakes of medical significance have usually been regarded as only four species: Russells Viper, Saw Scaled Viper, Common Cobra and Common Krait. These species were believed to be causing all fatalities in India. However this concept has led to some serious problems:

1. ASV Manufacturers only produce antivenom against these species
2. The assumption that only 'The Big 4' can cause serious symptoms and death has led to mis-identification of species.
3. Other deadly snakes may be going un-noticed and causing death and disability! The recent discovery of the Hump-nosed Pitviper as a species capable of causing life threatening symptoms has demonstrated this.

In order to determine the actual list of medically significant species in India, the old concept of 'The Big Four' is to be abandoned for a newer more flexible model that enables better classification of species. The W.H.O. Model, produced in 1981, has been adopted as the Indian preferred method for categorising snakes of medical importance. The model is shown below:

Snakes of Medical Significance based on W.H.O. (1981)

- **Class I: Commonly Cause Death or Serious Disability**
  Russells Viper/Cobra/Saw Scaled Viper
- **Class II: Uncommonly cause bites but are recorded to cause serious effects (Death or Local necrosis)**
  Krait/Hump-Nosed Pit Viper/King Cobra
- **Class III: Commonly cause bites but serious effects are very uncommon**.

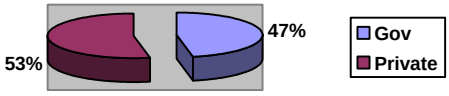
Further research is being undertaken to establish a definitive list of medically significant snakes in India.

Appendix 4
National Snakebite Conference Demographics

States and Union Territories Represented

Chandigargh (UT)	**Karnataka**	**Tamil Nadu**
Delhi (UT)	**Kerala**	**West Bengal**
Pondicherry (UT)	**Maharashtra**	
	Manipur	
Andhra Pradesh	**Orissa**	
Goa	**Punjab**	
Gujarat	**Rajasthan**	

Percentage of Government Institutions and Private in Expert Committee



Major Medical Colleges and Institutions Represented on Expert Committee

Amrita Institute of Medical Sciences & Research, Cochin, Kerala
Little Flower Hospital, Angamaly, Kerala
All India Institute of Medical Sciences (AIIMS), New Delhi
Armed Forces Medical College (AFMC), Pune
St Johns Medical College, Bangalore
J.I.P.M.E.R., Pondicherry
Post Graduate Institute of Medical Education & Research (PGI), Chandigargh
Madras Medical College (MMC), Chennai
Kasturba Medical College, Mangalore
Calcutta National Medical College, Kolkata
Mahatma Gandhi Institute of Medical Sciences, Sevagram, Maharashtra
Central Forensic Science Laboratory, Hyderabad, Andhra Pradesh