
Stinging and biting insect allergy: an Australian experience

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Background: Stings and bites from various insects are responsible for many anaphylactic events.

Objective: To document the clinical features of specific forms of anaphylaxis and investigate clinical concerns regarding stinging and biting insect allergy.

Methods: All patients presenting for evaluation of adverse reactions to insect stings or bites between December 1980 and December 1997 had the clinical details of their reactions recorded and their reactions classified.

Results: The spectrum of clinical symptoms and signs is similar to that seen in anaphylaxis from other sources; stings on the head or neck are not more likely to cause life-threatening reactions than stings elsewhere on the body; a lesser reaction will not necessarily lead to a more serious reaction from a future sting; asthmatic patients do appear to have an increased risk of asthma as a feature of their anaphylactic response; anaphylaxis is usually confined to a particular insect species for the individual patient; patients who have had multiple stings at one time may have experienced true anaphylaxis and not a "toxic" response; and patients who have had anaphylaxis from other sources are at no greater risk than that of the general population of reacting similarly to insect stings or bites.

Conclusions: Anaphylactic events from insect stings show the same clinical features as those from other sources. Systemic reactions seem confined to a specific insect species. Patients who experience RXN3 reactions from multiple stings at one time should undergo specific venom testing, because many have experienced true anaphylaxis and not a toxic response. Future consideration should be given to the role of β -adrenergic antagonists and ACE inhibitors in patients with systemic reactions.

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INTRODUCTION

Stings and bites from a variety of insects are responsible for anaphylactic events in many people. Although previous studies¹ have classified allergic reactions to insect stings and bites, problems are still encountered with currently available diagnostic reagents. For example, currently available diagnostic reagents in Australia for wasp sting anaphylaxis fail to detect specific IgE antibodies in approximately 30% of patients, suggesting significant species differences between native Australian and North American paper wasps.

This report documents the clinical features of specific forms of anaphylaxis and, in particular, investigates clinical concerns regarding stinging and biting insect allergy. For example, this study explores whether (1) a patient would be more likely to have a major systemic response (ie, a generalized, potentially life-threatening reaction characterized by dizziness, faintness, coma, and/or dyspnea) if stung on the head or neck than if stung on the lower parts of the body; (2) a less severe response is likely to be followed by a more severe one; (3) asthmatic patients are more likely to experience asthma as a feature of anaphylaxis than nonasthmatic patients; (4) patients who react to one type of insect also react to other insects; (5) patients who experience systemic reactions following multiple stings at one time have an allergic or a toxic response; and (6) a person who has had anaphylaxis to

a food or to other allergens is at high risk of reacting severely to an insect sting or bite as well.

METHODS

Patients

All patients who presented to my clinic at the Watkins Medical Centre, Brisbane, Queensland, for evaluation of adverse reactions to insect stings or bites between December 1980 and December 1997 were included in this study. The patients were primarily drawn from Queensland, a state occupying approximately one fifth of Australia's total land mass and lying in tropical and subtropical zones.

Clinical details of each reaction were recorded, with emphasis on (1) the interval between the sting or bite and the onset of the adverse response, (2) the site on the body of the sting or bite, (3) the sequence of symptoms and signs in chronological order, (4) the patient's previous experience with insect stings or bites, (5) personal history of hypersensitivity to other allergens or of atopy, (6) family history of stinging or biting insect allergy, (7) associated medical conditions, and (8) concomitant medications.

Classification of Reactions

Reactions were classified as follows: RXN0, no adverse response; RXN1, large, local swelling only; RXN2, minor systemic response (ie, urticaria and/or angioedema confined to the skin); RXN3, major systemic response (ie, evidence of airway obstruction and/or impairment of blood circulation or of consciousness); and RXN4, ill-defined responses (ie, se-

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rum sickness-like responses or nonallergic responses in patients receiving multiple stings [>40] at one time).

Diagnostic Tests

Skin testing, in vivo and in vitro testing, study methods, and reagents have been previously described.¹

RESULTS

Patients

A total of 1,787 patients participated in this study, of whom 1,213 (69%) were male. The patients ranged in age from 15 months to 77 years, with a mean age of 26.3 years; 714 (40%) were younger than 18 years and 512 of these were male.

Insect Types

Of the 1,787 patients, 924 (52%) believed that honeybee stings caused the reaction, usually confirmed by the presence of a stinger at the site; 678 (38%) identified paper (*Polistes* sp or *Ropalidia* sp) or, rarely, mud wasps (hornets); and 111 (6%) nominated various types of ants (eg, 47 identified greenhead ants [*Rhytidoponera metallica*], 9 jumper ants [*Myrmecia pilosula*], 6 bull [*Myrmecia* sp], 1 fire ant, and 48 unidentified). These native Australian ant species belong to genera of the subfamily Myrmicinae, which is different from the genus *Solenopsis*, and thus their venom proteins do not have significant immunologic cross-reactivity with the fire ant, *Solenopsis invicta*.^{2,3} Twenty patients reacted to bites from March flies (*Tabanus* sp, insects similar to deerflies or horseflies) and 27 to infestation from scrub ticks (*Ixodes holocyclus*). Somewhat surprisingly, only 7 people were not confident of the insect type (with 1 suspecting a honeybee sting and 5 suspecting an ant sting).

Time to Onset

Estimation of the time interval between the sting or bite and the first symptom is detailed in Table 1. A total of 799 patients (45%) stated that the reaction began immediately or in less than 5 minutes, with a further 327 (19%) believing that the reaction began within 10 minutes (ie, in 64%, the reaction developed within 10 minutes). A further 276 (16%) nominated an interval of between 10 and 30 minutes. Of the remainder, 164 (9%) listed more than 2 hours, but most of these (89%) recorded the least severe response, large, local

swelling only. In contrast, 81% of those with RXN3 reactions and 69% with RXN2 reactions recorded the onset of symptoms at less than 10 minutes. In addition, the reactions began beyond 30 minutes in only 12 (4.7%) of 257 patients with RXN2 reactions and 19 (1.6%) of 1,194 with RXN3 reactions. Although it is not documented herein, most of the patients in the RXN2 and RXN3 categories estimated that the full response peaked anywhere between 5 and 30 minutes.

Spectrum of Symptoms and Signs

The symptoms and signs of the 1,194 patients with anaphylaxis (RXN3) are as follows. Urticaria or erythroderma with dyspnea (laryngeal and/or bronchial) were the most represented of the symptoms and signs, both occurring in 775 (65%) of the patients. Itching was usually the first symptom detected, followed within a few minutes by urticaria or erythroderma. A total of 425 (36%) had facial angioedema, with or without urticaria, and 77 had tongue angioedema. Asthma was reported in 457 patients. A total of 454 patients (38%) reported faintness, dizziness, or weakness, with an additional 179 (15%) who lapsed into unconsciousness or were difficult to arouse. In regard to dyspnea, the level of airways obstruction was thought to have been the chest in 456 (38% of those with RXN3 reactions) or the larynx in 318 (27%). Some of these patients believed that both areas were affected at once, whereas 86 patients (7%) reported laryngeal swelling but no dyspnea. Cardiac rhythm disturbances were observed in only 85 (7%) (83 with tachycardia and 2 with bradycardia). Associated symptoms and signs included headaches in 13 (in some, quite severe), gastrointestinal symptoms in 13, seizures in 6, conjunctivitis in 1, and rhinitis in 10, whereas in 2 women uterine cramping was a major response. Eighty-two patients (7%) experienced dizziness, faintness, weakness, or coma as their sole expression of anaphylaxis, 48 (4%) had airways obstruction only, whereas an additional 79 (7%) had a combination of these two only.

Although large, local swellings were apparent in 72 patients, for most of the RXN3 group (94%) there was no or minimal swelling at the site of the sting or bite. One sign that created difficulty in evaluation was the significance of drowsiness, often leading to sleep, in young children (13 children younger than 6 years experienced drowsiness).

Table 1. Response Type and Duration to Onset*

Response	Time to onset, min†							
	5	10	15	20	30	45	60	>120
RXN1	35	15	6	7	9	1	11	150
RXN2	94	67	23	13	24	1	6	5
RXN3	670	245	82	54	57	3	12	4
RXN4	0	0	1‡	0	0	0	0	5
Total	799	327	112	74	90	5	29	164

* Data are number of patients.

† Estimated time by patient.

‡ Patient had polyarthritis as the sole adverse response.

Table 2 lists the types of insects implicated in the various reaction types. In patients with RXN3 reactions, honeybees and wasps were responsible in 52% and 38% of the reactions, respectively.

Relationship of Current Response with Previous Adverse Response

A total of 441 patients had had a previous RXN3 reaction, and of these 406 (92%) presented with a new RXN3 reaction (Table 3). A total of 512 had not recorded any type of adverse response previously; 360 (60%) of these had an RXN3 reaction as the first expression of hypersensitivity. One hundred (66%) of the 151 patients with previous RXN2 reactions presented with new RXN3 reactions. However, these 100 patients comprise only 8% of the total number with RXN3 reactions. Furthermore, 284 patients (24%) with prior RXN1 reactions were identified in the group of patients with RXN3 reactions. A total of 309 had RXN1 reactions on presentation; 215 (70%) of these had not recorded any adverse response other than the RXN1 reaction. Thirty-seven of the 100 in the group who progressed from an RXN2 to an RXN3 reaction were children.

Relationship of Sting Site with the Type of Response

As detailed in Table 4, 265 patients (24%) who had an RXN3 reaction were stung on either the head or neck, 349 (32%) were stung on the upper limb, 127 (12%) on the trunk, and 351 (32%) on the lower limb. On further analysis (Table 5), of those who experienced laryngeal involvement, either dyspnea or swelling only, 100 (26%) received stings on the head or neck, 113 (29%) on an upper limb, 50 (13%) on the trunk, and 126 (32%) on a lower limb. Of this group with laryngeal edema, 134 (34%) were stung on either the hand or foot. Of those with asthma, 97 (23%) were stung on the head or neck compared with 165 (38%) on a lower limb. In addition, of the group presenting with evidence of hypotension, 148 (25%) were stung on the head or neck in contrast to 175 (29%) with stings on a lower limb.

Association with Other Allergic Disorders

Of the total group, 311 (18%) gave positive family histories of stinging or biting insect allergy of either the RXN2 or RXN3 type. A total of 977 patients (55%) had not had an allergic response of any other type, and 154 of these had

RXN1 reactions only. A total of 291 (16%) had also had asthma, 263 (15%) allergic rhinitis, and 45 (2.5%) eczema. A total of 106 (6%) had had adverse reactions to more than one insect type. Of these, 38 had experienced RXN3 reactions: 18 to honeybee and wasp, 15 to wasp and ant, 2 to honeybee and ant, 1 to honeybee and tick, and 2 to wasp and tick.

Analysis of Asthmatic Patients

Of the group, 291 (16%) had histories of past or present asthma. A total of 206 (71%) had RXN3 reactions, with 113 (55%) of these exhibiting asthma. Of the 1,476 nonasthmatic patients, 988 (67%) had RXN3 reactions, and of these, 343 (35%) showed an asthmatic response.

Analysis of Patients with Multiple Stings

Eight patients experienced at least 40 to 50 stings at one time. Four reacted to honeybees (all 4 had RXN3 reactions); the other 4 reacted to wasps (with 3 showing RXN3 reactions and 1 an RXN1 reaction). Each of the patients who had been stung by a honeybee had a positive allergy-specific test result. In the wasp sting group, only 2 underwent allergy-specific tests, with 1 positive result. Regarding the other 2, the patient with the RXN1 reaction was not tested, whereas the other was not skin tested because he was taking a β -blocking agent and then was lost to review. None of the 7 with RXN3 reactions had previously experienced a systemic response from the involved insect.

Relationship of Systemic Reactions with Specific Tests

Of the 727 patients who presented with RXN2 or RXN3 honeybee sting reactions, 646 underwent allergy-specific tests. Twenty-four (3.7%) showed negative responses on either skin testing and/or radioallergosorbent testing (RAST). In contrast, 140 (26%) of the systemic reactors to wasp stings failed to show positive responses.

Analysis of Patients Who Reported Adverse Reactions to First Sting

Sixty-four patients presented with adverse reactions to a type of stinging insect that they claimed they had never encountered before. Of the 14 in the bee sting group, 3 had RXN2 reactions and 8 had RXN3 reactions, whereas all but 2 of the 46 wasp sting patients had systemic reactions, with 34 of the reactions classified as RXN3. Two ant sting patients and one

Table 2. Types of Insects Implicated in Reactions*

Insect type	Types of reactions				Total
	RXN1	RXN2	RXN3	RXN4	
Honeybee	195	138	589	2	924
Wasp	97	86	492	3	678
Ant	11	28	72	1	111
March fly	2	0	17	1	20
Tick	5	5	17	0	27
Uncertain type	0	0	7	0	7
Total	309	257	1,194	7	1,767

* Data are number of patients.

Table 3. Current Response to Previous Response*

Previous response	Current response				Total
	RXN1	RXN2	RXN3	RXN4	
RXN0†	68	84	360	0	512
RXN1	215	94	284	0	593
RXN2	6	45	100	0	151
RXN3	15	20	406	0	441
First sting‡	5	14	44	1	64
RXN4	0	0	0	6	6
Total	309	257	1,194	7	1,767

* Data are number of patients.

† No previous adverse response.

‡ First known sting from the insect causing the response.

Table 4. Sting Site with Response Type*

Response type	Sting site							Total
	Head	Neck	Arm	Hand	Trunk	Leg	Foot	
RXN1	50	9	35	61	7	31	72	260
RXN2	29	18	40	34	18	24	81	244
RXN3	188	77	170	179	127	126	225	1,092
RXN4	1	0	1	1	0	0	1	4
Total	268	104	241	275	152	181	379	1,600†

* Data are number of patients.

† The total number of patients is less than the study population because it does not include those with multiple stings at one time or those in whom the sting site was not recorded.

Table 5. Sting Site and Type of RXN3 Reaction*

Response	Sting site							Total
	Head	Neck	Arm	Hand	Trunk	Leg	Foot	
Laryngeal								
Swelling	15	8	16	13	15	8	12	87
Dyspnea	54	23	42	42	35	39	67	302
Total	69	31	58	55	50	47	79	389
Asthma	72	25	60	67	42	53	112	431
Faintness or dizziness	64	20	78	70	50	62	82	436
Coma	47	17	25	23	18	13	18	161
Total	252	93	221	215	160	175	301	1,417

* Data are number of patients. Individual patients may have had more than one response.

March fly patient experienced systemic reactions. Of the total number, 19 claimed that they had not had a prior insect sting or bite of any type, with 16 of these having either RXN2 or RXN3 reactions. Eleven of the group were young children; 4 of these had had stings from other insects. Thirty-eight of the patients who responded to bee or wasp stings had experienced previous stings from ants.

Analysis of Patients Taking Concomitant Drugs

Eighteen patients who were taking β -adrenergic antagonists at the time of the adverse response experienced systemic reactions; 13 of these reactions were classified as RXN3.

Only 3 of the group had had RXN3 reactions previously. Nine of 11 patients who experienced RXN3 reactions were taking angiotensin-converting enzyme (ACE) inhibitor reagents at the time, and 5 had had prior RXN3 reactions.

DISCUSSION

Stinging or biting insect allergy represents an unambiguous event in a study of anaphylaxis. Typically, the patients are well before the episode, there is little doubt that they received a sting or bite, and there are no other competing circumstances. Although one of the limitations of this study is that

it relies on the patient's accurate recall of the sting or bite, the observations made herein still provide important information in understanding the condition.

The study confirms the expected time sequence of a classic IgE antibody-mediated event; that is, in almost two thirds of the patients who experienced generalized responses (RXN2 and RXN3), the initial symptom appeared within 10 minutes of exposure and within 30 minutes in 98%. In contrast, in approximately two thirds of those with RXN1 reactions only, the response did not begin before 2 hours, suggesting that IgE antibodies probably did not induce many of these.

Most patients with RXN3 reactions had a combination of symptoms and signs. A common sequence of events was an onset with itching, followed quickly by a frank rash and/or facial angioedema, and then the more serious features of choking, asthma, and/or evidence of hypotension, leading to impaired consciousness. Although symptoms indicative of hypotension were commonly reported (in 53% of this group), only 15% experienced loss of consciousness. This compares with an incidence of 20% in a previous report.⁴ It was noted that 209 (18%) of this group developed either dyspnea and/or evidence of hypotension without any other associated clinical feature. Uterine cramping was reported in only 2 women; in retrospect, in the early years of the study, this specific query was not pursued in women who recorded abdominal pain and is probably underreported. The most severe reactions rarely were accompanied by large swellings at the sting site (ie, only 72 with RXN3 reactions had these responses compared with 412 of the total group). One difficult sign to evaluate accurately was drowsiness, because a number of very young children fell asleep shortly after being stung. However, they typically were easily aroused and thus probably were not hypotensive. Other systemic responses, presumably not IgE antibody mediated (eg, immune complex or perhaps cell-mediated reactions), have been reported following insect stings.⁵ These were observed in only 7 of the total group.

Of the 1,194 patients with RXN3 reactions, 360 (30%) had never previously reacted adversely to that specific insect. There were an additional 284 (24%) who had had prior RXN1 reactions only. In addition, 92% of this RXN3 group had had similar reactions previously. It therefore seems that most do not follow a progression from RXN1 to RXN2 to RXN3 reactions.

A possible exception to this belief is the RXN2 group. One of the hardest decisions to make in the evaluation of patients who present with RXN2 reactions is whether or not to commit them to a long and somewhat unpleasant course of specific venom immunotherapy. The general consensus is that adults who present with RXN2 reactions warrant such therapy, whereas children probably do not.^{6,7} Whereas it is possible that patients with RXN2 reactions may be less likely to consult an allergist than those with RXN3 reactions, two thirds of the former group subsequently develop RXN3 reactions. Furthermore, 37 of these 100 patients were younger than 18 years, which suggests that in this study children with RXN2 reactions are at just as much risk as adults to progress

to RXN3 reactions from future stings from the specific insect. This finding is in contrast to previous observations.⁶ On the other hand, 45 patients with RXN2 reactions had had the same response previously; 18 (40%) were children, a rate similar to the number who progressed to RXN3 reactions. The observations reported herein tend to support the notion that minor systemic reactions may be followed by more serious ones from future stings.

The sting site on the body does not seem to correlate with the severity of the response. Indeed, fewer patients with RXN3 reactions received stings on the head or neck than on either the upper or lower limb. In particular, those experiencing laryngeal involvement were just as likely to have been stung on the hand or foot than on the head or neck. These findings were also seen in patients with evidence of hypotension and asthma, respectively. An earlier study supports these findings, with the exception that those who experienced loss of consciousness were more likely to have had stings to the head.⁴

More than half of the patients had not experienced allergic reactions to anything other than the specific insects. A small number of these (16%) experienced RXN1 reactions only, indicating that systemic reactions to insect stings or bites do not necessarily occur in people with atopy, a finding previously reported.⁴ The respective prevalence rates of asthma (16%), allergic rhinitis (15%), and eczema (2.5%) in this group are little different from those of the Australian population.⁸ Only a few patients had RXN3 reactions to more than one insect type. This supports the belief that, in Queensland, the specific allergens are unique for the individual; thus, if patients have had RXN3 reactions to one insect type, they are at low risk of experiencing a similar response to an insect of a different species.

The *Vespula* sp and hornets are not found in tropical and subtropical climates. Many patients in North America have shown allergic cross-reactivity to *Vespula*, hornets, and paper wasps.⁹ This study confirms an earlier study¹ that showed that testing with the Albay paper wasp venom (Hollister-Stier, Spokane, WA) together with specific RAST determination fails to detect specific allergic antibodies in a significant number of patients who have clearly had systemic allergic responses to paper wasp stings in Queensland. This applies not only to stings from the *Ropalidia* sp but also to stings from the *Polistes* sp. On the other hand, the honeybee venom preparation appears to be highly accurate in this population.

In contrast to a previous report,¹⁰ asthma does not appear to be a risk factor in the development of RXN3 reactions; in this cohort of patients, 71% experienced RXN3 reactions compared with 67% of the entire group. However, asthma was a more common expression in these patients than in the non-asthmatic patients with RXN3 reactions.

Anaphylactoid (ie, non-IgE antibody-mediated) reactions, usually referred to as toxic,¹¹ can occur following multiple stings at one time. The number of stings needed to cause toxic reactions is debatable. Only 8 of the entire group reported at least 40 or more stings at one time; 2 of these patients were

attacked by a swarm of honeybees. Of those tested, all but 1 who exhibited anaphylaxis showed a positive specific venom response. The negative responder had multiple stings from *Ropalidia* sp. Thus, in that instance, the response may have been falsely negative. Since this series of patients was closed, another patient presented with an RXN3 reaction following an attack by a swarm of bees, but this reaction proved negative on testing; nevertheless, this study supports the notion that patients who experience RXN2 or RXN3 reactions following multiple stings at once should be considered to have had a specific IgE antibody response unless proven otherwise.

It is a general principle that a patient will not mount a significant allergic response to a reagent on its initial exposure. This study indicates that a small number of the patients have apparently experienced either an RXN2 or RXN3 reaction to a sting or bite from an insect not previously encountered. It is possible that some of these had had stings and had forgotten about them. This however does not seem to apply to the young children in this group (ie, their parents were adamant that there were no prior stings). It seems possible that patients who had had stings from a different type of insect may possess venom proteins common to both types. Indeed, a number of patients in this study with systemic reactions to both paper wasp and ant stings who have undergone immunotherapy with the wasp venom extract have tolerated subsequent stings from both insect types. Unfortunately, specific testing for these particular ant venoms is currently unavailable.

β -Adrenergic antagonists and probably ACE inhibitors can potentiate allergic reactions.^{12,13} It is thus possible that the 10 patients with RXN3 reactions who were taking β -adrenergic antagonists and the 4 who were taking ACE inhibitors when the adverse reactions occurred and who had not previously had RXN3 reactions may not have had such a severe reaction.

In summary, anaphylactic events from insect stings show the same clinical features as those from other sources (ie, foods and drugs). Typically, the reactions begin within 5 to 10 minutes of exposure to the allergen and peak rapidly. Major systemic reactions can develop independently of the site of the insect sting or bite. Most patients who experience these types of responses have never previously had a minor systemic response, but many of those who had had a minor reaction progressed to a major one. Asthma was a feature in many who had never previously experienced a reaction; however, asthmatic patients had a higher incidence of an asthmatic response. As a general rule, systemic reactions appear to be confined to a specific species for the individual patient. Those who had experienced RXN3 reactions from multiple stings at one time should undergo specific venom testing, because many have experienced true anaphylaxis and not a toxic response. Atopic patients or those who have had anaphylaxis from other reagents do not appear to be at an increased risk of developing anaphylaxis from insects. Finally, consideration must be given to the role of β -adrenergic

antagonists and possibly ACE inhibitors in patients with systemic reactions.

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