

## NOTES ON BLACK MAMBA (*DENDROASPIS POLYLEPIS*) ENVENOMATION TREATMENT USING THE PRESSURE/ IMMOBILISATION FIRST AID TECHNIQUE

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### INTRODUCTION

Bites from the Black Mamba *Dendroaspis polylepis*, still today remain a very serious and traumatic experience despite the highly effective polyvalent antivenom available. Being a large, aggressive front-fanged snake, bites resulting in envenomation can easily occur during handling or when the snake is threatened.

Treatment and case histories of envenomation from this species have been described. Various snakebite treatments have been applied over the years. These included the cutting and sucking of the bite site, potassium permanganate, petroleum, tourniquets, ice packs, etc. In addition a veritable cornucopia of unctions, extracts, ointments and elixirs, have been swallowed, inhaled or smeared into the bite sites. Success in the treatment of black mamba envenomations varied, from a near 100% fatality (Chapman, 1968) to a much higher success rate with the aid of modern medicine (Harvey, 1985; Hilligan, 1987).

Clear distinction must be made between the immediate first-aid treatment of snakebite, and its subsequent treatment under medical supervision. In the first instance treatment is involved with delaying the spread of the venom and/or the prevention of the onset of symptoms. Antivenom remains the most efficient method of neutralising the venom and thus stopping the symptoms. If antivenom is necessary, it is best given under medical supervision in a hospital.

Recently the pressure/immobilisation treatment was introduced to the South African herpetological scene as a first aid measure (Branch, 1985). It was subsequently used successfully in the treatment of several elapid envenomations including the Eastern Green Mamba, *Dendroaspis angusticeps* (Patterson & Morgan, 1985), the Egyptian Cobra, *Naja haje annulifera* (Els, 1988) and the Mozambique Spitting Cobra, *Naja mossambica* (Haagner, 1988).

### THE PRESSURE/IMMOBILISATION TREATMENT

This method originated in Australia where it has been successfully used on several neurotoxic envenomations (Pearn, *et al*, 1981; Sutherland, *et al*, 1980; Murrell, 1981). According to Branch (1985) its efficacy originates from a reappraisal of the importance of the lymphatic drainage of the bite site. The lymphatic circulation acts as an auxillary system, collecting interstitial fluid forced by the body movements from the blood vessels into the surrounding tissues. Small valves in the lymph vessels prevent back flow and body movements force lymph along the vessels. The fluid eventually drains into the blood system.

Important aspects regarding this method are that :

1. 98% of snakebites occur on limbs
2. venom is mostly injected into tissue, and not into the blood circulation
3. the lymphatics collect interstitial fluid draining from the tissues
4. lymph vessels are thin-walled and can be occluded by simple pressure from a broad elastic bandage, while full blood circulation to the limb is maintained.

The efficiency of this method in preventing the spread of small molecular weight elapid neurotoxins does not seem in doubt. However, reservation still exists on the advisability of this method for cytotoxic envenomations where local necrosis and severe swelling are normally present.

## **Application of this Method**

The practical application of this first-aid treatment is well illustrated by Marais (1985) and Branch (1985). Do not apply a tourniquet as tourniquets can cause unnecessary pain and even severe tissue damage. Apply a firm pressure to the bite site immediately by using your hand. Then wrap a firm bandage over the bite site as tight as for a sprained ankle. Now wrap the entire limb in the pressure bandage and then into a splint to minimise movement. The victim must be kept still and carried to a vehicle and transported to hospital immediately. The pressure bandage must stay on the limb until the patient is hospitalised and under medical care. If possible, take the snake (live or dead) with you for identification. If a pressure/crepe bandage is not available, improvise by using any clothing, towels or even socks.

## **CASE HISTORY OF BLACK MAMBA ENVENOMATION TREATED WITH THIS METHOD**

### **The Snake**

A large, aggressive diurnal elapid occurring throughout Northern Transvaal and Natal (South Africa), Botswana, Zimbabwe, Zambia further north through Africa to Nigeria and Senegal in the west (Hakansson & Madsen, 1982). The Black Mamba is a slender snake which often reaches 300 cm (10 feet) in length with the largest recorded at 425 cm (14' 3") (Bennetts, 1956). The average size is between 180 cm (6 foot) and 240 cm (8 foot). The snake's colour varies from a uniform light to dark brown to a lead grey. Despite its name, the snake is never jet black. The only black colouring on the snake is the interior of its mouth which distinguishes it from any other South African snake.

If provoked it will raise the anterior part of its body with an open mouth, displaying a narrow hood. If not immediately left alone, the snake will attempt to escape and strike as it passes. It does not hold on after a bite, but will deliver several quick bites in rapid succession when aggravated. Black Mambas feed on warm-blooded prey such as rodents, birds, squirrels, and other smaller mammals (Broadley, 1983). Although not essentially arboreal, the snake will spend some time basking in large trees. In more open bush-veld, old termitaria and hollow trees are favoured retreats.

### **The Victim**

An adult, 28 year Caucasian male, 93 kg.

### **The Specimen**

A three year old, captive raised male snake, measuring 1920 mm.

### **Bite Site**

The victim was bitten on the top of the right index finger on 6 March 1989, with only one fang penetrating: The bite was a result of negligence while feeding the snake and an act of greed by the snake rather than aggression.

### **Relevant Previous Medical History**

The victim had previously experienced several snakebites, including *Bitis a. arietans*, *Bitis caudalis*, *Dendroaspis angusticeps*, *Naja haje annulifera* and *Naja mossambica*, receiving antivenom for all except the *Bitis* bites. The victim showed severe allergy/hypersensitivity to the *Naja mossambica* venom with itching rash (urticaria) and bronchospasm (Haagner, 1988).

### **Symptoms and Treatment**

12h42: immediate bleeding from bite site with burning pain. Walked to first aid kit (4 metres away) and applied crepe bandage within 20 seconds after bite.

12h45: drove to reception office and asked staff to phone hospital at Acornhoek. Strange taste in mouth and intense burning in hand. Sense of panic for victim was unable to find somebody to transport him to hospital. Feeling dizzy and nauseous.

12h48: collected antivenom and left for hospital, 45km away. Feeling very nauseous and pulse 97/min – onset of respiratory difficulty.

12h52: Pulse 97/min – pain in hand, difficulty in breathing and speaking. ‘Pins and needles’ feeling in finger tips and lips.

12h55: increased difficulty in breathing, losing control over facial muscles and unable to focus eyes.

13h10: arrived at hospital. Unable to get out of vehicle – paralysed. Breathing and speech erratic. Rushed to Outdoor Patients Department.

13h12: Ringers Lactate 500 ml IV immediately with 200mg Solu-Cortef. Victim feeling 99% sure that he is going to die.

13h15: 10 ampules (100 ml) antivenom administered intravenously through drip. Intense burning sensation as antivenom entered veins with ‘pins and needles’ all over body. BP 140/60 Pulse 98/min. Victim still conscious but unable to speak, convulsions and vomiting.

13h58: 0.5ml anti-tetanus intra-muscularly and 2 ml Proc Pen intramuscular. Reacted to the penicillin – rash on neck and arms.

14h02: 200mg Solu-Cortef IVI in drip. Intense pain in hand – crepe bandage removed and bite site disinfected.

14h22: 10 mg morphine intramuscular. BP 130/80 Pulse 84/min. Kept on Ringers Lactate. Hospitalised.

16h00: easier breathing and able to move body but focus still watery. BP 120/80 Pulse 88/min. Hand still painful. Still very nauseous, sporadic vomiting.

20h00: feeling better but still nauseous and unable to clear focus. 30mg Serapex to sleep. Slept through night.

Day 2: hand still sensitive but no swelling. Itchy feeling round bite site which remained for several days.

Day 3-5: kept under observation but no further symptoms developed from envenomation. Discharged from hospital and went home.

## DISCUSSION

The rapid onset of symptoms are not surprising and have been previously described by Hilligan (1987) and Read & Foster (1959). In this instance, the onset of symptoms were extremely rapid with paralysis after only 15 minutes, despite the immediate application of the crepe bandage. This can probably be understood if the victim’s hypersensitivity is taken into consideration. According to the medical staff at the hospital, the immediate application of the crepe bandage contributed greatly to the survival of the victim.

Although the onset of symptoms can be very rapid, it has been reported that patients have survived only receiving treatment as long as 5 hours after having been bitten (Louw, 1967). Black Mambas can inject large quantities of venom, up to 400 mg per single bite, with 10-15 mg being a fatal dose for the average human (Broadley, 1983). Therefore, large quantity of antivenom is sometimes needed to effectively neutralise the venom. Visser & Chapman (1978) reported a case where the victim died 8 hours after envenomation, having received approximately 80ml polyvalent antivenom. In this case the victim was bitten three times resulting in too large quantities of venom to be effectively neutralised by the available antivenom. Interesting to note that Harvey (1985) reported total recovery of a victim without the aid of antivenom. The victim reached the hospital 4.5 hours after envenomation and was kept alive by a respirator. The victim made complete recovery and was discharged after 7 days.

Tilbury (1989) commented that the author’s immune system has been sensitised as a result of previous bites from different species, and that further envenomation will result in more serious forms of hypersensitivity – acute anaphylaxis or even death. Despite Tilbury’s (1989) prediction, no sign of allergy was shown by the victim towards Black Mamba venom. With

the victim's earlier bite by a Green Mamba no sign of allergy was present, with slower onset of respiratory symptoms (Haagner, 1987).

### CONCLUSION

The venom of the Black Mamba is a very potent neurotoxin that is rapidly absorbed, and as with other elapids causes paralysis of the nerves, especially those controlling the lungs, leading to respiratory arrest. At the same time, it paralyses the inhibitory nerve regulating the heart, causing a rapid and irregular pumping there-of. The victim will experience more and more difficulty in breathing until eventually death results from suffocation. The onset of symptoms appears to be a result of the amount of venom injected as well as the natural immune system of the victim. The onset of symptoms varies and is normally obvious in one to two hours after envenomation.

Due to the author's experience in the efficiency of the pressure/immobilisation treatment, this method can be strongly recommended as a first aid measure.

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