At first I thought I was becoming paranoid but each time I moved towards it, it repeated the same threatening action. Although the pain was not getting worse I now decided, slow learner that I am, to make discretion the better part of valour and left the water, sans captive catfish, with seemingly no one on the banks or footbridge any the wiser concerning my ignominious retreat.

I ruefully walked back to our friends' nearby holiday accommodation overlooking the river mouth, and after getting out of my wetsuit in a hot shower I obtained virtually immediate pain relief by immersing my arm in a bucket of very hot water, as is the well-documented first aid for most mild to moderate fish spine or barb envenomations, including stingray barbs.

Within seconds of removing my arm from the bucket the pain would return at the same intensity and there was an increasingly unpleasant additional component involving a burning sensation exacerbated by touching or rubbing the forearm. So I spent two hours sitting in front of the TV thanking various attendants (initially fascinated, but later bored!) for their boiled-kettle deliveries and occasional bucket-decanting manoeuvres so necessary to top up the bucket and maintain adequate water temperature.

There was a seemingly trivial puncture wound in the back of my forearm, but the skin of the forearm had a generalised mottling which lasted about 24 hours, localised mild swelling to a diameter of about 8 cm, and diffused but very mild forearm swelling lasting about 48 hours. I was slightly feverish and "weak and wobbly" on the Tuesday (Anzac Day) but by Wednesday, when I returned to work I felt well apart from minor tiredness which however was not easily explained by my modest activity levels over Easter. This had resolved in another day or so.

Minor local swelling (2 mm elevation, diameter 2 cm) has persisted till today (15/5/2000), ie some three weeks, but apart from very slight tenderness directly over the puncture site there has been no real pain since day one, only a mild ache which did not limit use of the arm or hand at all.

However the tiny (1–2 mm diameter) puncture wound, which bled only weakly during the first few hours and only ever looked mildly inflamed, took about a week to develop a dry scab and was very itchy from about day 7 to day 14. It is now only occasionally itchy but retains a tiny, slightly depressed scab and so has not yet completely healed.

Despite this event I can recommend this snorkel site as being refreshingly different (and although I didn't know it then, Phillip Hall and other Marine Life Society of South Australia members have also snorkelled here) and I hope to repeat it next summer or autumn, possibly with a camera.

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Applying pain theory in fish spine envenomation

David Muirhead

Key words
Envenomation, first aid, injuries, toxins, pain, marine animals

Abstract
Personal experience of catfish spine envenomation leads the author to question the long-accepted heat-labile toxin denaturation hypothesis as explanation for the established and very effective first aid treatment using hot water immersion of the envenomed limb. An alternative hypothesis compatible with contemporary pain theory is proposed.

Pain hypotheses in current usage, including Gate Control theory and Diffuse Noxious Inhibitory Control (DNIC) theory, have evolved substantially from observations that interference stimuli such as vibration, heat or cold, applied to the peripheral skin can induce pain relief at remote anatomical sites.

Have we overlooked the obvious in continuing to accept the hypothesis, entrenched in the diving medical community,[1,2,3] that heat-labile properties of fish spine toxins explain the well-documented analgesic effectiveness of hot water limb immersion in fish spine envenomation? A literature search has revealed a remarkable paucity of papers addressing this issue. Those that do, appear to assume that the proven heat lability of the few fish toxins so far analysed is the actual mechanism.

In April 2000, the author received a minor envenomation by an Estuary Catfish, *Cnidoglanis macrocephalus*, while snorkelling in an estuary south of Adelaide as described above.

As a South Australian coastal general practitioner, with occasional experience of treating mostly minor marine fish-spine injuries, I am familiar with the core first aid management using hot water (approx 46ºC) immersion of the affected limb. I expected excellent pain relief as I placed my envenomed right forearm into a bucket of hot water,

after first testing the water with my contralateral hand to avoid burns.

My confidence was vindicated, with almost instantaneous pain relief. But I was puzzled as to why, if the hot water was indeed inactivating the toxin, the pain would recur so promptly and at the same intensity upon removal of my arm from the hot water. I initially reasoned that until all the venom had been denatured, pain would continue, but this begged the question: why the dramatic pain relief within seconds of immersion in the first place? Perhaps the toxin is reversibly inactivated by heat? It might be capable of reconstitution with falling temperature, at least until persistent exposure to heat effects a more permanent decomposition, for example by allowing irreversible binding of component molecules to tissue substrate.

This explanation fails to address the fact that whilst both my puncture wound and those of patients I'd treated by hot water immersion were small in external appearance, there could be little doubt that the sting had penetrated to a depth of at least some millimetres into the soft tissues. Diving medical texts recommend that water used for pain relief be in the 45–50ºC range, yet human tissues other than perhaps the dermis, necrose before reaching these temperatures.

It seems improbable that exposure of the cutaneous portion of a puncture wound to such temperature would be capable of raising more deeply embedded subcutaneous or intramuscular residuae to temperatures sufficient to inactivate toxin without also causing significant tissue necrosis to the full depth of the puncture wound. This would in itself be very painful and thus defeat the objective. Further, all the fish spine wounds I've successfully treated by this method have been accompanied by sufficient localised oedema and serosanguinous ooze to make it unlikely in the first place that hot water could traverse the length of the puncture track, again discrediting this theory.

If one reviews current pain theory,[4,5] support for the role of Gate Control and DNIC pain theories in fish spine envenomation may be found in the extensive range of fish species whose spine envenomations are known to respond to hot water immersion. This is specifically with regard to local pain relief as opposed to any systemic sequelae of envenomation. It even appears probable that envenomations from creatures in other phyla such as Cnidaria [6] will respond to thermal treatment, and application of icepacks is already an accepted first aid analgesic measure for jellyfish stings.

Examples do exist in the marine environment of identical or nearly identical toxins being utilized defensively by phylogenetically disparate organisms, notably tetrodotoxin in puffer fishes (vertebrates) and blue-ring octopi (invertebrates). However, it seems improbable that hundreds, even thousands of marine animal species share toxins so similar that they are all inactivated by such a conveniently small elevation in temperature.

A comprehensive worldwide literature search via Medline dating back to 1966, has failed to find a single study whose specific aim was to demonstrate heat-labile properties of non-scorpaeniform fish spine venoms. A limited number of papers were found purporting to delineate haemolytic, dermonecrotic, oedema-promoting, vasospastic, and lethal components of catfish venom and skin toxins.[7]

These include studies of the oriental catfish (Plotosus lineatus),[8] North American species,[9,10] and the comparative toxicity of two catfish genera Ictalurus and Schilbeodes.[11] None of the above studies addressed heat-lability.

As long ago as 1966, Pacy in his review of Australian catfish injuries proposed, largely on the basis of a single case report involving a long-tailed catfish (Family Plotosidae, genus Plotosus), that the venom had vasospastic properties as well as possibly transient neurotoxic effects.[12]

Pacy stated:

> "Fish venoms tend to become rapidly inactive by change of pH (Wiener, 1960) and therefore instantaneous irrigation of the wound channel with sodium bicarbonate solution…is likely to destroy much of the poison."

and:

> "As stingray venom is destroyed by temperatures above 60ºC the possibility of this applying to catfish venom cannot readily be excluded. However, it is the great symptomatic relief that indicates hot bathing."

Pacy refers to earlier work specifically on stingray venom, and the quoted minimum temperature of 60ºC needed to destroy venom would seem to negate the relevance of heat-lability as the major reason for the effectiveness of hot water immersion in stingray envenomation.[13,14]

However, Pacy's case report does contain the following statement:

> "The patient repeatedly tried to take her hand out of the hot water, in order to get some sleep, but immediately the hand was withdrawn, the pain returned within a few seconds, disappearing only after renewed immersion."

This account perfectly matches my own experience, and is supported by a prospective observational case series of 22 fish stings, at least eight of which were from catfish, where hot water immersion treatment was completely effective in 73% of cases.[15]

The Estuary Catfish (*Cnidoglanis macrocephalus*) is the only marine member of the Plotosidae family known to occur in southern Australia, but most northern Australian catfish-spine injuries are also due to this family.[16]

The 'Poisindex Managements'[17] first aid treatment guidelines for catfish state:

> "HOT WATER – The injured part should then be submerged in hot water at as high a temperature as the patient can tolerate without injury (less than 113 degrees F or 45 degrees C), for 30 to 90 minutes or more."

However, none of the three references provided contain proof of heat-lability of catfish venom. Indeed, Sutherland and Tibballs[18] in their chapter titled *Venomous fish other than stonefish*, state:

> "Little is known about the nature of the venoms, which are associated with the spines of the many stinging fish found in Australian waters...Most of these fish venoms are presumably unstable in heat, and the aim of such treatment is to inactivate the venom present superficially and under the skin."

The text *Venomous and Poisonous Marine Animals, a Medical and Biological Handbook*,[16] while covering in considerable detail many aspects of catfish envenomation, contains only one direct comment on the possible heat-labile nature of fish venoms:

> "Fish venoms are predominantly unstable large proteins (Halstead 1988). As such molecules are dissociated with changes in pH and temperature, hot-water immersion might cause denaturation of the venom in the tissues. However, the return of pain on extraction of the part from hot water casts some doubt on this rationale. The analgesic efficacy of hot-water immersion for venomous fish injuries cannot be disputed and should always be adopted by first-aiders as a first measure for pain relief in venomous fish stings."

In summary, certain facts emerge concerning catfish envenomation. Most (probably all) catfish of the Plotosidae family contain venom apparatus and are a common cause worldwide of fish spine envenomations in humans. No scientific study has ever demonstrated heat-lability of catfish venom (to the best of my knowledge).

Might not the Gate Control and DNIC theories of pain explain the underlying mechanism for fish spine envenomation analgesia by hot water immersion?

Kakigi and Watanabe have shown that interference stimulations using vibration, active and passive movements of the hand or foot, noxious warming by hot water (46ºC) and noxious cooling by ice water (0ºC) all caused significant reduction in pain perception in normal human volunteers who were experiencing painful stimulation of either ipsilateral or contralateral hand or foot via CO_2 laser.[5] Specifically, they noted markedly reduced pain amplitude using noxious warming and cooling stimulation applied to the peripheral skin close to and remote from the site where laser stimulation was applied.

They deduced that, since the hot and cold stimuli mainly ascend through the small fibres, this pain relief could be better accounted for by DNIC theory than Gate Control theory, and they refer to clinical studies indicating that the site responsible for DNIC is the brainstem. Whilst an account of DNIC theory is beyond the scope of this paper, its application in the above study is clearly relevant to the phenomenon of hot water analgesia in fish spine envenomation, particularly as the study used water at 46ºC as the noxious stimulus. Further, standardised pain scores three to six minutes after taking the hand from the hot water (after-effect) did not show any significant change from the control session, consistent with my personal experience of rapid return of pain following arm removal from hot water.

Two questions are posed. Has hot water immersion been trialled for above-water envenomations, such as arachnids, hymenoptera and arthropods? Some of these toxins are heat-stable so a demonstrable efficacy would challenge the role of heat-lability as already discussed. Interestingly, although application of hot packs to jellyfish stings has been found to have only mild analgesic effect, immersion of the affected part in hot water has been found to be very effective.[6]

Secondly, would hot-water immersion of the contralateral limb also be effective in marine fish spine envenomations, or even an upper limb immersion where the lower limb is envenomed, or vice versa?

In conclusion, an extensive literature search has failed to find evidence supporting the denaturation theory. Further research is needed to investigate the mechanism(s) underlying pain relief by hot water immersion of the affected limb following fish spine envenomation. The author hypothesises that modern pain theory provides a better explanation than heat denaturation of toxins.

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On being a patient in a hyperbaric chamber

John Knight

Keywords
General interest, hyperbaric oxygen, osteoarthritis, hip arthroplasty, medical conditions

I remember as a medical student hearing a consultant say soon after the patient, a nurse, was asleep, "We will have to take extra care. Unexpected things go wrong with nurses and doctors, especially if they have red hair." As far as I know there are no statistics to confirm or deny this edict.

Just before I turned 65, I had my osteoarthritic left hip replaced. Some of my acquaintances suggested, tongue in cheek, that I should make a claim against the Royal Australian Navy on the grounds that dysbaric osteonecrosis had occurred as a result of my naval diving (four dives, one using an oxygen rebreather, none over 6 m). Unfortunately, there was a strong family history of osteonecrosis of the hip, so I could not consider indulging in creative litigation.

My post-operative course was smooth for five months. Then I developed acute pain in the left hip. To shorten a long story, four months later infection was diagnosed and I started on flucloxacillin. Within three days I no longer needed analgesics.

A year later, three weeks after stopping flucloxacillin, the pain came back. Back to flucloxacillin and the pain went but my mobility was quite impaired. I was advised strongly to undergo hyperbaric oxygen therapy (HBOT). So off I went to the Alfred Hospital Hyperbaric Medicine Unit, via an assessment panel of two orthopaedic surgeons, and an infectious diseases and a hyperbaric physician. The prescription was 40 sessions of an hour at 2.5 bar (absolute) (15 msw). With the HBOT went three antibiotics, flucloxacillin, sodium fucidate and rifampicin.

I joined a group of patients – the number varying depending on whether or not anyone was a stretcher case – none of whom were still being treated when I finished. The diagnoses included diabetic and other chronic ulcers resistant to all treatment, decompression sickness, radiation necrosis and osteonecrosis. The maximum number for the chamber was six patients and one hyperbaric nurse. Before we were allowed to climb into the chamber, we had to change out of everyday clothes into cotton theatre garb, to avoid static sparks. Getting into the chamber was awkward as the circular door of the chamber was 900 mm in diameter. For many the only option was to crawl through. There was a special hoist for stretchers, which reached into the chamber and deposited the stretcher and patient onto a bunk. The other patients sat on the opposite bunk.

Treatment in a multiplace recompression chamber is dull. The only excitement happens during compression, when