BMJ Best Practice

Insect bites and stings

The right clinical information, right where it's needed

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Summary

- Reactions range from local irritation and mild swelling to gross oral swelling and difficulty in breathing.
- Treatment is usually entirely supportive, aimed at alleviating immune reactions. Severe reactions and anaphylaxis require immediate intervention to maintain airway patency and prevent cardiovascular collapse. Injected adrenaline (epinephrine) is the mainstay treatment of severe reactions.
- Fatal spider bites are exceedingly rare. Most spider bites can be treated with supportive care.
- Guidelines are mainly based on clinical experience and expert consensus, owing to the variability and unpredictability of reactions.
- There is a risk of late-phase (delayed) anaphylactic reactions.
- Primary care physicians should be notified of severe reactions involving their patients.

BASICS

Definition

The term 'insect' refers to a distinct taxonomic class. However, it is used generically throughout this monograph to represent a multitude of bugs, including arachnids. Encounters with insects or arachnids can result in bites or stings.

Bites stem from puncture with a proboscis (animal head appendage) or fangs, or from chewing with mouth parts. Depending on the source, bites range from being immediately painful to completely unfelt, and health consequences may be caused by local trauma, the injection of various substances (irritants, venoms, poisons, toxins, anaesthetics, enzymes, anticoagulants), disease transmission, secondary infection, or systemic allergic reactions (rare).

Stings from insects are administered from ovipositors (animal organs used for laying eggs that may also inject various noxious substances) or modified ovipositors (e.g., barbed stingers of bees
[Fig-1]

[Fig-2]

and wasps). Stings are almost always immediately painful. Reactions range from local irritation and swelling to life-threatening anaphylactic reactions.

Epidemiology

Owing to lack of relevant reporting mandates, there are no clear epidemiological statistics on insect bites and stings. Biting and stinging insects are globally ubiquitous (with the exception of Antarctica) but are more common in warmer climates and during summer months. In the summer, more encounters are probably due to people being outdoors and bugs being more plentiful. However, exposures may present to practitioners at any time of the year. Some insects are more active during certain times of the day. The incidence of spider bites is unknown, but it is likely that the majority of patients claiming to have been bitten by spiders are in fact suffering from local skin infections or other reactions not due to spider bites.[8] [9] Geographical distribution of some species (e.g., fire ants and brown recluse spiders) has expanded greatly in recent decades.

Serious anaphylactic reactions account for at least 40 deaths annually in the US.[10] It is estimated that only about 0.4% to 0.8% of children and 3% of adults will manifest potentially life-threatening systemic reactions if they experience an insect sting.[11] [12] Historically, about half of fatal reactions have occurred in people without any previous allergic reactions to stings.[13]

Aetiology

Bites occur from the mouthparts of insects as they attempt to gain meals, or as they assume defensive postures. Common biting insects include mosquitoes, ticks, kissing bugs (assassin bugs), and some flies (including horseflies). Insects like mosquitoes bite by introducing a proboscis into the skin. Horseflies use a chewing mechanism.

Stings result from defensive or offensive attacks, usually via modified ovipositors (stingers) of bees and wasps,
[Fig-6]

physical structures (e.g., barbs from honeybees

[Fig-2]

[Fig-1]

), or injection of noxious chemicals (e.g., from fire ants).

Medically important spider bites are the result of the spider defending itself. Bites most commonly come when the spider is being pressed or crushed against skin inadvertently. Black widow spiders
[Fig-3]

[Fig-7]

[Fig-8]

often reside in dark corners of garages, tool sheds, cabinets, well houses, and other rarely used sites. Bites occur when people reach around blind corners and encounter the spider. Recluse spiders
[Fig-9]

[Fig-10]

prefer dry, dark areas such as wardrobes and vehicle boots. The common cause of a spider bite is putting on clothing containing a spider, and pressing the spider against the skin.

Pathophysiology

Chemicals involved in stinging are more likely to produce severe reactions and anaphylactic responses than bites. Hymenoptera venom has been studied extensively.[14] [15] [16] There is significant similarity in the venoms from hornets, wasps, and yellow jackets. Patients with severe reactions to the stings of one of these insects have a likelihood of sensitivity to the stings from another. Wasp venoms contain molecules such as phospholipases A and B, hyaluronidases, and invertebrate neurotoxin. Bee venoms are immunochemically distinct from wasp venoms, and therefore sensitivity to one family does not imply sensitivity to the other. Bee venoms contain hyaluronidase, phospholipase A2, acid phosphatase, meletin, and other kinins. Reactions can result from sensitivity or allergies to any of these components. Disease transmission is unlikely from stings.

The release of histamine (a potent vasodilator) in response to venom exposure accounts for the majority of reactions. In local reactions, this leads to swelling, oedema, and pain.
[Fig-11]

[Fig-12]

IgE-mediated histamine release in severe reactions can result in hypotension. The relatively high concentration of histamine receptors in the skin, lungs, and GI mucosa leads to symptoms felt predominately in these organ systems.

Fire ant stings contain only small amounts of venom, but it is contained in an unusual suspension of alkaloid toxins. These components cause localised tissue necrosis and the formation of a characteristic sterile pustule about 24 hours after the sting.
[Fig-13]

This is not an infectious process and vesicles should be left intact. If opened, these lesions can serve as a portal for secondary infection. They should be kept clean and covered.[17] [18]

Black widow spider species venoms contain neurotoxins that cause pre-synaptic release of neurotransmitters including acetylcholine, noradrenaline (norepinephrine), dopamine, glutamine, and enkephalins. Bites can cause effects both locally and systemically including pain, muscle cramps, diaphoresis, tremors, paraesthesias, nausea/vomiting, and headache. Autonomic instability can lead to profound hypertension and tachycardia.[19] [20]

Recluse spider bites contain enzymes that cause local tissue destruction.
[Fig-14]

[Fig-15]

The enzyme sphingomyelinase D is thought to be the most clinically relevant. It is directly toxic to skin and tissue, activates other degradative enzymes, and activates complement and other inflammatory components. These wounds are at high risk of secondary infections.[19] [21]

Anaphylaxis is an IgE-mediated hypersensitivity response to the exposure of an antigen in a pre-sensitised individual.[22] [23] [24] Exposure to the antigen leads to rapid mast cell degranulation and release of histamine (and other vasoactive substances/kinins). This causes capillary leak and oedema with myriad manifestations. Anaphylactoid reactions are non-IgE-mediated idiosyncratic reactions to certain exposures. They do not require pre-sensitisation.

Classification

Biological taxonomy

Insects are members of the class Insecta within the phylum Arthropoda. Insect bodies are divided into a head, thorax, and abdomen, and they have 3 pairs of legs.

Arachnids compose the class Arachnida, within the Arthropoda phylum, and are characterised by a 2-segment body (cephalothorax and abdomen) and 4 pairs of legs. Spiders and ticks are different orders within the arachnid class.

Biting insects and arachnids

Common biting insects include mosquitoes, kissing bugs (assassin bugs), and some flies (including horseflies). Biting arachnids include ticks, black widow spiders (*Latrodectus* species),
[Fig-3]

and recluse spiders (*Loxosceles* species).
[Fig-4]

Stinging insects

Stings most commonly result from the order Hymenoptera. The most common culprit families include:

- Bees (Apidae): bumblebees, Africanised honeybees ('killer bees'), domestic honeybees
  [Fig-1]

- Wasps (Vespidae): paper wasps, hornets, yellow jackets [Fig-5]
- Ants (Formicidae): fire ants, harvester ants.

A honeybee can sting only once; the barbed stinger remains in the victim, and the bee eviscerates itself as it struggles to escape. Most other insects are capable of multiple stings. An Africanised honeybee sting is the same as a regular honeybee sting, but the more aggressive nature of the Africanised bees can lead to a swarm attack with large numbers of stings resulting.

Reaction to bite or sting

Local reactions

- There may be localised irritation or swelling to physical or chemical properties of the bite or sting.

Systemic reactions

- Life-threatening reactions are rare, but require prompt treatment to avert serious outcomes.
- Anaphylaxis (IgE mediated) or anaphylactoid reactions (non-IgE mediated) resulting from exposure to bites or stings may be immediately life threatening.
- Clinical presentation and management of anaphylactic and anaphylactoid reactions are similar, and both will be considered as the same entity for the purposes of this monograph.
- There are 3 presentations consistent with anaphylaxis:
    - Acute onset of a reaction that includes the skin and involvement of the respiratory tract and/or a decrease in blood pressure
    - Rapid onset of a reaction after exposure to a likely allergen that involves 2 organ systems (respiratory tract, skin, decrease in blood pressure, and/or persistent gastrointestinal symptoms)
    - Reduced blood pressure after exposure to a known allergen.[1] [2] [3]
- Injected adrenaline (epinephrine) is the mainstay treatment of severe reactions. There are no absolute contraindications to adrenaline (epinephrine) treatment in severe reactions.

Secondary infection

- Infection may develop at the site of a bite or sting.
- This is not inflicted by the insect, but is a result of the breakdown of the normal skin barrier.

Disease transmission

- Mosquito bites may transmit malaria, dengue fever, yellow fever, Zika virus, and some forms of encephalitis.
- Tick bites may transmit Lyme disease and Rocky Mountain spotted fever.

Serum sickness

- Although uncommon, this delayed reaction may occur about 1 week after envenomation.
- Symptoms include fever, myalgias, arthralgias, rash, adenopathy, and headache. Very rarely, vasculitis and immunological complications can lead to glomerulonephritis, Guillain-Barre syndrome, haemolytic anaemia, thrombotic thrombocytopenic purpura, transverse myelitis, optic neuritis, and other neuropathies.[4] [5] [6] [7]

Primary prevention

Primary avoidance of contact is the most important factor in preventing bites and stings. Common sense goes a long way. It is important to try to avoid exposure at the times the bugs are most active. For example, mosquitoes are most active around dawn and dusk. Avoidance of perfumes and scented cosmetics seems wise as some insects are attracted to these odours. There are many commercially available topical sprays and lotions, as well as area-deterrents (e.g., foggers, coils). Wearing long sleeves, long trousers, and head nets can prevent most bites; some clothing also comes pre-treated with insect repellents.[26] It is also advisable to look properly before reaching around blind corners or into the back of cabinets to help avoid spider bites and wasp encounters; wearing gloves when it is necessary to reach into places with limited visibility is also helpful. Insect repellents are available in sprays, lotions, and liquids, although these are not often useful at preventing hymenoptera or arachnid stings and bites.

The US National Institute for Occupational Safety and Health (NIOSH) has published information for employers and their workers about the risk of exposure to insects and scorpions, and prevention of stings and bites. [CDC: insects and scorpions]

Screening

The low incidence of severe reactions makes screening for sensitivity to insect bites and stings impractical. The frequency of false-positive and false-negative results further decreases the practicality of screening. Patients with severe reaction should be evaluated by their primary doctor regarding referral to allergists/immunologists for further testing. This may involve skin testing, in vitro testing, and possibly desensitisation therapy. Screening is covered in detail in guidelines from AAAAI/ACAAI (American Academy of Allergy, Asthma & Immunology; American College of Allergy, Asthma & Immunology).[29]

Secondary prevention

A prescription for 2 adrenaline (epinephrine) auto-injectors must be given after any episode of anaphylaxis.[3] [48] The patient or caregiver should carry both at all times and be familiar with their use.[2] For children at risk of anaphylaxis, the adrenaline (epinephrine) auto-injectors should be prescribed in conjunction with a personalised, written emergency plan.[2] [49] [American Academy of Pediatrics: allergy and anaphylaxis emergency plan]

Immunotherapy (i.e., desensitisation therapy) with extracted insect venoms may be used to decrease the severity of reactions in patients who are highly sensitive to certain insect stings.[1] This can significantly improve quality of life in susceptible patients, but should only be performed by qualified physicians with training in this field as exposure to the venoms carries a small but real risk of serious adverse reactions during treatment.[63]

Tetanus-prone wounds (wounds that are deep, dirty, necrotic, or from severe recluse spider envenomation) should receive tetanus prophylaxis if the last immunisation was more than 5 years ago. Wounds not prone to tetanus (e.g., stings, black widow bites) should trigger tetanus prophylaxis if the last immunisation was more than 10 years ago.

Case history

Case history #1

A healthy 30-year-old woman presents for accident and emergency department triage half an hour after direct contact of her right foot with some fire ants at a birthday party. She describes experiencing swelling in her right foot and an itchy, raised rash on her legs, belly, back, and neck within minutes of the exposure. She reports that this had happened to her before, so she immediately took 100 mg of diphenhydramine. She says she decided to go to the hospital because she felt like her tongue was swelling and she was becoming breathless. On examination her vital signs are BP 121/85 mmHg, heart rate 132 bpm, respiratory rate 26, and O2 saturation level 99%. Skin examination shows a diffuse, raised, red erythematous rash. Examination of head, eyes, ears, nose, and throat shows minimal tongue swelling but no stridor or drooling. Lung examination reveals rapid respirations with good air movement but trace wheezes. Cardiac examination shows tachycardia, but no murmurs and equal distal pulses. Abdominal examination shows a soft, non-tender abdomen with active bowel sounds. Neurological examination is non-focal except for an anxious alert sensorium.

Case history #2

A 9-year-old boy is brought to the accident and emergency department after being stung by a bee at a picnic. He is crying hysterically. After 15 minutes of calming him down, examination reveals a swollen, tender upper lip but no tongue swelling, no drooling, no stridor, no rash, and no other complaints.

Other presentations

Patients may not always know they have been bitten or stung. Many present to medical practitioners looking for diagnosis and treatment of rashes, pustules, swelling, or skin infections. Some patients may be asymptomatic but are concerned about the possibility of transmission of diseases from bites or stings. Development of local or systemic reactions may develop hours to days after the original insult. Black widow spider bites may present with abdominal cramping mimicking an acute surgical abdomen. Rarely, progressive local tissue necrosis may occur subsequent to a recluse spider bite.

Step-by-step diagnostic approach

Diagnosis is almost always based on history, physical examination, and clinical judgement. The first step is discerning whether the reaction is severe (life-threatening anaphylaxis/anaphylactoid reactions). Any suspicion of anaphylaxis should prompt immediate action. Less severe reactions can be treated with comforting measures. Little, if any, formal testing is helpful in acute encounters; often, definitive diagnosis is not achievable acutely.

Risk stratification

A risk stratification approach is critical to guiding treatment and must be made on clinical grounds alone. It is important to remember to re-evaluate the patient frequently, as their condition may deteriorate quickly.

Primary ABC assessment: abnormalities in any of these aspects signal the need for immediate intervention (immediate treatment and preparation for transfer to an accident and emergency department or advanced-care setting):

- Airway patency and ability to maintain airway
- Evidence of mouth or tongue swelling
- Presence of stridor heard over the neck or throat
- Swallowing of own secretions or the presence of drooling
- Problems with spontaneous breathing, dyspnoea, wheeze, cough, air hunger
- Palpable pulses, pulse rate, and rhythm
- Skin flushing (vasodilated) or skin pallor with clamminess (vasoconstricted).

Determining severity of initial reaction

Local reactions

- Usually present with oedema and pain.
  [Fig-11]
- Acutely, spider bites may show one or two small fang marks.
- Non-anaphylactic or non-anaphylactoid allergic reactions to bites and stings are characterised by pain, wheal and flare formation, warmth, and pruritis at the bite/sting site.[27]
  [Fig-12]

  Although they may have the appearance of cellulitis, there is almost never infection initially
- Usually self-limiting and confined to skin and soft tissues.
- Abscesses usually have palpable fluctuance.
- May be difficult or impossible to distinguish between a bite and cellulitis acutely.
- If there is no improvement after a few days, a secondary infection may have developed.
- Late cutaneous allergic reactions are common. Skin may remain sensitive for days to weeks following a sting. Permanent skin discoloration can occur in some individuals.

Systemic reactions

- Severity of anaphylaxis can range from any combination of urticaria and angio-oedema, bronchospasm, oedema of the large airway, or hypotension through to life-threatening anaphylaxis involving respiratory failure and cardiovascular collapse.[1] Anaphylaxis is likely when any 1 of the following 3 criteria is satisfied:[1] [2] [3]
  - Acute onset of a reaction that involves the skin (mucosal tissue), and respiratory compromise and/or reduced blood pressure
  - The rapid onset of a reaction after likely allergen exposure that involves 2 or more organ systems (respiratory tract, skin, decrease in blood pressure, and/or persistent gastrointestinal symptoms)
  - Reduced blood pressure after exposure to a known allergen.

Historical factors

In addition to determining the symptoms of local reactions and symptoms of anaphylactic/anaphylactoid reactions, it is important to elicit historical factors such as time course of onset, behaviours that

predispose to insect exposure, and witnessing of bites. The history should include the what, where, when, and how of the sting. It should include enquiring about the number of exposures, discovering whether the bite or sting was witnessed, determining how much time has passed since the encounter, eliciting what the patient was doing during the exposure, and determining whether the patient had taken any medications or performed any self-treatments.

Time course of onset

- Some bites and stings (e.g., horsefly bites, wasp stings) result in immediate pain and local injury/bleeding. Others, like tick bites, are not felt immediately. Rarely, recluse spider bites [Fig-10]

  can develop slow, but relentlessly progressive, local tissue necrosis. [Fig-14]

  [Fig-15]

  Fire ant stings cause localised tissue necrosis and the formation of a characteristic sterile pustule about 24 hours after the sting.[17] [18] [Fig-13]
- There may be delayed reactions to substances introduced from the bite/sting and symptoms, which may not be noticed until hours/days later. Infections transmitted by the bite are also often not felt immediately.
- Serum sickness is an uncommon delayed reaction, usually occurring about a week after envenomation. Symptoms include fever, myalgias, arthralgias, rash, adenopathy, and headache. Very rarely, vasculitis and immunological complications can lead to glomerulonephritis, Guillain-Barre syndrome, haemolytic anaemia, thrombotic thrombocytopenic purpura, transverse myelitis, optic neuritis, and other neuropathies.[4] [5] [6] [7]

Past medical history may reveal previous exposures (history of severe reaction, history of atopy/allergies, multiple previous exposures). It may also reveal previous infections or immunocompromising states, fevers and chills, and/or contacts with infectious sources (pointing towards infection). Immunocompromising states place patients at higher risk of secondary infection following bites/stings. It is also important to determine previous cardiovascular disease that can be aggravated by both the insult and treatment.

Drug history may reveal use of medications that blunt treatment effects (e.g., beta-blockers).

Physical examination

Most bites and stings cause only dermatological manifestations of pain, itching, or rash. However, a directed physical examination must first focus on more immediate threats and evaluate the respiratory, cardiovascular, and gastrointestinal systems. Only after ensuring that there are no immediate life-threatening effects should the attention be turned to the dermatological examination.

Secondary assessment: if the patient appears stable, the directed examination must include assessment of at least the following systems. Anaphylaxis is diagnosed if there are abnormalities in 2 or more of the 4 systems listed below.

- Cardiovascular: assess for heart rate, rhythm, murmurs, blood pressure, pulse strength, capillary refill time, colour of extremities, temperature of extremities.

- Respiratory: assess for respiratory rate, work of breathing, oxygen saturation, stridor (usually inspirational, heard over neck), wheeze (usually expirational, heard over lungs), rales or crackles (sign of cardiovascular failure).
- Neurological: assess for alertness, sensorium clarity, autonomic nervous system instability (fluctuating pulse or blood pressure, localised or generalised diaphoresis), tremors, cramps, fasciculations, localised pain, generalised pain. Black widow spider bites may cause neurological effects.
  [Fig-3]

  [Fig-8]
- Gastrointestinal/genito-urinary: assess for presence, location, and quality of abdominal pain (rigidity, guarding, rebound tenderness, uterine cramping); presence of bowel sounds. Black widow spider bites may present with acute abdominal cramping mimicking an acute surgical abdomen.

Dermatological assessment

- Is the bite or sting site visible? Is there a large skin defect? Are fang marks visible?
- What is the location and number of bites or stings?
- Is there a stinger still present?
- Is the rash raised and itchy (wheals/urticaria are typical for bee and wasp stings)?
  [Fig-12]
- Is there local redness or swelling?
  [Fig-11]
- Is there diffuse or generalised skin rash?
- Does the appearance resemble cellulitis?
- Is there evidence of abscess formation (subcutaneous or deep-tissue fluctuance)?
- Is there a shape or pattern to the rash? Linear raised rash may represent urticating caterpillars, or a contact dermatitis (like poison ivy) rather than an insect sting.
- Are pustules present? A ring of small pustules is indicative of fire ant stings.
  [Fig-13]
- Bullseye-shaped rash following tick bites is indicative of Lyme disease.
  [Fig-16]
- Involvement of palms and soles following tick bites is indicative of Rocky Mountain spotted fever.
- Is there necrosis? Rarely, recluse spider bites
  [Fig-10]

  can develop slow, but relentlessly progressive, local tissue necrosis.
  [Fig-14]

  [Fig-15]

Tryptase levels

In unclear cases, drawing blood to obtain tryptase levels within 1 to 5 hours of onset of symptoms can verify that mast cells were activated and may help establish a diagnosis of anaphylaxis.

Elevated baseline serum tryptase is closely correlated with the risk of severe anaphylaxis to insect stings.[1] [3]

Sensitivity testing

Skin testing, including venom skin tests or radioallergosorbent tests, can be helpful for determining whether individuals who have suffered a previous severe attack are good candidates for desensitisation treatment.[1] [14] [28] This should not be done in the acute setting, as depletion of mediators from the reaction may result in a false-positive response. Skin testing should be delayed for 2 to 6 weeks after the acute event. The low incidence and idiosyncratic nature of severe (anaphylactic or anaphylactoid) reactions makes generalised screening impractical.

Risk factors

Strong

occupational or recreational exposure to insects

- People are more likely to get bitten or stung if they engage in behaviours that place them in close contact with culprit bugs. Camping, gardening, landscaping, and spring-cleaning are high-risk activities.
- Bites and stings occur more often in the spring and summer months, when the bugs are more prevalent and people are outside more.

geographical exposure to insects

- Many spiders and some insects are found in distinct geographical locales, although the ease and frequency of travel in recent decades makes it impossible to rule out exposure based on geography alone.
- Spider bites usually result from reaching blindly into the environment of a spider or accidentally pressing the skin against the spider (e.g., cleaning the garage or putting on clothing that has been kept in storage).

previous history of anaphylactic (or anaphylactoid) response

- Patients who have experienced severe reactions to bites or stings in the past have a high likelihood (although not guaranteed) of experiencing similar reactions to future exposures. However, there is an outgrowing phenomenon (severe reactions in childhood do not predict adult sensitivity as strongly as previous reactions in adulthood).[25]

History & examination factors

Key diagnostic factors

presence of risk factors (common)

- Occupational, recreational, and geographical exposure to insects can increase the risk of being bitten or stung. History of anaphylactic or anaphylactoid responses increases the risk of severe allergic reactions.

witnessed bite/sting (common)

- Only when encounters are witnessed can diagnosis of bite or sting be made with certainty. [Fig-1]

It is likely that the majority of patients claiming to have been bitten by spiders are in fact suffering from local skin infections or other reactions not due to spider bites.[8] [9]

local oedema (common)

- Swelling at the bite/sting site is usually present and develops acutely.
[Fig-11]

May also be a sign of cellulitis if it develops a few days after the sting, although secondary infection is rare.

local pain (common)

- May or may not be present or may be a late sign.

local warmth (common)

- May or may not be present.

local markings (common)

- Acutely, spider bites may show one or two small fang marks. Stinging ants may leave circle of stings because they bite with their mouth and rotate while stinging with abdominal organs.
[Fig-13]

pruritus (common)

- Characteristic of non-anaphylactic/non-anaphylactoid allergic reactions.

wheal and flare (common)

- Such formation is characteristic of non-anaphylactic/non-anaphylactoid allergic reactions.
[Fig-12]

skin pallor (common)

- May indicate vasoconstriction in cases of severe allergic response.

clamminess (common)

- May indicate vasoconstriction in cases of severe allergic response.

signs of airway compromise (uncommon)

- Sign of life-threatening anaphylactic/anaphylactoid response. May occur due to oropharyngeal oedema. Stridor is usually inspirational and may be heard over the neck or throat. Drooling may occur when patients are unable to swallow their own secretions. Air hunger may also be present.

oropharyngeal oedema (uncommon)

- There may be swelling of the lips, mouth, tongue, or pharynx, which may be a sign of life-threatening anaphylactic/anaphylactoid response. This may lead to airway obstruction.

dyspnoea (uncommon)

- Patients may be working hard to breathe or breathing fast in life-threatening anaphylactic/anaphylactoid responses.

rales/crackles/wheeze (uncommon)

- May be a sign of cardiovascular failure in life-threatening anaphylactic/anaphylactoid responses. Wheeze is usually expirational and heard over the lungs.

tachycardia (uncommon)

- May be a sign of autonomic instability and life-threatening anaphylactic/anaphylactoid response.[19] [20]

irregular pulse (uncommon)

- May be a sign of life-threatening anaphylactic/anaphylactoid response.

skin flushing (uncommon)

- May indicate vasodilation in cases of severe allergic response.

altered alertness (uncommon)

- May indicate severe allergic response. May be due to the neurological effects of black widow spider bites.
  [Fig-3]

  [Fig-8]

Other diagnostic factors

pustules (common)

- May be present. A ring of small pustules is indicative of fire ant stings.
  [Fig-13]

bullseye-shaped rash (common)

- Following tick bites, this is indicative of Lyme disease.
  [Fig-16]

rash on palms and soles (common)

- Following tick bites, this is indicative of Rocky Mountain spotted fever.

linear raised rash (common)

- May represent urticating caterpillars or a contact dermatitis (like poison ivy) rather than an insect sting.

hypertension (common)

- May be a sign of autonomic instability and life-threatening anaphylactic/anaphylactoid response.[19] [20] May be due to the neurological effects of black widow spider bites.

fever (common)

- Fever may be present due to local infection, cellulitis, or serum sickness (uncommon delayed reaction usually occurring about a week after envenomation).

muscle cramp (common)

- May indicate severe allergic response. Myalgia may also occur due to serum sickness (uncommon delayed reaction usually occurring about a week after envenomation). May be due to the neurological effects of black widow spider bites.

diaphoresis (common)

- May indicate severe allergic response. May be due to the neurological effects of black widow spider bites.

tremor (common)

- May indicate severe allergic response. May be due to the neurological effects of black widow spider bites.

paraesthesia and/or fasciculations (common)

- May indicate severe allergic response. May be due to the neurological effects of black widow spider bites.

generalised pain (common)

- May indicate severe allergic response. May be due to the neurological effects of black widow spider bites.

nausea/vomiting (common)

- May indicate severe allergic response.

headache (common)

- May indicate severe allergic response. It may also indicate the presence of serum sickness (uncommon delayed reaction usually occurring about 1 week after envenomation).

abdominal pain (common)

- May indicate severe allergic response.
- Black widow spider bites may present with acute abdominal cramping mimicking an acute surgical abdomen.

hx of immunocompromised state (common)

- Places patients at higher risk of secondary infection following bites or stings.

hx of cardiovascular disease (common)

- Cardiovascular disease can be aggravated by both the insult and the treatment. Beta-blockers and other medications can interfere with medications used to treat the insult.

abscess (uncommon)

- May be present and usually has palpable fluctuance.

necrosis (uncommon)

- Rarely, recluse spider bites
  [Fig-10]

  can develop slow, but relentlessly progressive, local tissue necrosis.
  [Fig-14]

  [Fig-15]

Fire ant stings cause localised tissue necrosis and the formation of a characteristic sterile pustule about 24 hours after the sting.[17] [18]
[Fig-13]

cough (uncommon)

- Allergic reactions may manifest as cough (e.g., variant asthma).

hypotension (uncommon)

- IgE-mediated histamine release in severe reactions can result in hypotension. May be due to the neurological effects of black widow spider bites.

arthralgia (uncommon)

- May occur due to serum sickness (uncommon delayed reaction usually occurring about a week after envenomation).

adenopathy (uncommon)

- May occur due to serum sickness (uncommon delayed reaction usually occurring about a week after envenomation).

Diagnostic tests

1st test to order

Test	Result
clinical diagnosis • Any suspicion of anaphylaxis should prompt immediate action. • Little, if any, formal testing is helpful in acute encounters; often definitive diagnosis is not achievable acutely.	**diagnosis is almost always based on history, physical exam, and clinical judgement**

Other tests to consider

Test	Result
FBC • Cellulitis or infected bites/stings will cause elevation of WBC levels.	**normal or elevated WBC**
serum tryptase • In unclear cases, drawing blood to obtain tryptase levels within 1 to 5 hours of onset of symptoms can verify that mast cells were activated and may help establish a diagnosis of anaphylaxis. • Elevated baseline serum tryptase is closely correlated with the risk of severe anaphylaxis to insect stings.[1] [3]	**normal or elevated**

Test	Result
sensitivity testing • Skin testing, including venom skin tests or radioallergosorbent tests, can be helpful for determining whether individuals who have suffered a previous severe attack are good candidates for desensitisation treatment.[1] [14] [28] • This should not be done in the acute setting, as depletion of mediators from the reaction may result in a false-positive response. Skin testing should be delayed for 2 to 6 weeks after the acute event. The low incidence and idiosyncratic nature of severe (anaphylactic or anaphylactoid) reactions makes generalised screening impractical.	**quantifiable response to specific antigen**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Cellulitis	• Hx of previous episodes of cellulitis; ulcer or wound; dermatosis; tinea pedis interdigitalis; lymphedoema; venous insufficiency or chronic leg oedema.	• FBC: elevated WBC. • Blood cultures: positive. • Purulent focus culture: positive.
Periorbital and orbital cellulitis	• Hx of previous sinus infection, lack of *Haemophilus influenzae* type b vaccination; stye or chalazion; recent eye trauma.	• FBC: elevated WBC. • Blood cultures: positive. • Microbiology eye swab: positive.
Abscess	• Red, tender, fluctuant mass; fever; purulent drainage.	• Incision and drainage yields pus/necrotic material. Culture can identify infectious organism.
Necrotising fasciitis	• Hx of immunosuppression due to chronic illness (e.g., diabetes mellitus, alcoholism); cutaneous trauma or ulcerative skin conditions; varicella zoster infections; hospitalisation.	• FBC: elevated WBC. • Urea and creatinine: elevated. • Sodium: normal or reduced. • Serum creatinine phosphokinase: elevated. • Blood cultures: positive. • ABG analysis: hypoxaemia and acidosis.
Folliculitis	• Recent hx of immersion in spa water; darkly pigmented male with curly hair; recent hx of shaving; umbilicated flesh-coloured papules.	• Gram stain: gram-positive cocci typical of *Staphylococcus aureus* infection. • Potassium hydroxide preparation: presence of hyphal forms suggestive of dermatophyte infection.

Condition	Differentiating signs / symptoms	Differentiating tests
Basal cell carcinoma	• Hx of UV radiation, sun exposure, x-ray exposure, arsenic exposure, xeroderma pigmentosa, Gorlin-Goltz syndrome, or transplant. • Papules with associated telangiectasias; plaques, nodules, and tumours with rolled borders; small crusts and non-healing wounds; non-healing scabs; pearly papules or plaques. Unlike bites and stings, these lesions do not appear acutely.	• Shave/punch biopsy: growth of nest(s) of varying size and shape.
Squamous cell carcinoma of the skin	• Hx of UV exposure, older age, immunosuppression, fair skin, human papillomavirus, hereditary skin conditions, exposure to ionising radiation/arsenic/ tar, actinic keratosis, male sex. Unlike bites and stings, these lesions do not appear acutely. • Erythematous papules or plaques; thin flesh-coloured or erythematous plaques/dome-shaped nodules; exophytic, fungating verrucous nodules or plaques.	• Biopsy: keratinocyte atypia.
Kaposi's sarcoma	• HIV infection; immunosuppressive therapy; transplantation, Central African ethnicity (e.g., from Uganda, Malawi, Zambia, Zimbabwe); human herpesvirus-8 infection. • Skin lesions may be multifocal, asymmetrically distributed, non-pruritic, varying in size (ranging from several millimetres to centimetres in diameter) and colour (pink, red, purple, brown, or blue), papular, nodular, plaque-like, bullous-like, fungating with skin ulceration and secondary infection, indurated (woody), or hyperkeratotic.	• HIV test: positive. • Biopsy: characteristic vascular lesion.

Condition	Differentiating signs / symptoms	Differentiating tests
Local trauma	• Hx of trauma.	• X-ray: may show evidence of trauma.
Intra-abdominal processes (differential of black widow spider bite)	• Abdominal tenderness with guarding or rebound tenderness. Bowel sounds may be absent. • May have risk factors for mesenteric ischaemia, gastroenteritis, bowel obstruction, etc.	• X-ray: may show dilated bowel loops. • FBC may be elevated in infectious process. Lactate may be elevated in mesenteric ischaemia. • Ultrasound/CT/MRI may show pathology.
Compartment syndrome of the abdomen	• Hx of excessive fluid resuscitation (>5 L in 24 hours), massive blood transfusion (>10 units in 24 hours), recent abdominal infection (especially peritonitis), haemoperitoneum, ileus, abdominal distention, oliguria.	• Trans-bladder measurement of intra-abdominal pressure: elevated. • ABG analysis: metabolic acidosis or mixed metabolic and respiratory acidosis.
Compartment syndrome of extremities	• Hx of trauma, bleeding disorder, compression support, thermal injury, intravenous infusion, venous obstruction, sports playing. Loss of muscle function, pain, pressure (tightness), paraesthesia, pulselessness, pallor, paralysis.	• Compartment pressure: differential pressure ≤20 mmHg. • Serum creatinine kinase: elevated. • Urine myoglobin: elevated.
Muscle spasm	• Episodic occurrence. Hx of muscle strain or trauma.	• Clinical diagnosis.
Migraine	• FHx of migraine. Risk factors include childhood motion sickness; caffeine intake; high altitude; female gender; menstruation; divorced, widowed, or separated; obesity; habitual snoring; stressful life events; overuse of headache medications; lack of sleep.	• Clinical diagnosis.
Subarachnoid haemorrhage	• Hx of hypertension, smoking, or autosomal dominant polycystic kidney disease; FHx of subarachnoid haemorrhage; photophobia; altered mental status.	• CT head: hyper-dense areas in the basal cisterns, major fissures and sulci. • Lumbar puncture: bloody CSF (xanthochromia).

Condition	Differentiating signs / symptoms	Differentiating tests
Acute myocardial infarction	• Cardiac risk factors, chest pain, SOB, nausea, diaphoresis, eliciting factors.	• ECG: ischaemic changes. • Cardiac enzymes: positive.
Toxic plant ingestion	• Hx of recent ingestion of plant matter.	• Clinical diagnosis.
Organophosphate poisoning	• Hx of recent ingestion of insecticides; distinctive odour; incontinence; visual disturbances.	• Atropine therapeutic trial: lack of anticholinergic effects. • Plasma cholinesterase: reduced activity.
Shock	• May be cardiogenic, septic, or hypovolaemic. • Hx of recent MI, recent surgery or immobilisation, severe infection, or haemorrhage.	• Lactate >2 mmol/L (>18 mg/dL) is suggestive of tissue hypoperfusion. • ABG analysis: pH <7.35 indicates acidosis.
Acute asthma	• Hx of viral infection, exposure to cigarette smoke, exposure to allergens, atopic eczema, environmental irritants, GORD, use of oral corticosteroids, or non-compliance to asthma medication.	• PEFR: <60% of predicted value if severe.
Acute COPD	• Hx of bacterial infection; viral infection; exposure to pollutants; change in weather.	• CXR: hyper-inflation, flattened diaphragm, bullae, and a small vertical heart. • ABG analysis: respiratory acidosis and compensatory metabolic alkalosis.
Foreign body aspiration	• Sudden-onset stridor or choking; hx of foreign body in mouth (especially in young children).	• CXR may show air-trapping and hyper-expanded lung field.
Viral syndrome	• Fever, chills, myalgias, upper respiratory infection symptoms, GI symptoms.	• Clinical diagnosis.
Drug reaction	• Usually diffuse rash developing after beginning new medication.	• Symptom improvement after removal of drug.
Atopy	• Recurrent sensitivity reactions following certain exposures.	• Allergy testing: positive.
Chemical exposure or sensitivity	• Hx of recent exposure.	• Clinical diagnosis.

Diagnostic criteria

Acute diagnosis

Diagnosis is based on history and physical examination findings. Only when the bite or sting is witnessed can the diagnosis be made with certainty.

Chronic hypersensitivity

Patients who have serious reactions to bites or stings are tested for hypersensitivity.

Step-by-step treatment approach

Initial management is usually based entirely on history, examination findings, and clinical suspicion. Any indication of anaphylaxis should prompt immediate supportive care, as the disease process may progress rapidly, leading to loss of airway patency and cardiovascular collapse. Stabilisation and treatment of life-threatening conditions are most often performed simultaneously. All patients with signs of a systemic reaction, especially hypotension, airway swelling, or difficulty breathing, should receive immediate intramuscular adrenaline (epinephrine) in the anterolateral thigh.

Close monitoring is still necessary in less severe reactions (or reactions responding to treatment), as rapid deterioration or rebound from treatment is possible.[22] [24] [30] [31] Patients with severe reactions that have responded well to treatment should be observed for at least 4 to 6 hours in the accident and emergency department for the return of symptoms as medications wear off. Any return of symptoms should lead to re-treatment and is likely to indicate a need for hospital admission.

Co-existing cardiovascular disease should be noted because some treatments can stress a susceptible heart.[32] Caution and close monitoring should be used when treating these patients. However, pre-existing heart disease is not a contraindication to treatment of suspected anaphylactic reactions.

Local reactions

Supportive care

- Local pain and swelling at the site of the bite or sting can be reduced with ice pack application. The pack should have a cloth barrier between the ice and skin to prevent local tissue damage. Applying the ice pack on and off at 15-minute intervals is a common regimen.
- Surgical consultation may be needed for severe or progressive local reactions at the site of suspected brown recluse spider bites.
  [Fig-14]

  [Fig-15]

  [Fig-10]
- Fire ant pustules should be left intact. If opened, these lesions can serve as a portal for secondary infection. They should be kept clean and covered.[17] [18]
  [Fig-13]
- All skin wounds should be assessed for tetanus prophylaxis. Tetanus-prone wounds (deep/dirty/necrotic/severe recluse spider envenomation) should receive tetanus prophylaxis if the last immunisation was >5 years ago. Non tetanus-prone wounds (e.g., stings/black widow spider bites) should trigger treatment if last immunisation was >10 years ago.

Stinger removal

- Retained stingers should be removed because they may still contain venom.
  [Fig-1]
- The venom sac is emptied within 30 seconds of exposure; therefore, the most important thing about removing the stinger is to get it out as quickly as possible.
- Traditional teaching suggests that squeezing the stinger (e.g., with tweezers) can inject more venom into the patient. The stinger should be removed by gently scraping the stinger with the edge of a plastic ID card (driver's licence or similar object). More recent research has called this

into question and suggests that time to removal is more important than method in minimising the amount of venom injected.[33]

Corticosteroids

- Corticosteroid treatment works to decrease vascular permeability and blunt the immune response to the inciting antigen.
- Effects take at least 1 hour to begin, so it is important to start treatment as early as possible.
- Corticosteroid treatment should be continued for 3 to 5 days in patients with moderate to severe reactions.
- In patients without previous corticosteroid dependency or adrenal insufficiency, there is no need to taper the dose. In patients with chronic corticosteroid use, previous corticosteroid dependency, or adrenal insufficiency, or at risk of rebound effects, carefully monitored tapering of treatment may be warranted. Dose packs or slowly decreasing doses of oral prednisolone serve this purpose. These patients must be followed closely by their physician.
- The data supporting the use of corticosteroids are limited due to difficulties in performing controlled studies. Nonetheless, corticosteroid treatment remains a current mainstay of therapy.[34]

H1 antagonists and H2 antagonists

- Antihistamines antagonise the effects of histamine release at cellular receptors, decreasing itching, erythema, and rash.
- Sedating H1 antagonists should be continued for about 3 days following an allergic reaction and can be tapered by the patient according to symptom severity.
- Non-sedating H1 antagonists can also be used.
- H2 antagonists can be used to further potentiate the antihistamine effect.
- Topical antihistamines should be used with caution, if at all. They probably do not add any beneficial effect when patients are already on systemic antihistamines. Their erratic absorption patterns can lead to anticholinergic toxicity. They can also be irritating to the skin and exacerbate dermatological symptoms.

Anti-inflammatory medications

- Treatment with non-steroidal anti-inflammatory drugs (NSAIDs) can decrease swelling and pain at the site of the bite or sting. These medications can be tapered by the patient according to their symptoms. Caution should be used in patients currently taking aspirin or anticoagulants, patients with sensitivity to these medications, or patients with risk factors for ulcers, GI bleeding, or thromboembolic disease.

Patients with black widow or recluse spider bites may require extra specific treatments.

Black widow spider bites

Most black widow spider bites produce only localised pain at the site of the wound; fatalities are exceedingly rare. The wound should be cleaned with soap and water, and tetanus status should be addressed. Elevation and application of ice packs can help alleviate pain. Mild to moderate pain often responds to paracetamol or non-steroidal anti-inflammatory drug (NSAID) treatment. Oral opioids (e.g., hydrocodone) may be needed for more severe bites or patients with low pain tolerance.
[Fig-3]

[Fig-8]

More severe bites can cause severe pain and muscle spasms in the affected limb or in a generalised fashion. Pain radiating into the chest or abdomen can confuse the diagnostic picture, and myocardial ischaemia or an acute abdomen may need to be ruled out. Patients may need high-dose intravenous opioids (e.g., morphine) for pain control. Severe muscle spasms may be relieved with benzodiazepines. Autonomic instability may lead to profound tachycardia and hypertension. These usually resolve as pain is addressed. In patients who may not tolerate these effects, beta-blockers may be a reasonable treatment.

There are several black widow spider antivenoms available commercially.[35] [36] Treatment with antivenom has historically been thought to reduce the pain and duration of symptoms, although more recent studies have shown little (if any) difference between antivenom and placebo.[37] Indications may include:

- Continued or severe pain despite aggressive opioid analgesia
- Autonomic instability (profound tachycardia and hypertension)
- Continued nausea or vomiting
- Shortness of breath
- Rapidly progressive symptoms.

The decision to use antivenom must include weighing the severity of the symptoms against the safety of antivenom treatment. Although rare, reactions can include serum sickness and life-threatening anaphylaxis. In countries other than the US, antivenom is used more often, and it seems to have a good safety profile.[20] [38]

Recluse spider bites

Most *Loxosceles* species
[Fig-10]

[Fig-9]

bites can be managed with local wound care, including washing with soap and water, elevation, cool compresses, and attention to tetanus status. Pain can be controlled with paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs), or oral opioids. Antibiotics are not indicated initially in confirmed bites (although frequently the diagnosis is uncertain and cellulitis is the first differential considered).

Despite their reputation, only a small number of recluse spider bites progress to become necrotic.[21]
[Fig-14]

[Fig-15]

Dapsone treatment has been used to prevent or slow the development of necrosis and reduce pain in necrotic lesions. No controlled trials have been conducted in humans, and data in animal models have been contradictory.[19] Patients should also be screened for glucose-6-phosphate dehydrogenase deficiency, as dapsone can cause severe haemolytic anaemia in these patients. It is not necessary to start dapsone treatment immediately for it to be beneficial, and screening results are usually available within 1 day.

Continued necrosis may need surgical debridement and subsequent skin grafting for full healing, although this is a rare occurrence. Necrotic tissue presents a prime substrate for secondary infection. Patients should be taught appropriate wound care and to be aware of signs of infection (e.g., fever, pus formation). Antibiotic treatment is often started empirically because the diagnosis of spider bite is often unclear, and

infections are the other major consideration in the differential diagnosis list. Antibiotic coverage should be appropriate for cellulitis in line with local susceptibility patterns for community-acquired MRSA.

Antivenoms are available in some South American countries. Some animal studies suggest efficacy at limiting necrosis, but no good human studies are available.[35]

Systemic reactions

Cardiopulmonary assessment and supportive measures

- Airway patency must be maintained, as airways can close within minutes when surrounding tissues swell.
- Prophylactic intubation is much preferred to rescue crichothyrotomy.
- Any subjective findings (e.g., swelling or tightness in the throat or oropharynx) or objective findings (e.g., stridor, hoarseness, visualised glottic or tongue oedema, cyanosis) warrant preparation for emergency airway management.
- Cardiovascular collapse should be treated with aggressive volume resuscitation (isotonic solutions e.g., 0.9% normal saline or Ringer's lactate) and vasopressor infusion.
- Patients requiring airway support or treatment for cardiovascular collapse must be moved to the accident and emergency department or critical care setting as rapidly as possible.
- Any retained stingers should be removed as soon as they are identified.

Adrenaline (epinephrine)

- All patients with signs of a systemic reaction, especially hypotension, airway swelling, or difficulty breathing, should receive immediate intramuscular adrenaline (epinephrine) in the anterolateral thigh.[2] [39] [40] [41] [42]
- The dose may be repeated every 5 to 15 minutes as needed.[22] [23] [43] [44]
- Administration in the anterolateral thigh is superior to intramuscular administration in the deltoid or a subcutaneous injection.[45] [46]
- If the patient has severe hypotension, intravenous adrenaline (epinephrine) is an option. Continuous infusion of adrenaline (epinephrine), titrated to effect, is reserved for experienced practitioners. No intravenous dose regimen is universally recognised.
- The alpha-1, beta-1, and beta-2 agonist actions of adrenaline (epinephrine) play a key role in reversing the effects of anaphylaxis. Stimulation of the alpha-1 receptors leads to increased vascular tone and thus reversal of the effects of massive vasodilation triggered by immune mediators. However, alpha-1 stimulation can also lead to severe hypertension, especially in those with poorly controlled hypertension. Beta-1 receptor stimulation has positive inotropic and chronotropic effects (i.e., the heart rate and contractility are increased), but an overshooting response can result in unwanted tachycardia, potentially harming a patient with coronary artery disease. Beta-2 agonism causes bronchodilation and impairs release of mediators from mast cells and basophils.[47]
- A prescription for 2 adrenaline (epinephrine) auto-injectors must be given after any episode of anaphylaxis.[3] [48] The patient or caregiver should carry both at all times and be familiar with their use.[2] For children at risk of anaphylaxis, the adrenaline (epinephrine) auto-injectors should be prescribed in conjunction with a personalised, written emergency plan.[2] [49] [American Academy of Pediatrics: allergy and anaphylaxis emergency plan]

Persistent respiratory symptoms after administration of adrenaline (epinephrine) may benefit from inhaled beta-2 agonists.[50]

Glucagon

- Patients treated with beta-blockers may be refractory to treatment with alpha-/beta-agonists. Glucagon works by bypassing the adrenergic receptors and directly activating cyclic adenosine monophosphate intracellularly.
- Glucagon often causes nausea and vomiting, which may further prompt the need for definitive airway control.
- Anti-emetics may be used in conjunction with glucagon treatment.

Corticosteroids

- Corticosteroid treatment works to decrease vascular permeability and blunt the immune response to the inciting antigen.
- Effects take at least 1 hour to begin, so it is important to start treatment as early as possible.
- Corticosteroid treatment should be continued for 3 to 5 days in patients with moderate to severe reactions.
- In patients without previous corticosteroid dependency or adrenal insufficiency, there is no need to taper the dose. In patients with chronic corticosteroid use, previous corticosteroid dependency, or adrenal insufficiency, or at risk of rebound effects, carefully monitored tapering of treatment may be warranted. Dose packs or slowly decreasing doses of oral prednisolone serve this purpose. These patients must be followed closely by their physician.

H1 antagonists and H2 antagonists

- Antihistamines antagonise the effects of histamine release at cellular receptors, decreasing itching, erythema, and rash.
- Sedating H1 antagonists should be continued for about 3 days following an allergic reaction and can be tapered by the patient according to symptom severity.
- Non-sedating H1 antagonists can also be used.
- H2 antagonists can be used to further potentiate the antihistamine effect.
- Topical antihistamines should be used with caution, if at all. They probably do not add any beneficial effect when patients are already on systemic antihistamines. Their erratic absorption patterns can lead to anticholinergic toxicity. They can also be irritating to the skin and exacerbate dermatological symptoms.

Anti-inflammatory medications

- Treatment with non-steroidal anti-inflammatory drugs (NSAIDs) can decrease swelling and pain at the site of the bite or sting. These medications can be tapered by the patient according to their symptoms. Caution should be used in patients currently taking aspirin or anticoagulants, patients with sensitivity to these medications, or patients with risk factors for ulcers, GI bleeding, or thromboembolic disease.

Secondary infection

Antibiotics should be directed at common skin pathogens (staphylococcal and streptococcal species) and directed by local resistance patterns.

Black widow spider bites do not become necrotic, and antibiotics are not needed unless signs of secondary infection develop over the next few days.

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Presumptive (summary)

Patient group	Tx line	Treatment
anaphylaxis/anaphylactoid reaction	1st	cardiopulmonary assessment and supportive measures
	plus	adrenaline (epinephrine)
	plus	supportive care
	plus	corticosteroid
	plus	H1 antagonist + H2 antagonist
	adjunct	nebulised salbutamol
	adjunct	stinger removal
	adjunct	non-steroidal anti-inflammatory drugs (NSAIDs)
▪ unresponsive to adrenaline (epinephrine)	plus	glucagon
▪ unresponsive to adrenaline (epinephrine)	adjunct	anti-emetic

Acute (summary)

Patient group	Tx line	Treatment
local reaction	1st	supportive care
	adjunct	stinger removal
	adjunct	corticosteroid
	adjunct	H1 antagonist + H2 antagonist
	adjunct	non-steroidal anti-inflammatory drugs (NSAIDs)
▪ black widow spider bites	adjunct	additional analgesia
▪ black widow spider bites	adjunct	benzodiazepine
▪ black widow spider bites	adjunct	beta-blocker
▪ black widow spider bites	adjunct	antivenom
▪ recluse spider bites	adjunct	additional analgesia

Acute (summary)

recluse spider bites	adjunct	dapsone
recluse spider bites	adjunct	surgical debridement + empirical antibiotics

Ongoing (summary)

Patient group	Tx line	Treatment
secondary infection	1st	antibiotics

Treatment options

Presumptive

Patient group	Tx line	Treatment
anaphylaxis/anaphylactoid reaction	**1st**	**cardiopulmonary assessment and supportive measures** » Airway patency must be maintained. Airways can close within minutes when surrounding tissues swell. Prophylactic intubation is much superior to rescue cricothyrotomy. Any subjective findings (swelling or tightness in the throat or oropharynx) or objective findings (stridor, hoarseness, visualised glottic or tongue oedema, cyanosis) warrant preparation for emergency airway management. » Cardiovascular collapse should be treated with aggressive volume resuscitation (isotonic solutions such as 0.9% normal saline or Ringer's lactate) and vasopressor infusion. This necessitates immediate transfer to an accident and emergency (A&E) department or critical care setting. » Patients requiring airway support or treatment for cardiovascular collapse must be moved to the A&E or critical care setting as rapidly as possible.
	plus	**adrenaline (epinephrine)** » All patients with signs of a systemic reaction, especially hypotension, airway swelling, or difficulty breathing, should receive immediate intramuscular adrenaline (epinephrine) in the anterolateral thigh.[2] [39] [40] [41] [42] » The dose may be repeated every 5 to 15 minutes as needed.[22] [23] [43] [44] Administration in the anterolateral thigh is superior to intramuscular administration in the deltoid or a subcutaneous injection.[45] [46] » If the patient has severe hypotension, intravenous adrenaline (epinephrine) is an option. Continuous infusion of adrenaline (epinephrine), titrated to effect, is reserved for experienced practitioners. No intravenous dose regimen is universally recognised. » A prescription for 2 adrenaline (epinephrine) auto-injectors must be given after any episode of anaphylaxis.[3] [48] The patient or caregiver should carry both at all times and be familiar with

Presumptive

Patient group	Tx line	Treatment
		their use.[2] For children at risk of anaphylaxis, the adrenaline (epinephrine) auto-injectors should be prescribed in conjunction with a personalised, written emergency plan.[2] [49] [American Academy of Pediatrics: allergy and anaphylaxis emergency plan]
		Primary options
		» adrenaline (epinephrine): 0.3 to 0.5 mg (1:1000 solution) intramuscularly every 5-15 minutes; 0.1 mg (1:10,000 solution) intravenously every 5 minutes; 1-4 micrograms/minute (1:10,000 solution) intravenous infusion
	plus	**supportive care**
		» Cardiovascular collapse should be treated with aggressive volume resuscitation (isotonic solutions e.g., 0.9% normal saline or Ringer's lactate) and vasopressor infusion. This necessitates immediate transfer to an accident and emergency department or critical care setting.
	plus	**corticosteroid**
		» Corticosteroid treatment decreases vascular permeability and blunts the immune response to the inciting antigen. Effects take at least 1 hour to begin, so treatment should be started as early as possible.
		» Corticosteroid treatment should be continued for 3 to 5 days. In patients without previous corticosteroid dependency or adrenal insufficiency, there is no need to taper the dose.
		» Carefully monitored tapering of treatment may be warranted in patients with chronic corticosteroid use, previous corticosteroid dependency, or adrenal insufficiency, or at risk of rebound effects. Dose packs or slowly decreasing doses of oral prednisolone serve this purpose.
		» The data supporting the use of corticosteroids are limited due to difficulties in performing controlled studies. Nonetheless, corticosteroid treatment remains a current mainstay of therapy.[34] [51]
		» These patients require close monitoring by their physician.
		Primary options

Presumptive

Patient group	Tx line	Treatment
		» methylprednisolone sodium succinate: 125 mg intravenously every 6 hours
		OR
		Primary options
		» prednisolone: 60 mg orally once daily
	plus	**H1 antagonist + H2 antagonist**
		» H1 antagonists antagonise the effects of histamine release at cellular receptors, decreasing itching, erythema, and rash. Sedating or non-sedating H1 antagonists may be used. These medications should be continued for about 3 days following an allergic reaction and can be tapered by the patient according to symptom severity.
		» H2 antagonists can be used to further potentiate the antihistamine effect. Cimetidine is the prototypical drug, but as it can influence the metabolism of other medications, ranitidine is a more common choice.
		Primary options
		» diphenhydramine: 50 mg orally/ intravenously every 6 hours **-or-** » loratadine: 10 mg orally once daily **-or-** » cetirizine: 10 mg orally once daily
		--AND--
		» cimetidine: 300 mg intravenously every 6 hours; 800-1600 mg/day orally given in 4 divided doses **-or-** » ranitidine: 50 mg intravenously every 8 hours; 150-300 mg orally once daily
	adjunct	**nebulised salbutamol**
		» Persistent respiratory symptoms after administration of adrenaline (epinephrine) may benefit from inhaled beta-2 agonists.[50]
		Primary options
		» salbutamol inhaled: 2.5 to 5 mg nebulised every 20 minutes for 3 doses, followed by 2.5 to 10 mg nebulised every 1-4 hours when required
	adjunct	**stinger removal**
		» Retained stingers should be removed because they may still contain venom. Traditional teaching

Presumptive

Patient group	Tx line	Treatment
		suggests that squeezing the stinger (e.g., tweezers) can inject more venom into the patient. The stinger can be removed by gently scraping the stinger with the edge of a plastic ID card (driver's licence or similar object). More recent research has called this into question and suggests that time to removal is more important than method in minimising the amount of venom injected.[33]
	adjunct	**non-steroidal anti-inflammatory drugs (NSAIDs)** » Treatment with ibuprofen or naproxen can decrease swelling and pain at the site of the bite or sting. » These medications can be tapered by the patient according to their symptoms. » Caution should be used in patients treated with aspirin or anticoagulants, or with sensitivity to these medications, or with risk factors for ulcers, GI bleeding, or thromboembolic disease. **Primary options** » ibuprofen: 300-400 mg orally every 6-8 hours when required, maximum 2400 mg/day **OR** **Secondary options** » naproxen: 250-500 mg orally twice daily when required, maximum 1250 mg/day
■ **unresponsive to adrenaline (epinephrine)**	**plus**	**glucagon** » Patients treated with beta-blockers may be refractory to treatment with beta-agonists. » Glucagon works by bypassing the adrenergic receptors and directly activating cyclic adenosine monophosphate intracellularly. » Give to patients not responding to beta-agonist treatment. » Often causes nausea and vomiting, which may further prompt the need for definitive airway control. **Primary options** » glucagon: 1-2 mg intravenously over 5 minutes initially, may be repeated according to response, followed by 0.005 to 0.05 mg/

Presumptive

Patient group	Tx line	Treatment
		min infusion; consult specialist for further guidance on higher doses
unresponsive to adrenaline (epinephrine)	adjunct	**anti-emetic** » Glucagon often causes nausea and vomiting, which may further prompt the need for definitive airway control. » Anti-emetics may be used in conjunction with glucagon treatment. » Metoclopramide should only be used for up to 5 days in order to minimise the risk of neurological and other adverse effects.[52] **Primary options** » metoclopramide: 10 mg intravenously every 8 hours when required for a maximum of 5 days, maximum 30 mg/day **OR** **Primary options** » ondansetron: 8 mg intravenously every 8 hours when required

Acute

Patient group	Tx line	Treatment
local reaction	**1st**	**supportive care** » Local pain and swelling at the site of the bite or sting can be reduced with ice pack application. The pack should have a cloth barrier between the ice and skin to prevent local tissue damage. Applying the ice pack on and off at 15-minute intervals is a common regimen. » The wound should be cleaned with soap and water, and tetanus status should be addressed.
	adjunct	**stinger removal** » Retained stingers should be removed, as they may still contain venom. Traditional teaching suggests that squeezing the stinger (e.g., tweezers) can inject more venom into the patient. The stinger can be removed by gently scraping the stinger with the edge of a plastic ID card (driver's licence or similar object). More recent research has called this into question and

Acute

Patient group	Tx line	Treatment
		suggests that time to removal is more important than method in minimising the amount of venom injected.[33]
	adjunct	**corticosteroid**
		» Corticosteroids decrease vascular permeability and blunt the immune response to the inciting antigen. Effects take at least 1 hour, so treatment should be started as early as possible.
		» Corticosteroid treatment should be continued for 3 to 5 days in patients with moderate to severe reactions. In patients without previous corticosteroid dependency or adrenal insufficiency, there is no need to taper the dose.
		» Carefully monitored tapering of treatment may be warranted in patients with chronic corticosteroid use, previous corticosteroid dependency, or adrenal insufficiency, or at risk of rebound effects. Dose packs or slowly decreasing doses of oral prednisolone serve this purpose.
		» The data supporting the use of corticosteroids are limited due to difficulties in performing controlled studies. Nonetheless, corticosteroid treatment remains a current mainstay of therapy.[34] [51]
		» These patients require close monitoring by their physician.
		Primary options
		» methylprednisolone sodium succinate: 125 mg intravenously every 6 hours
		OR
		Primary options
		» prednisolone: 60 mg/day orally
	adjunct	**H1 antagonist + H2 antagonist**
		» H1 antagonists antagonise the effects of histamine release at cellular receptors, decreasing itching, erythema, and rash. Sedating or non-sedating H1 antagonists may be used. These medications should be continued for about 3 days following an allergic reaction and can be tapered by the patient according to symptom severity.
		» H2 antagonists can be used to further potentiate the antihistamine effect. Cimetidine is

Acute

Patient group	Tx line	Treatment
		the prototypical drug, but as it can influence the metabolism of other medications, ranitidine is a more common choice. **Primary options** » diphenhydramine: 50 mg orally/ intravenously every 6 hours **-or-** » loratadine: 10 mg orally once daily **-or-** » cetirizine: 10 mg orally once daily **--AND--** » cimetidine: 300 mg intravenously every 6 hours; 800-1600 mg/day orally given in 4 divided doses **-or-** » ranitidine: 50 mg intravenously every 8 hours; 150-300 mg orally once daily
	adjunct	**non-steroidal anti-inflammatory drugs (NSAIDs)** » Treatment with ibuprofen or naproxen can decrease swelling and pain at the site of the bite or sting. » These medications can be tapered by the patient according to their symptoms. » Caution should be used in patients taking aspirin or anticoagulants, or with sensitivity to these medications, or with risk factors for ulcers, GI bleeding, or thromboembolic disease. **Primary options** » ibuprofen: 300-400 mg orally every 6-8 hours when required, maximum 2400 mg/day **OR** **Secondary options** » naproxen: 250-500 mg orally twice daily when required, maximum 1250 mg/day
■ black widow spider bites	adjunct	**additional analgesia** » Mild to moderate pain often responds to paracetamol or non-steroidal anti-inflammatory drug (NSAID) treatment. » Oral opioids may be needed for more severe bites or patients with low pain tolerance. » More severe bites can cause severe pain in the affected limb or in a generalised fashion. Pain

Acute

Patient group	Tx line	Treatment
		radiating into the chest or abdomen can confuse the diagnostic picture, and myocardial ischaemia or an acute abdomen may need to be ruled out. Patients may need high-dose intravenous opioids for pain control. **Primary options** » paracetamol: 500-1000 mg orally/rectally every 4-6 hours when required, maximum 4000 mg/day **OR** **Secondary options** » paracetamol/hydrocodone: 5-10 mg orally every 6-8 hours when required Dose refers to hydrocodone component. Maximum 4000 mg/day of paracetamol component. **-or-** » oxycodone: 5-10 mg orally every 8 hours when required **OR** **Tertiary options** » morphine sulfate: 10-30 mg orally (immediate-release) every 3-4 hours when required; 2.5 to 10 mg intravenously every 2-6 hours when required
black widow spider bites	**adjunct**	**benzodiazepine** » More severe bites can cause severe pain and muscle spasms in the affected limb or in a generalised fashion. Severe muscle spasms may be relieved with benzodiazepines. **Primary options** » diazepam: 2-10 mg orally twice to four times daily
black widow spider bites	**adjunct**	**beta-blocker** » Tachycardia and hypertension may occur but usually resolve as pain is addressed. In patients who may not tolerate these effects, beta-blockers may be a reasonable treatment. » These spikes in BP and heart rate tend to be transient. They often resolve spontaneously or after treatment of the pain or anxiety associated with the bite or sting.

Acute

Patient group	Tx line	Treatment
		» Intravenous medications allow for tighter control and decrease the chance of inadvertently exceeding targets with longer-acting medications. Accidentally exceeding targets can cause BP or heart rate to fall to dangerously low levels. **Primary options** » labetalol: 20 mg intravenous bolus initially, followed by 40-80 mg every 10 minutes according to response, maximum 300 mg total dose
black widow spider bites	**adjunct**	**antivenom** » There are several black widow spider antivenoms on the market.[35] [36] Although fatalities from bites are exceedingly rare, treatment with antivenom may reduce the pain and duration of symptoms slightly. Indications may include continued or severe pain despite aggressive opioid analgesia, autonomic instability (profound tachycardia and hypertension), continued nausea or vomiting, SOB, and rapidly progressive symptoms. The modest benefits of treatment must, however, be weighed against the safety of antivenom treatment.[37] Although rare, reactions include serum sickness and life-threatening anaphylaxis. » In countries other than the US, antivenom is used more often and seems to have a good safety profile.[20] [38] » Some large hospitals will stock the antivenom, but it usually needs to be obtained from a zoo or poison centre.
recluse spider bites	**adjunct**	**additional analgesia** » Pain can be controlled with paracetamol or an opioid. **Primary options** » paracetamol: 500-1000 mg orally/rectally every 4-6 hours when required, maximum 4000 mg/day **OR** **Secondary options** » paracetamol/hydrocodone: 5-10 mg orally every 6-8 hours when required

TREATMENT

Acute

Patient group	Tx line	Treatment
		Dose refers to hydrocodone component. Maximum 4000 mg/day of paracetamol component. **-or-** » oxycodone: 5-10 mg orally every 8 hours when required **OR** **Tertiary options** » morphine sulfate: 10-30 mg orally (immediate-release) every 3-4 hours when required; 2.5 to 10 mg intravenously every 2-6 hours when required
recluse spider bites	**adjunct**	**dapsone** » Despite their reputation, only a small proportion of recluse spider bites become necrotic.[21] [Fig-14] [Fig-15] » Dapsone treatment has been used to prevent or slow the development of necrosis, and has effects on reducing pain in necrotic lesions. No controlled trials have been conducted in humans. Data in animal models have been contradictory. » Patients should be screened for glucose-6-phosphate dehydrogenase deficiency. Dapsone can cause a severe haemolytic anaemia in these patients. Screening is usually available within 1 day, and it is not necessary to start dapsone treatment immediately for it to be beneficial. **Primary options** » dapsone: 50 mg orally twice daily for 10 days
recluse spider bites	**adjunct**	**surgical debridement + empirical antibiotics** » Continued necrosis may need surgical debridement and subsequent skin grafting for full healing, although this is a rare occurrence. Necrotic tissue presents a prime substrate for secondary infection. Patients should be taught appropriate wound care and to be aware of signs of infection (e.g., fever, pus formation). » Antibiotic treatment is often begun empirically, because the diagnosis of spider bite is often not clear, and infections are the top other consideration on the differential diagnosis list.

Acute

Patient group	Tx line	Treatment
		Antibiotic coverage should be appropriate for cellulitis in line with local susceptibility patterns for community-acquired MRSA.

Ongoing

Patient group	Tx line	Treatment
secondary infection	**1st**	**antibiotics** » Antibiotics should be directed at common skin pathogens (staphylococcal and streptococcal species) and directed by local resistance patterns. » Black widow spider bites do not become necrotic; antibiotics are not needed unless signs of secondary infection develop over the next few days.

Emerging

Recluse spider antivenom

Antivenoms for recluse spider bites are available in some South American countries. Some animal studies suggest efficacy at limiting necrosis, but there haven't been any good clinical studies.[53]

FOLLOW UP

Recommendations

Monitoring

Because of the possibility of a biphasic anaphylactic reaction after resolution of the initial presentation, monitoring as an inpatient is indicated. Recommended durations of monitoring vary from a minimum of 4 to 6 hours in patients who have experienced anaphylaxis,[57] to 24 hours or more in severe cases.[58] Differing observation periods, or the effect of these on relevant patient outcomes, have not been studied.[59]

Patients who respond well to treatment and whose symptoms do not return within the observation period can be discharged home with close follow-up and instructions to return if symptoms reappear. The patient's ability to recognise symptoms and to self-administer adrenaline (epinephrine) should inform this decision.[60] Patients should notify their primary care physician about their condition and obtain prompt follow-up with an allergist.

Patient instructions

Patients who suffer severe reactions to insect bites or stings must be educated that they are at high risk of similar (or worse) reactions in the future. Patients who have had respiratory symptoms or anaphylactic reactions should be discharged with a prescription for two adrenaline (epinephrine) auto-injectors (e.g., EpiPen, Twinject) and be instructed in their proper use.[3] [48] For children at risk of anaphylaxis, the adrenaline (epinephrine) auto-injectors should be prescribed in conjunction with a personalised written emergency plan. [American Academy of Pediatrics: allergy and anaphylaxis emergency plan] [2] [49]

Because auto-injectors need to be available quickly, patients should carry both with them at all times.[3] [48] This allows for extra doses to be available in case of rebound or biphasic reactions.[31] [61] [62] Patients need to be advised that their use is only intended as an adjunct (not a substitute) for prompt medical care. EpiPen instructions caution about exposing auto-injectors to extremes of temperature and specifically recommends against storing in the glove compartment of a car.

Patients should also be educated about the possibility of rebound reactions as their treatment medications wear off. They should be told to return if they experience any breathing difficulties (e.g., wheezing, SOB) or swelling/tingling in their mouth or throat. Patients with minor dermatological reactions should also be instructed about the possibility of more severe reactions in the future. They can be taught to self-treat with antihistamines and anti-inflammatory medications if symptoms are limited to dermatological reactions, but they should return if any respiratory or oral symptoms are experienced.

Patients should be instructed to continue taking medications as prescribed by their doctor, which may include corticosteroids (e.g., prednisolone) or antihistamines (e.g., diphenhydramine). They should also be instructed to inform their regular doctor about the event and any regular medication prescribed. Patient information on insect bites and stings [NHS choices: insect bites and stings] is available.

Complications

Complications	Timeframe	Likelihood
delayed anaphylactic or anaphylactoid reactions	**short term**	**low**
Anaphylactic or anaphylactoid reactions most often occur within minutes of the initial exposure, but can be delayed or rebound as initial treatments wear off. Any symptoms involving SOB, swelling or tingling in the mouth or throat, or generalised weakness should prompt immediate medical care.		

Complications	Timeframe	Likelihood
secondary infection	**long term**	**low**
Cellulitis or abscess development may occur in the days following a bite or sting. These occur more commonly in older or immunocompromised patients. Although rare, necrotic lesions from recluse spider bites are a prime substrate for secondary infection.		

Prognosis

The prognosis is excellent for patients who have noxious insect encounters and experience only local effects (pain, itching). Frequently, educating patients about things they can do to treat the exposure at home will prevent unnecessary trips to the accident and emergency department or doctor's surgery.

Severe reactions, such as anaphylaxis, have a good prognosis if recognised and treated expeditiously. Prompt airway and cardiovascular support will prevent most adverse outcomes from anaphylactic reactions. It is extremely important to educate victims of severe reactions that they are at high risk of another severe event. Education, provision of two adrenaline (epinephrine) auto-injectors,[3] [48] and referral for possible desensitisation therapy are very important for these patients.

The long-term prognosis for black widow bites is very good. Outcome from brown recluse envenomation is variable, with some risk of visible scarring or disfigurement.

All patients discharged from the accident and emergency department should be warned of late-phase (delayed) anaphylactic reactions. Some physicians feel that all patients with anaphylaxis should be hospitalised for 24 hours because late-phase reactions are not uncommon.

Patients with mild to moderate reactions should be followed up by their primary doctor to monitor resolution of symptoms. Continued or worsening redness, pain, or fever may indicate secondary infection. Spider bites and cellulitis are often clinically indistinguishable. In the absence of a witnessed bite, antibiotics are often started empirically. If antibiotics are used, coverage should be directed at common skin pathogens (*Staphylococcus* , *Streptococcus*) and community-acquired MRSA (according to local susceptibilities).

Patients with a severe allergic reaction should be evaluated by their primary doctor regarding referral to an allergist/immunologist for further testing.[28] [54] [55] [56] It may involve skin testing, in vitro testing, and possibly desensitisation therapy. This is covered in detail in guidelines from AAAAI/ACAAI (American Academy of Allergy, Asthma & Immunology; American College of Allergy, Asthma & Immunology).[1]

Diagnostic guidelines

North America

Stinging insect hypersensitivity: a practice parameter update 2016

Published by: American Academy of Allergy, Asthma & Immunology; American College of Allergy, Asthma & Immunology **Last published:** 2016

Second symposium on the definition and management of anaphylaxis: summary report - second National Institute of Allergy and Infectious Disease/ Food Allergy and Anaphylaxis Network symposium

Published by: National Institute of Allergy and Infectious Disease; Food Allergy and Anaphylaxis Network **Last published:** 2006

Treatment guidelines

Europe

Anaphylaxis: assessment and referral after emergency treatment

Published by: National Institute for Health and Care Excellence **Last published:** 2011

Guideline for the management of acute allergic reaction

Published by: College of Emergency Medicine **Last published:** 2009

North America

Epinephrine for first aid management of anaphylaxis

Published by: American Academy of Pediatrics **Last published:** 2017

Stinging insect hypersensitivity: a practice parameter update 2016

Published by: American Academy of Allergy, Asthma & Immunology; American College of Allergy, Asthma & Immunology **Last published:** 2016

Allergen immunotherapy: a practice parameter third update

Published by: American Academy of Allergy, Asthma & Immunology; American College of Allergy, Asthma & Immunology; Joint Council of Allergy, Asthma & Immunology **Last published:** 2011

Second symposium on the definition and management of anaphylaxis: summary report - second National Institute of Allergy and Infectious Disease/ Food Allergy and Anaphylaxis Network symposium

Published by: National Institute of Allergy and Infectious Diseases; Food Allergy and Anaphylaxis Network **Last published:** 2006

Oceania

Envenomation from tick bites and bee, wasp and ant stings

Published by: Australian Resuscitation Council; New Zealand Resuscitation Council **Last published:** 2016

ASCIA guidelines for prevention of anaphylaxis in schools, pre-schools and childcare: 2015 update

Published by: Australasian Society of Clinical Immunology and Allergy **Last published:** 2015

Online resources

1. CDC: insects and scorpions *(external link)*
2. American Academy of Pediatrics: allergy and anaphylaxis emergency plan *(external link)*
3. NHS choices: insect bites and stings *(external link)*

Key articles

- Golden DB, Demain J, Freeman T, et al. Stinging insect hypersensitivity: a practice parameter update 2016. Ann Allergy Asthma Immunol. 2017 Jan;118(1):28-54. Abstract
- Vetter RS, Bush SP. The diagnosis of brown recluse spider bite is overused for dermonecrotic wounds of uncertain etiology. Ann Emerg Med. 2002 May;39(5):544-6. Full text Abstract
- Dominguez TJ. It's not a spider bite, it's community-acquired methicillin-resistant Staphylococcus aureus. J Am Board Fam Pract. 2004 May-Jun;17(3):220-6. Full text Abstract
- Golden DB. Insect sting anaphylaxis. Immunol Allergy Clin North Am. 2007 May;27(2):261-72 Full text Abstract
- Freeman TM. Hypersensitivity to hymenoptera stings. N Engl J Med. 2004 Nov 4;351(19):1978-84. Abstract
- Vetter RS, Isbister GK. Medical aspects of spider bites. Ann Rev Entomol. 2008;53:409-29. Abstract
- Soar J, Pumphrey R, Cant A, et al; Working Group of the Resuscitation Council (UK). Emergency treatment of anaphylactic reactions - guidelines for healthcare providers. Resuscitation. 2008 May;77(2):157-69. Abstract
- Golden DB, Demain J, Freeman T, et al. Stinging insect hypersensitivity: a practice parameter update 2016. Ann Allergy Asthma Immunol. 2017;118:28-54. Abstract
- Doshi D, Foex B, Body R, et al; College of Emergency Medicine. Guideline for the management of acute allergic reaction. December 2009 [internet publication]. Full text
- Clark S, Camargo CA Jr. Emergency treatment and prevention of insect sting anaphylaxis. Curr Opin Allergy Clin Immunol. 2006 Aug;6(4):279-83. Abstract

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Images

Figure 1: Honeybee stinging flesh

Courtesy of Rick Vetter

Figure 2: Close-up of honeybee stinger

Courtesy of Rick Vetter

Figure 3: Black widow (Latrodectus species); the red hourglass marking is not always this shape and may not be present

Courtesy of Rick Vetter

Figure 4: Brown recluse spider (Loxosceles species); note the violin shape, darker colouration on cephalothorax, and 3 pairs of eyes at the base of the violin

Courtesy of Rick Vetter

Figure 5: Yellow jacket

Courtesy of Rick Vetter

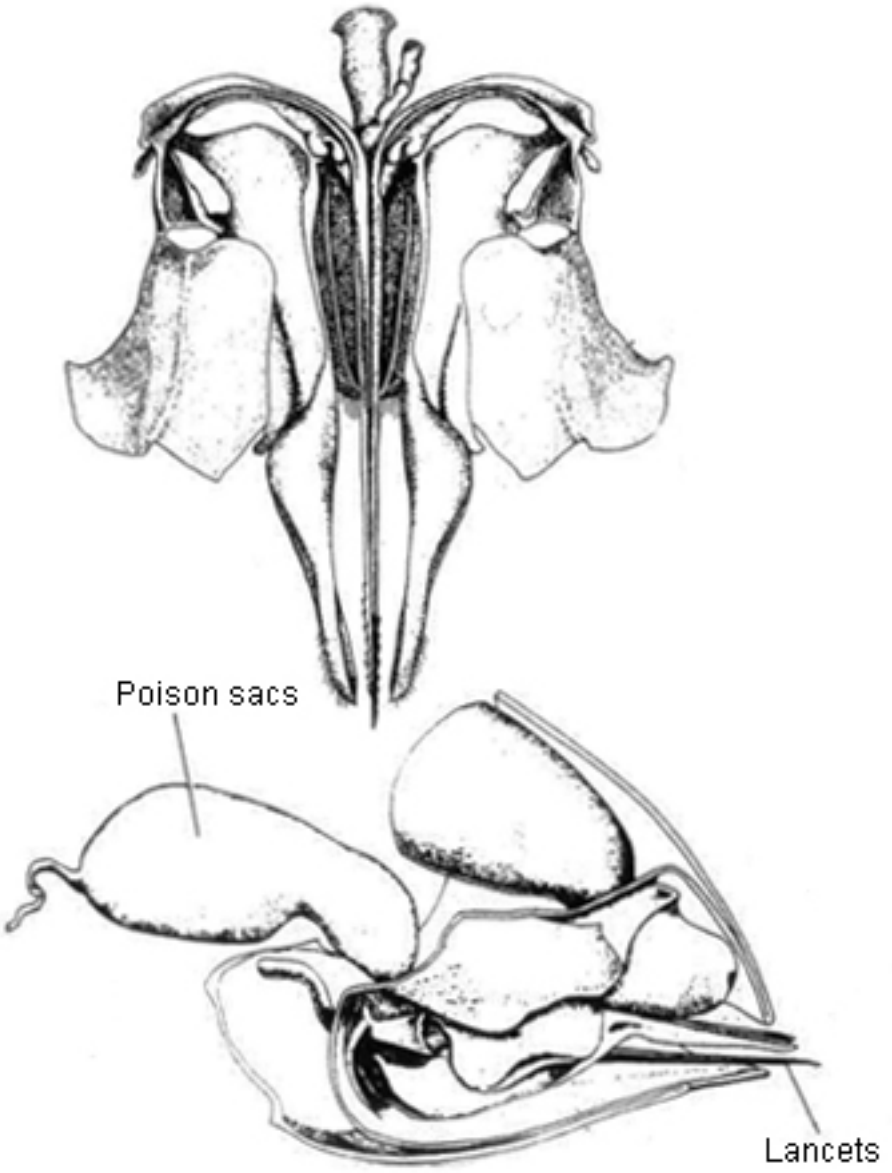

Figure 6: Honeybee stinger anatomy

Courtesy of Rick Vetter

Figure 7: Black widow (Latrodectus species)

Courtesy of Bill Banner

Figure 8: Black widow (Latrodectus species)

Courtesy of Bill Banner

Figure 9: Brown recluse spider (Loxosceles species); note violin shape, darker colouration on cephalothorax, and 3 pairs of eyes at the base of the violin

Courtesy of Rick Vetter

Figure 10: Brown recluse spider (Loxosceles species)

Courtesy of Rick Vetter

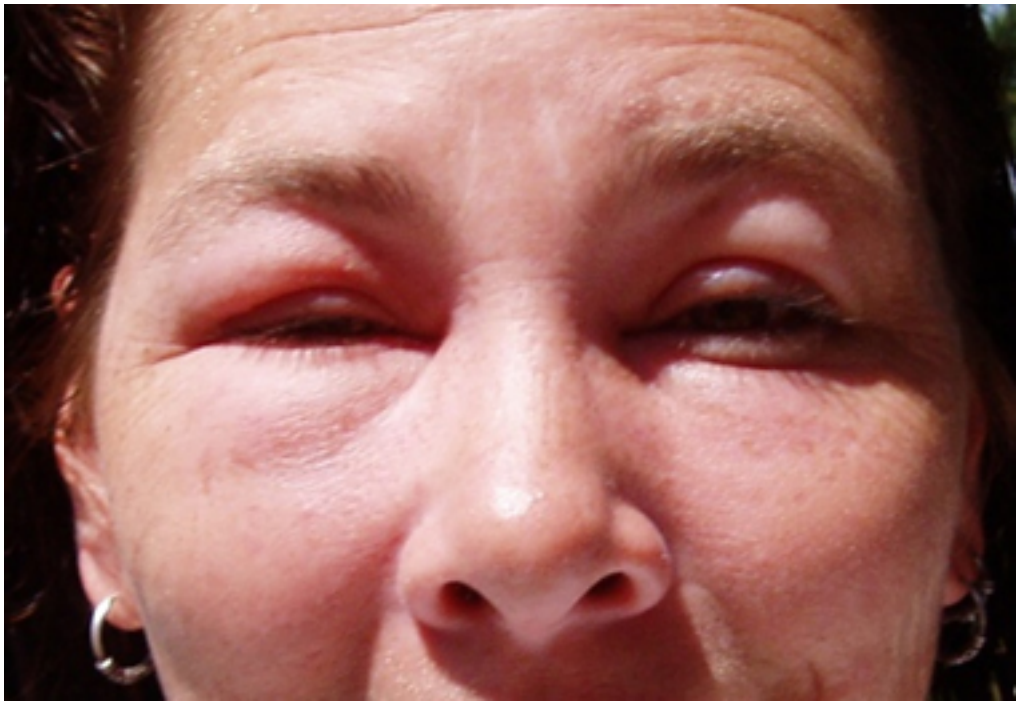

Figure 11: Periorbital swelling 24 hours after yellow jacket sting above right eye

Courtesy of Tom Morrissey

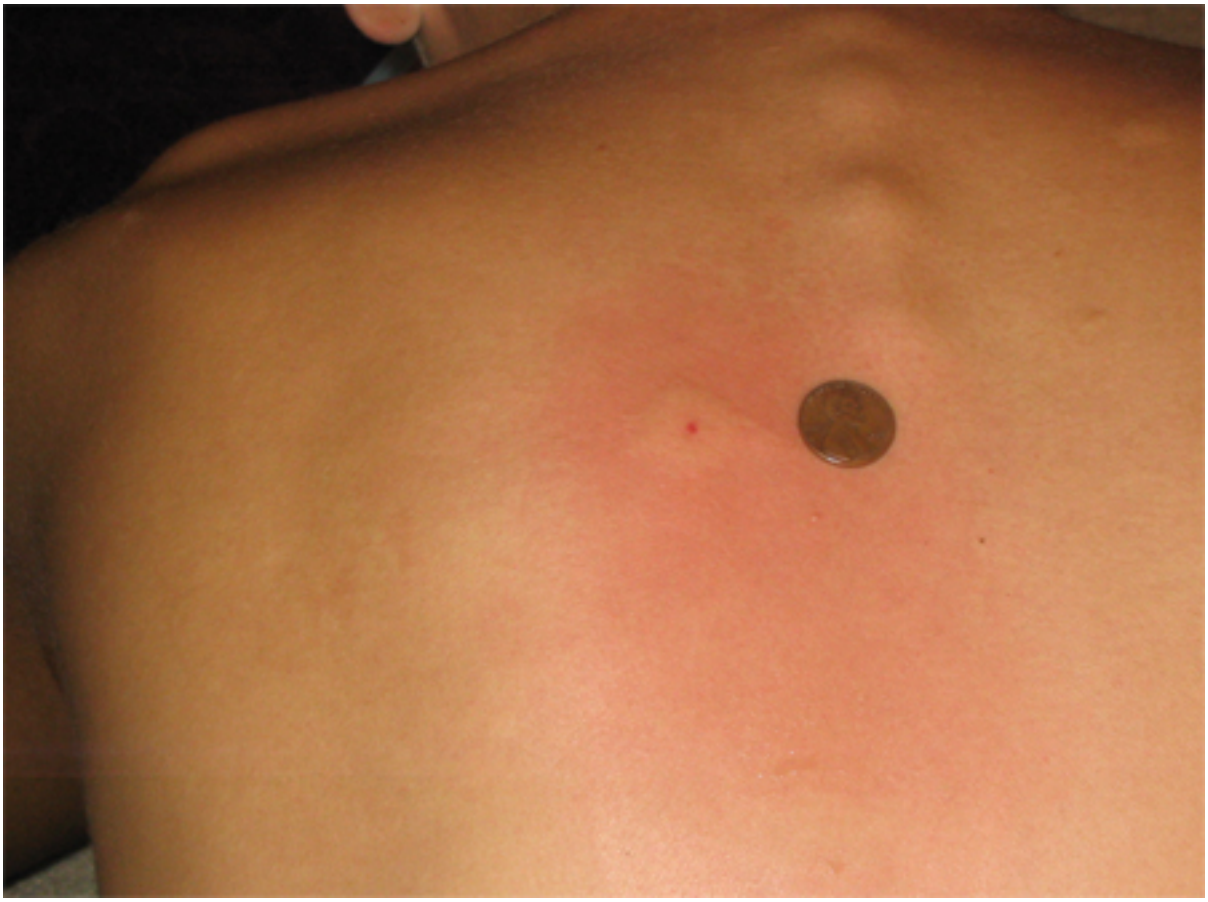

Figure 12: Wheal formation following wasp sting

Courtesy of Theodore Freeman

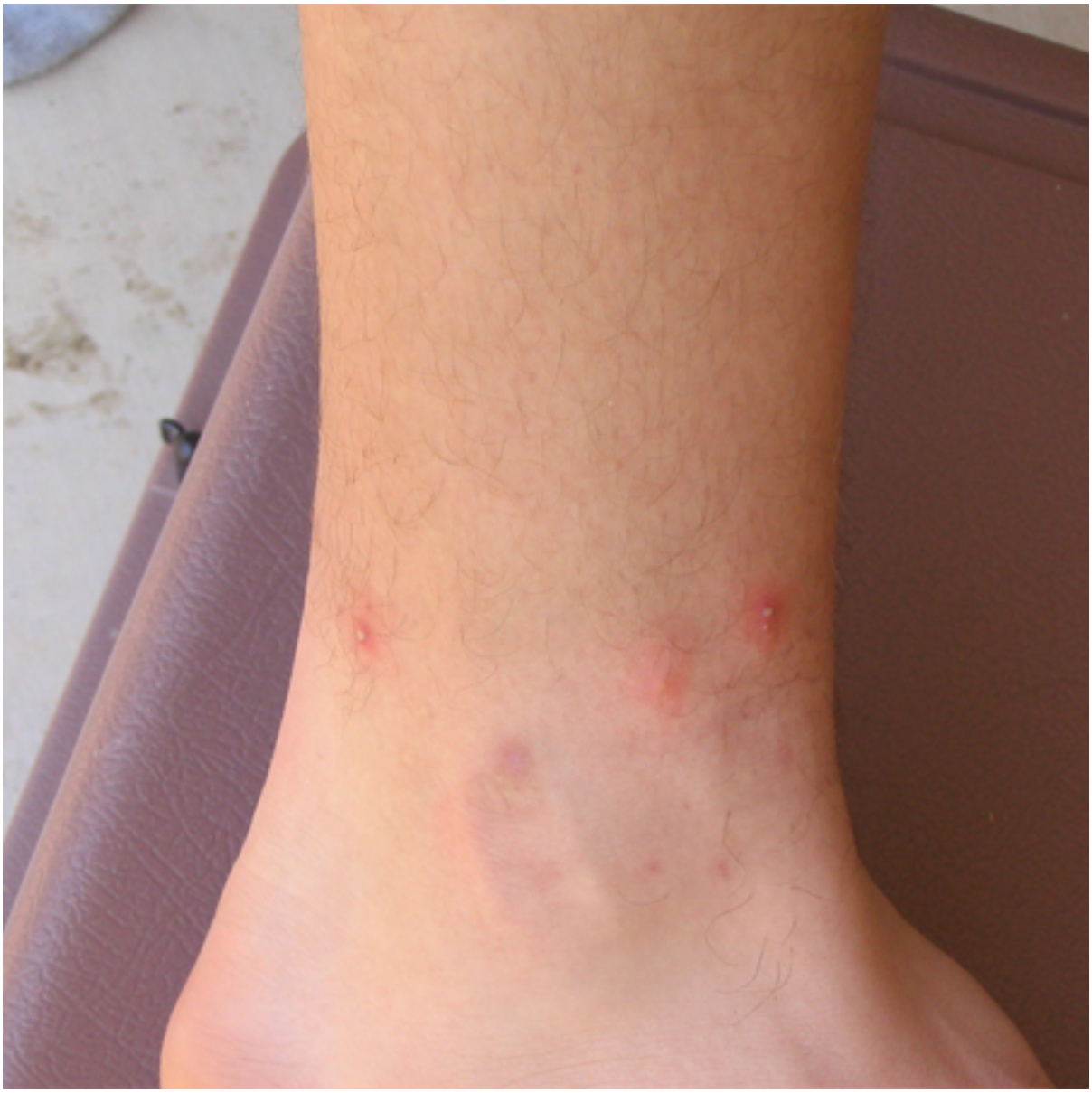

Figure 13: Pseudopustule formation following fire ant sting

Courtesy of Theodore Freeman

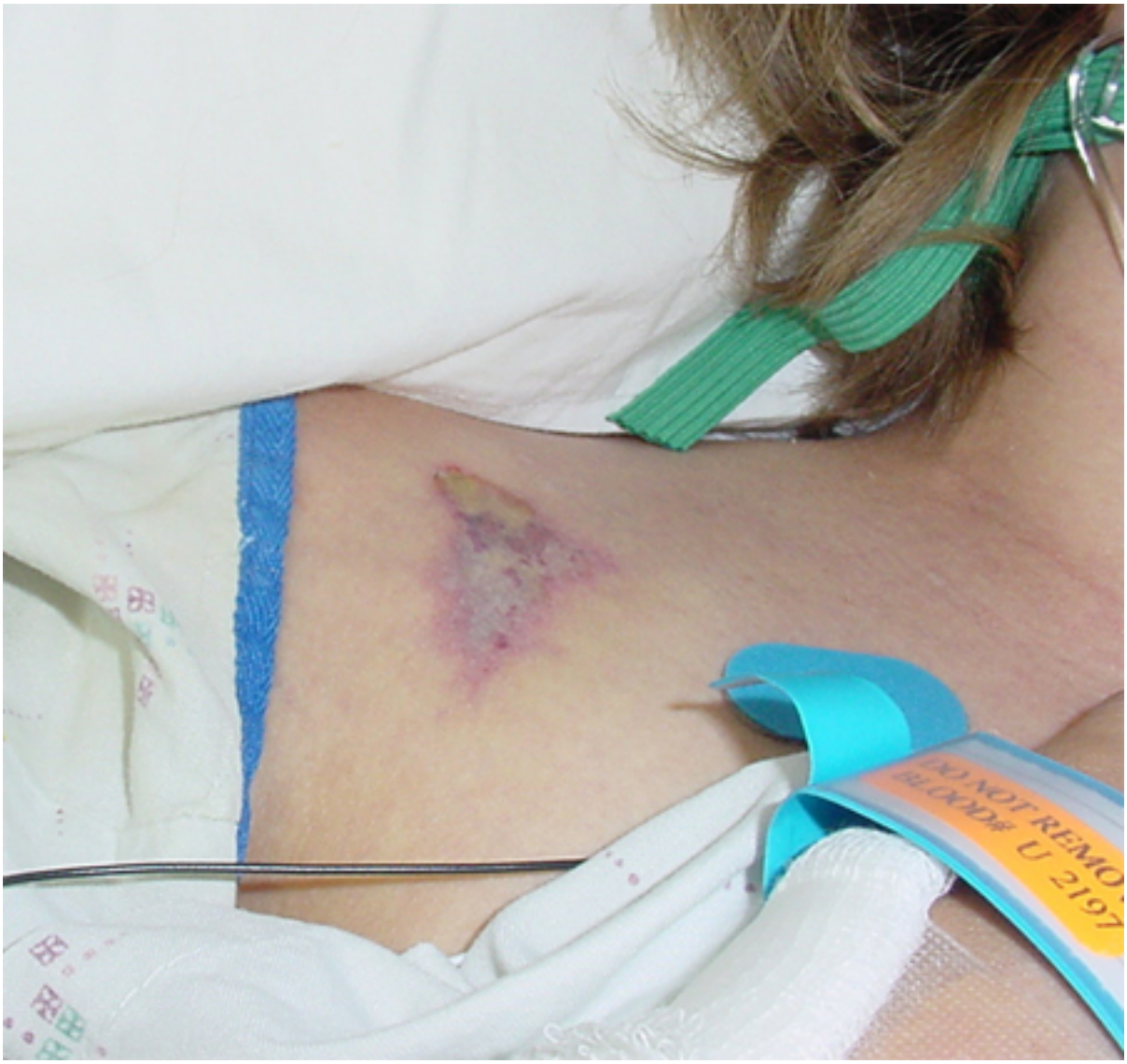

Figure 14: Lesions from reported brown recluse (Loxosceles species) envenomation

Courtesy of Theodore Freeman

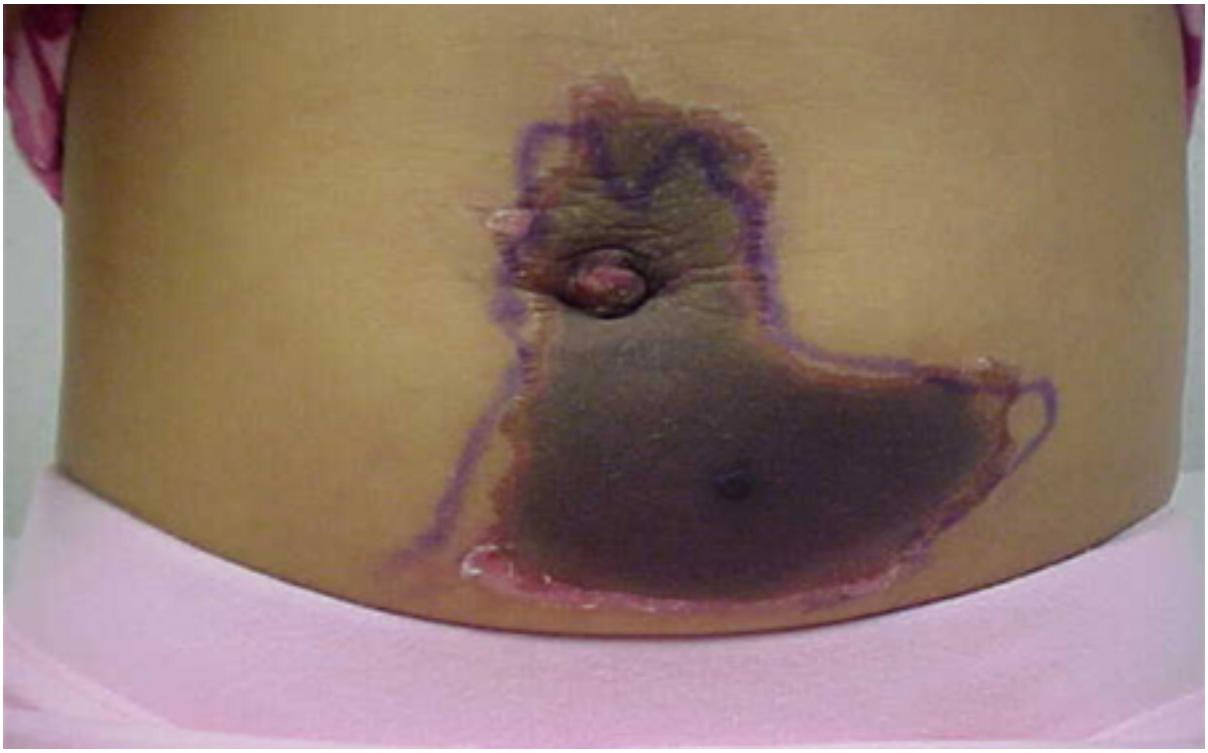

Figure 15: Lesions from reported brown recluse (Loxosceles species) envenomation

Courtesy of Theodore Freeman

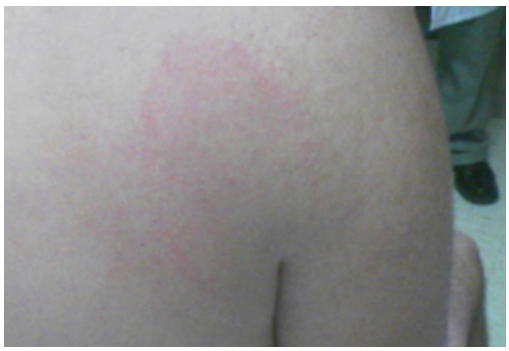

Figure 16: Erythema migrans of Lyme disease

Courtesy of Janak Koirala, MD, MPH; Tin Han Htwe, MD; and Christian Speil, MD

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