

Jellyfish Envenomation Syndromes Updated

Jellyfish venoms are mixtures of toxic and/or antigenic polypeptides and enzymes pathogenic to human beings. As newer therapeutic agents become available to treat the various reactions to stings caused by these animals, an accurate diagnosis of the type of reaction the patient experiences and of the offending species will be necessary. Fatal reactions may be caused either by anaphylaxis or by the action of toxins in the venom on the heart, respiratory center, or kidneys. Cutaneous eruptions after envenomation may be local, generalized, exaggerated, recurrent, delayed, persistent, or occur at sites distant from the primary sting. Fat atrophy, pigmentary changes, vasospasm, and contractures with gangrene can occur after jellyfish stings. Identification of the envenoming animal can be made by actual visualization, examination for nematocysts on skin scrapings, or serologically. It may also be predicted based on knowledge of location, time, and environmental circumstances of the encounter. First-aid measures designed to prevent additional nematocyst rupture are species-specific. Anaphylaxis should be prevented by the appropriate lifesaving measures. Other syndromes, caused by the toxins of the venom or mediated by humoral or cellular immune mechanisms, should be treated specifically. [Burnett JW, Calton G]: Jellyfish envenomation syndromes updated. Ann Emerg Med September 1987;16:1000-1005.]

INTRODUCTION

Jellyfish venoms are mixtures of toxic and/or antigenic polypeptides and enzymes pathogenic to human beings. Human reactions to these compounds may result in fatal, systemic, or local syndromes. Envenomation occurs when the victim's skin contacts the jellyfish tentacle to trigger the release of a venom-coated tubule from an epithelial intracytoplasmic organelle. These structures, called nematocysts, eject their threads with an approximate force¹ of 40,000 g to strike the skin at two to five pounds per square inch (TO Pochler, unpublished observations, July 1984). This energy enables the projectile to penetrate into the upper dermis, where the viscous venom, adherent to the thread, is released into the circulation² to produce any of several envenomation syndromes (Figure).

LOCAL REACTIONS

The most common envenomation syndrome is a local sting that results in a linear, urticarial, and painful eruption at the areas of tentacular contact. The pain produced by the sting may be due to the action of exogenous or endogenous chemical mediators on cutaneous sensory nerves.³ One particular mediator found in these venoms that may be responsible for much of the pain is the "kinin-like" factor. The eruption may begin as blanched papules or urticaria, but soon becomes erythematous. The lesions are of variable duration, usually minutes to hours, and may be vesicular, hemorrhagic, necrotizing, or ulcerative.² Localized hyperhidrosis, lymphadenopathy, subsequent fat atrophy, vasospasm, limb necrosis, gangrene, and contracture can occur.⁴⁻⁶ Persistent rubbing can produce lichenification.⁴ These stings are similar to those reported in cases of sea anemone envenomation. In cases of eye contact, conjunctivitis, chemosis, corneal ulcerations, and lid edema may result. Because the pain appears instantly and occurs with the patient's initial contact with the jellyfish, and because no immunity has been reported, it is believed that these stinging reactions are toxic phenomena rather

Joseph W Burnett, MD
Gary J Calton, PhD
Baltimore, Maryland

From the Dermatology Division,
Department of Medicine, University of
Maryland School of Medicine, and the
International Consortium for Jellyfish
Stings, Baltimore, Maryland.

Received for publication February 22,
1987. Revision received April 24, 1987.
Accepted for publication May 18, 1987.

Presented at the UAEM/IRIEM Research
Symposium on Environmental
Emergencies in Clearwater Beach,
Florida, February 1987.

Address for reprints: Joseph W Burnett,
MD, Division of Dermatology, University of
Maryland Hospital, 22 South Greene
Street, Baltimore, Maryland 21201.

than allergic events.

A case of an exaggerated local response to jellyfish envenomation also has been reported. This individual, initially exposed to *Chrysaora quinquecirrha* and *Pelagia noctiluca*, developed a large local edematous response lasting ten to 14 days after being stung by either jellyfish. Repeated investigations of his serum revealed a high antigen-specific IgE level to sea nettle and man-of-war antigen, which has persisted for many years.^{7,8} Additionally, his basophils were found to release histamine in abundance when exposed to sea nettle antigen in the laboratory (L Lichtenstein, JW Burnett, AG Togias, et al, unpublished observations, December 1983). Studies also were undertaken to determine whether this patient had abnormal lymphokines released by his white cells after exposure to jellyfish venom.⁹ The results were suggestive but not conclusive. It is our thesis that this eruption was analogous to large local reactions produced by arthropods.¹⁰

Studies on the patient's peripheral blood leukocytes, collected two years after the last envenomation and exposed to these jellyfish venoms, showed that lymphocyte migration inhibition was at high normal levels but increased when indomethacin was added to the cell culture medium.⁹ It is thought from these observations that IgE was an important component in the reaction to his envenomation; the role of the suppressor T cells was uncertain. That patients exhibiting large angioedematous reactions to a venom might later develop anaphylaxis to that agent is suspected but not proved. In the case cited in the fourth paragraph of the fatal reaction section, a female patient with anaphylaxis following a *Pelagia* sting had noticed large local reactions with some prior envenomations.¹¹

At least 13 patients with recurrent, linear, and cutaneous eruptions appearing at varying intervals after a sole, primary envenomation have already been observed;^{8,12-16} another three case histories are in press,¹⁷ and one more was reported to us (MJA Walker, personal communication, August 1985). The interval between the

FIGURE. Classification of jellyfish envenomation syndromes.

primary envenomation reaction and the recurrent eruption was five to 30 days. The interval between eruptions increases and the duration of the cutaneous lesions decreases with each recurrent episode. The recurrent episodes may be either more or less severe than the initial one, yet the painful symptoms decrease to pruritus with the recurrent attacks. In two patients tested, only a superficial dermal lymphocytic infiltrate was present in a biopsy specimen of the eruption at the time of the recurrence. One patient, approximately one month after her original sting by *Physalia*, had antigen-specific IgG and IgE antibodies in her sera to both sea nettle and man-of-war antigens.⁸ Four years after the sting, peripheral blood leukocytes did not exhibit lymphokine activity when exposed to *Physalia* venom.⁹ These cutaneous eruptions were thought to recur by an immunologic mechanism triggered either by a sequestered antigen residing intracutaneously or by exposure to a cross-reacting venom.

Another case, recently seen by us, occurred after contact with *Stomolophus meleagris* in a 32-year-old woman.¹³ Examination of a biopsy specimen taken from her ankle at the time of recurrence showed a significant lymphocytic perivascular infiltrate with eosinophils in the dermis. No nematocysts were identified in the biopsy. Direct immunofluorescence of this lesion was negative for IgG, IgM, and C3.¹³ Peripheral blood leukocytes, drawn both at the time of recurrence and a month later, did not show any increased lymphokine activity after exposure to *Stomolophus*, *Pelagia*, *Physalia*, and *Chrysaora* venom,⁹ even though she had elevated serum IgG antibodies against *Stomolophus*, *Physalia*, and *Pelagia* antigens, as well as normal IgE antibody levels against *Stomolophus* antigen.¹³ In one of the cases of recurrent jellyfish eruptions, oral prednisone suppressed the original cutaneous lesions.¹⁷

In addition to these known cases of recurrent cutaneous eruptions follow-

Local Reactions

- Toxin-induced
- Exaggerated local reaction (angioedema)
- Recurrent reactions up to four episodes
- Delayed persistent reactions up to several months
- Distant site reactions
- Contact dermatitis
- Papular urticaria up to one month

Long-Term Reactions

- Keloids
- Pigmentation
- Fat atrophy
- Contractions
- Gangrene
- Vascular spasm
- Mononeuritis
- Autonomic nerve paralysis
- Ataxia

Post-Episode Dermatitis

- Herpes simplex
- Granuloma annulare

Reactions From Jellyfish Ingestion

- Gastrointestinal symptoms
- Urticaria

Systemic Reactions

- Toxin-induced
- Irukandji reaction
- Respiratory acidosis

Fatal Reactions

- Toxin-induced
 - Immediate cardiac arrest
 - Rapid respiratory arrest
 - Delayed renal failure
- Anaphylaxis

ing a single jellyfish envenomation, our group is aware of five others, most occurring off Carolina beaches. Even though our referral area encompasses the endemic area of *Chrysaora* in the Chesapeake Region, we know of only one case with recurrent eruption that was primed by that animal's venom¹⁷ and none that were triggered by sea nettle stings. This would indicate that certain species (probably oceanic *Physalia*) are more likely to induce

these recurrent reactions. It is also our clinical impression that persons experiencing recurrent eruptions were not envenomed frequently prior to the triggering sting. The female:male case ratio is 5:1.

Persistent, delayed granulomatous nodules subsequent to jellyfish envenomation also have occurred.¹⁴ One patient had moderate pain with the initial transient sting. Four to seven days later, a delayed chronic eruption began at the site of tentacular contact. This healthy young woman, presumably envenomed by *Pelagia noctiluca* in the Mediterranean sea, developed a refractory facial lesion that persisted for seven months. This patient had normal serum levels (equal to control sera) of antigen-specific IgG and IgE antibodies (anti-*Chrysaora*, anti-*Physalia*, and anti-*Pelagia*) in her sera three years after the envenomation, as shown in tests performed by our laboratory. At the same time, however, peripheral blood mononuclear leukocytes from this patient, exposed to *Chrysaora* antigen, had slight increases in their lymphokine activity, which could be reversed by the addition of indomethacin to the culture medium. This suggests that prostaglandins played a role in the lymphokine response.⁹

The cutaneous eruption on the patient's cheek was an erythematous and indurated nodule 3 to 4 cm in diameter. Examination of a biopsy specimen of this lesion showed a dermal cellular infiltrate similar in morphologic features to that of the delayed hypersensitivity reaction in human beings.¹⁴ This pathologic picture differed markedly from the scant dermal perivascular mononuclear cell infiltrate usually present in coelenterate envenomations.² Because of this fact and the chronicity of the eruption, the patient's reaction to envenomation was thought to be an example of delayed cell-mediated immunity. Other persistent eruptions lasting several weeks also have been reported after envenomation by other coelenterates.^{14,17,18} The lesions in these patients, however, were much smaller than the lesion of the patient described in this paragraph, and in addition, were multiple rather than

singular.

A case of an erythematous, inflammatory eruption appearing at a site distant to the envenomed area was reported after a *Physalia* sting.¹⁹ This patient, who experienced envenomation on her right ankle and foot, developed an exudative and erythematous inflammatory response on the right ear, gingiva, and cheek a few hours later. This distant eruption progressed for two days, stabilized, then subsided after ten additional days. Although her inflammatory response resembled the anticipated reaction to eating a jellyfish,⁴ repeated questioning failed to explain this eruption on the basis of nematocyst contact with the head and neck. Seven years after envenomation, no antigen-specific IgE or IgG was detected in our laboratory against *Physalia* antigen in her serum.

An unusual contact dermatitis caused by the tentacles of *Physalia* and other jellyfish was reported recently by a Japanese group.¹⁶ Although no T cell studies were performed, repeated topical patch test challenges on clinically normal skin were conducted and the results were positive.

A papular urticarial eruption was reported in the case of a pregnant 29-year-old atopic Japanese housewife, who contacted a powdered jellyfish that she was preparing to use as a delicacy for a meal.²⁰ Fifteen minutes after soaking the animal in hot and cold water, her hands began to "burn." This reaction was quickly followed by generalized pruritus and giant elevated urticaria. On the following morning, the patient had multiple small scattered urticarial lesions that were 1 to 2 cm in diameter and persisted for 30 days. This patient's urticarial lesions could be controlled with low doses of chlorpheniramine and topical drying agents. Because this patient's dermatitis was caused by a dried, yellow purchased jellyfish, adequate species identification was not possible (it was presumably *Rhopalonema*). Neither immunofluorescence nor cytologic studies were performed on the eruption, and it is assumed that these lesions were a hypersensitivity reaction analogous to papular urticaria.

In another example of papular ur-

ticarial lesions, severe *Chironex fleckeri* stings resulted in tiny, pruritic, papular, erythematous, and punctate lesions in nonenvenomed skin a few days after a significant sting was experienced nearby.²¹ In one of the recurrent eruption cases, papular, urticarial lesions distant from the original envenomation site accompanied an exacerbation.¹⁷

CHRONIC OR LONG-TERM REACTIONS

Long-term local sequelae of jellyfish envenomation include the appearance of keloids, hyperpigmentation, fat atrophy, gangrene, contraction, and scarring.^{5,6,21} Subcutaneous fat atrophy has appeared at the site of envenomation from an Indian Ocean jellyfish, starting six weeks after the sting and persisting for more than six months.⁶ Four cases of vascular spasm of the upper extremity are currently known²² [JA Williamson, personal communication, September 1986; D Mebs, personal communication, January 1987]. All of these cases occurred in the Indo-Pacific region. A self-resolving mononeuritis multiplex on the extremity occurred after a suspected man-of-war sting off the coast of North Carolina.²³ Temporary paralysis of multiple autonomic nerves in a swimmer followed an envenomation in Malaysian waters.²⁴ This patient had a distended abdomen and urinary bladder, and was unable to develop an erection, urinate, or lacrimate. The R-R interval on ECG did not change with breathing or carotid sinus massage. In a similar case, a victim exhibited 48 hours of low-grade fever, bladder atony, diplopia, and ataxia after a *Physalia* sting.

Post-Envenomation Dermatitis

In one patient, an episode of herpes labialis appeared a day after a severely painful sting on a New Jersey beach. In another instance in Hawaii, granuloma annulare lesions appeared on the leg a few days after a sting [RM Mandojana, personal communication, February 1986].

JELLYFISH INGESTION

Human reactions to jellyfish ingestion include toxin-induced cramping

and abdominal pain.²⁵ One case of generalized cutaneous eruption following jellyfish ingestion has been cited.²⁶ The nature of the latter reaction is unknown. A puzzling observation has been that no case of serum sickness after jellyfish envenomation, either by ingestion or natural tentacle contact, has been reported to date.

SYSTEMIC REACTIONS

In addition to the cutaneous eruption, some stings are accompanied by such systemic symptoms as malaise, weakness, ataxia, dizziness, local cramping, muscle spasms, paresthesias, low-grade fever, nausea, and vomiting, all of which are thought to represent constitutional toxic reactions to the venom.^{2,25-27} In some of these cases, it appears that movement of the envenomed limb leads to increased mobilization of the venom from the inoculation site as a "muscle pump" is activated.

An "Irukandji" syndrome was described several years ago after envenomation by a small jellyfish, *Carukia barnesi*.²⁵ The name of this syndrome is derived from an aboriginal Australian tribe living on northeast Queensland where *Carukia* is endemic. A small but transient sting, frequently invisible and comparable to a minor insect bite, initiates this reaction. Cutaneous pain may be delayed five to 40 minutes, after which it starts at the sacrum or abdomen as a severe, boring pain that spreads to the thighs and chest, where the sensation is described as cramping. Severe, excruciating waves of generalized myalgias occur for hours (personal communication, P Finner, May 1987). Subsequently, the Irukandji syndrome persists over the next 24 to 48 hours. The other signs and symptoms may include tremor, anxiety, piloerection, hyperpnea, headache, nausea, vomiting, sweating, restlessness, pyrexia, tachycardia, coughing, blood-streaked sputum, prostration, pallor, cyanosis, and oliguria. Spontaneous recovery gradually follows.²⁵ Neither histologic nor immunologic studies of these victims have been reported. In one case a lumbar puncture was done; the cerebrospinal fluid was normal (JA Williamson, personal communication, October 1984). In another case, left ventricular failure requiring intubation was detected (JA Williamson, personal communication, January 1987). Anecdotally, administration of phen-

tolamine appears to reverse the hypertension seen in these cases.²⁸ This syndrome is assumed to represent a toxic reaction to the venom of the small jellyfish.

FATAL REACTIONS

Human fatalities may occur as a result of anaphylaxis or toxic mechanisms. Although no human beings have been intensively studied at the time of a fatal envenomation, it appears most likely that various toxins within the venom may be lethal by any of three methods. The most potent marine venom known is that of the southeast Pacific box jellyfish (*Chironex fleckeri* and *Chiropsalmus quadrigatus*), which kill experimental animals by inducing cardiac or respiratory arrest.²⁹ These deaths may occur within a few seconds with concentrated venom or may be delayed for several hours. Those animals dying instantly appear to die from cardiac arrest.⁷ Coronary vasospasm may play a role in the lethal action.³⁰ Respiratory arrest can occur rapidly and also will appear in animals receiving treatment to counteract the cardiotoxin of these jellyfish.³¹ One patient developed respiratory acidosis after a *Physalia* sting.³² This state of hypoxia may have been a forerunner of the fatal respiratory arrest seen in more severely envenomed patients. Some cases of jellyfish venom-induced delayed death in experimental animals are thought to occur because of hyperkalemia resulting from a "shocked" kidney syndrome.³³

It has been estimated that at least 50 feet of box jellyfish tentacles must touch the skin of an adult in order to deliver a fatal dose.³⁴ Each tentacle imprint marks the site of additional intradermal venom injection. Perfusion of the injured skin and mobilization of the venom are increased as the stricken patient writhes or struggles because of the pain. Jellyfish deaths in human beings can occur as a result of central respiratory failure appearing many minutes after envenomation. The instant death found in animals injected intravenously with jellyfish venoms has not yet been seen in human beings, probably because the blood venom level has not achieved a sufficient threshold to fatally injure the heart before respiration ceases. However, a recent death of a 5-year-old child in Australia from *Chironex* envenomation may have been due to

cardiac causes. (JA Williamson, personal communication, February 1987).

A human case of acute renal failure associated with intravascular hemolysis and hemoglobinuria in a 4-year-old girl stung on three extremities by the Portuguese man-of-war, *Physalia physalis*, has been reported.^{35,36} A six-hour latent period elapsed before she became acutely ill, and her renal output was suppressed for at least two weeks. Recovery required a two-week hospitalization. Although this case was not fatal, it is our hypothesis that it is analogous to cases resulting in delayed death of experimental animals.³³

Jellyfish envenomation may also kill human beings who suffer anaphylaxis.⁴ Some of the initial studies on anaphylaxis were performed in dogs with the use of coelenterate (sea anemone) protein as antigen.³⁷ A recent case report illustrates anaphylaxis after jellyfish envenomation. The serum of a healthy 59-year-old woman, found hypotensive and pulseless 15 minutes after being stung on the knee, presumably by *Pelagia noctiluca*, contained elevated levels of IgE directed against the venom of a related jellyfish species (*Chrysaora quinquecirrha*) nine months later. At that time, her basophils were capable of releasing increased levels of histamine when contacting this antigen, and a heat-labile factor in her serum could transfer the jellyfish venom sensitivity to normal human basophils.¹¹

DIAGNOSIS AND THERAPY

The correct therapy for jellyfish envenomation syndromes requires both an adequate diagnosis of the disorder and accurate identification of the offending species. This fact will become apparent as technology yields better pharmaceutical and immunologic therapeutic agents,⁷ as well as improved protein-separating procedures to isolate venoms and delineate the pathogenesis of envenomation. The treatment of anaphylaxis consists of maintenance of the airway, fluid resuscitation, oxygen supplementation, and the prompt parenteral administration of epinephrine. Therapy for a potentially fatal toxic reaction from *Chironex fleckeri* includes the maintenance of ventilation and cardiac function. Intravenous verapamil drug therapy may prove to be useful.^{31,38} Although this agent has not been used in human beings, experimental evi-

dence in rats and mice suggests that it may be of value in human *Chironex* envenomation.

In cases of *Chironex* envenomation, IV species-specific hyperimmune sera or antivenin is available from the Commonwealth Serum Laboratory in Melbourne, Australia. Immunotherapy should be considered to counteract the systemic symptoms and pain in severely stung patients,²¹ but side effects of anticomplementary activity and serum sickness can occur. Reassurance of the patient and immobilization of the envenomed part are essential. First-aid therapy should include methods to suppress additional nematocyst rupture and may be species-specific. The patient's skin should be sprayed with vinegar if the offending animal is thought to be *Chironex fleckeri* or *Physalia physalis*; a slurry mixture of baking soda should be used for stings by *Chrysaora quinquecirrha*.³⁹⁻⁴¹ Once the nematocysts are counteracted by these measures, manual removal of adherent tentacular strips follows. The use of systemic corticosteroids will be ineffective in cases of envenomation produced by toxic reactions. The application of local heat or cold is not beneficial for *Chrysaora* stings. Therapy with indomethacin, ibuprofen, or non-steroidal analgesics should be entertained in cases of prolonged, persistent reactions.⁴² Refractory hyperpigmentation may be controlled by hydroquinone bleaches, and keloids can be treated in the standard manner, with injectable corticosteroids to alleviate the cutaneous elevation and local tenderness. Some animal experimental evidence indicates that methysergide and indomethacin may reduce the cutaneous vasopermeability induced by *Physalia* and *Chironex* venoms, respectively.⁴² The therapy recommended here will probably change as the field continues to advance;⁴³ for this reason we have recently founded the International Consortium for Jellyfish Stings to better collect and review envenomation cases.

SUMMARY

Numerous recent advances in the management of jellyfish envenomation syndromes necessitate knowledge of the offending species and components of its venom. The pathogenic reaction in human beings most probably is a mixture of toxic and allergic mechanisms. In view of these facts,

the emergency physician should make his therapy species specific and capable of counteracting the effects of the multiple polypeptides injected into the patient that produce the specific envenomation syndrome.

REFERENCES

- Holstein T, Tardent P: An ultrahigh-speed analysis of exocytosis: Nematocyst discharge. *Science* 1984;223:830-833.
- Ioannides G, Davis JH: Portuguese man-of-war stinging. *Arch Dermatol* 1965;91:448-451.
- Burnett JW, Calton GJ: The chemistry and toxicology of some venomous pelagic coelenterates. *Toxicon* 1977;15:177-196.
- Maratec Z, Russell FE: Stings by the sea anemone *Anemonia sulcata* in the Adriatic sea. *J Trop Med Hyg* 1963;32:891-896.
- Gunn MA: Localized fat atrophy after jellyfish sting. *Br Med J* 1947;2:687.
- Drury JK, Noonan JH, Pollock JH, et al: Jellyfish sting with serious hand complications. *Injury* 1980;12:66-68.
- Hartman KR, Calton GJ, Burnett JW: Use of radioallergosorbent tests for the study of coelenterate toxin-specific immunoglobulin E. *Int Arch Allergy Appl Immunol* 1980;61:389-393.
- Burnett JW, Cobbs CS, Kelman SN, et al: Studies on the serologic response to jellyfish envenomation. *J Am Acad Dermatol* 1983;9:229-231.
- Burnett JW, Hepper KP, Aurelian L: Lymphokine activity in coelenterate envenomation. *Toxicon* 1986;24:104-107.
- Mauriello PM, Barde SH, Georgitis JW, et al: Natural history of large local reactions from stinging insects. *J Allergy Clin Immunol* 1984;74:494-498.
- Togias AG, Lichtenstein LM, Kagey-Sobotka A, et al: Anaphylaxis following contact with a jellyfish. *J Allergy Clin Immunol* 1985;75:672-675.
- Mansson T, Randle HW, Mandojana RM, et al: Recurrent cutaneous jellyfish eruptions without envenomation. *Acta Derma Venereol (Stockh)* 1985;65:72-75.
- Burnett JW, Calton GJ: Recurrent eruption following a solitary envenomation by *Stomolophus meleagris*. *Toxicon* 1985;23:1010-1014.
- Reed KM, Bronstein BR, Baden HP: Delayed and persistent cutaneous reactions to coelenterates. *J Am Acad Dermatol* 1984;10:462-466.
- Williamson JA: *The Marine Stinger Book*, ed 3. Queensland, Australia, Brisbane Surf Life Saving Association of Australia, 1985, p 37.
- Ohtaki N, Satoh A, Azuma H, et al: Delayed flare up reactions caused by jellyfish. *Dermatologica* 1986;172:98-103.
- Burnett JW, Hepper KP, Aurelian L, et al: Recurrent eruptions following unusual solitary cutaneous envenomation. *J Amer Acad Dermatol* 1987 (in press).
- Peters H: Hydroiden-dermatitis. *Hautarzt* 1967;18:396-400.
- Matusow RJ: Oral inflammatory responses to a sting from a Portuguese man-o-war. *J Am Dent Assoc* 1980;100:73-75.
- Yaffee HS: A delayed cutaneous reaction following contact with jellyfish. *Derm Int* April-June 1968, 75-77.
- Williamson JA, LeRay LE, Wohlfart M, et al: The acute management of serious box-jellyfish (*Chironex fleckeri*) stings. *Med J Aust* 1984;141:851-853.
- Adiga KM: Brachial spasm as a result of a sting. *Med J Aust* 1984;1:181-182.
- Filling-Katz MR: Mononeuritis multiplex following jellyfish stings. *Ann Neurol* 1984;15:213.
- Chand RP, Selliah K: Reversible parasympathetic dysautonomia following stinging attributed to the box-jellyfish (*Chironex fleckeri*). *Aust New Zealand J Med* 1984;14:673-675.
- Halstead BW: Phylum Coelenterata, in *Poisonous and Venomous Marine Animals of the World*, vol 1. Washington, DC, US Government Printing Office, 1965, p 297-536.
- Marr JF: Portuguese man-of-war envenomization: A personal experience. *JAMA* 1967;199:155.
- Maguire EJ: *Chironex fleckeri* (sea wasp) sting. *Med J Aust* 1968;2:1137-1138.
- Fenner PJ, Williamson J, Callanan VI, et al: Further understanding of, and a new treatment for "Irukandji" (*Carukia barnesi*) stings. *Med J Aust* 1986;145:569-574.
- Endean R, Duchemin C, McCollm D, et al: A study of the biological material derived from nematocysts of the cubomedusan (*Chironex fleckeri*). *Toxicon* 1969;6:179-204.
- Lee CY, Lee SY, Lin WW, et al: A study on the cause of death due to sea nettle venom, in Kornalik F, Mebs D (eds): *Proceedings of the 7th European Symposium on Animal, Plant and Microbial Toxins*. London, Pergamon Press, 1986, p 118-119.
- Burnett JW, Calton GJ: Response of the box jellyfish (*Chironex fleckeri*) cardiotoxin to intravenous administration of verapamil. *Med J Aust* 1983;2:192-194.
- Kizer KW, Piel M: Arterial blood gas exchange with bluebottle envenomation. *Hawaii Med J* 1982;41:193-194.
- Burnett JW, Calton GJ: Some chemical and pharmacological studies on two venomous jellyfish, in Ohsaka A, Hayashi K, Sawai Y (eds): *Animal, Plant and Microbial Toxins*, vol. 2. New York, Plenum Press, 1976, p 337-350.
- Endean R: The box jellyfish, or "sea wasp" in Pearn J (ed): *Animal Toxins in Man*. Brisbane, Queensland, The Division of Health Education and Information, 1981, p 46-54.
- Spelman FJ, Bowe EA, Watson CB, et al: Acute renal failure as a result of *Physalia physalis* sting. *South Med J* 1982;75:1425-1426.
- Guess HA, Savitcer PL, Morris CR: Hemolysis and acute renal failure following a Portuguese man-o-war sting. *Pediatrics* 1982;70:979-981.
- Portier P, Richet C: De l'action anaphylactique de certain venins. *Compt Rend Soc Biol (Paris)* 1902;54:170-172.
- Burnett JW, Gean CJ, Warnick JE, et al: The effect of verapamil on the cardiotoxin of the

Portuguese man-o'-war (*Physalia physalis*) and the sea nettle (*Chrysaora quinquecirrha*). *Toxicon* 1985;23:681-689.

39. Hartwick R, Callanan V, Williamson J: Disarming the box jellyfish: Nematocyst inhibition in *Chironex fleckeri*. *Med J Aust* 1980;1:15-20.

40. Turner B, Sullivan P, Pennefather J: Disarming the blue-bottle: Treatment of *Physalia* envenomation. *Med J Aust* 1980;2:394-395.

41. Burnett JW, Rubinstein H, Calton GJ: First aid for jellyfish envenomation. *South Med J* 1983;76:870-872.

42. Burnett JW, Calton GJ: Pharmacological effects of various venoms on cutaneous capillary leakage. *Toxicon* 1986;24:614-617.

43. Burnett JW, Calton GJ, Burnett HW: Jellyfish envenomation syndromes. *J Amer Acad Dermatol* 1986;14:100-106.