World Allergy Forum Symposium:
“Life-Threatening Allergy — An Homage to Von Pirquet”
Sunday, 11 June 2006, 13:30 - 15:15
Austria Center Vienna, Hall A
XXV Congress of the EAACI
Vienna, Austria

The World Allergy Organization (WAO) is an international organization of 74 regional and national allergy and clinical immunology societies. WAO’s mission is to be a global resource and advocate in the field of allergy, advancing excellence in clinical care, education, research and training through a world-wide alliance of allergy and clinical immunology societies.
“Life-Threatening Allergy — An Homage to Von Pirquet”

Program

Chairs:
Michael A. Kaliner, Institute for Asthma and Allergy
Wheaton, Maryland, U.S.A.

Anthony J. Frew, Brighton General Hospital
Brighton, United Kingdom

1. Welcome to the World Allergy Forum Symposium and Introduction to “Life-Threatening Allergy — An Homage to Von Pirquet”
   Michael A. Kaliner, Institute for Asthma and Allergy
   Chevy Chase, Maryland, U.S.A.

   Anthony J. Frew, Brighton General Hospital
   Brighton, United Kingdom

2. Epidemiology of Anaphylaxis
   Aziz Sheikh, The University of Edinburgh
   Edinburgh, Scotland

3. Mechanisms of Anaphylaxis
   Richard F. Lockey, University of South Florida College of Medicine
   Tampa, Florida, U.S.A.

4. Management of Anaphylaxis
   F. Estelle R. Simons, University of Manitoba
   Winnipeg, Canada

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World Allergy Organization (WAO)
The World Allergy Organization (WAO) is an international umbrella organization of 74 regional and national allergy and clinical immunology societies. By collaborating with member societies, WAO provides direct educational outreach programs, symposia and lectureships to WAO individual members around the globe.

The World Allergy Organization Mission
WAO’s mission is to be a global resource and advocate in the field of allergy, advancing excellence in clinical care, education, research and training through a world-wide alliance of allergy and clinical immunology societies.

Programs of the World Allergy Organization

GLORIA
The GLORIA program promotes good practice in the management of allergic diseases through programs developed by panels of world experts. GLORIA educates medical professionals worldwide through regional and national presentations and local training initiatives. GLORIA educational modules promote the World Allergy Organization’s (WAO) mission – to optimize allergy care worldwide.

GLORIA Modules
Module 1: Allergic Rhinitis and Allergic Conjunctivitis
Module 2: Allergic Conjunctivitis (Expanded Version)
Module 3: Allergic Emergencies
Module 4: Immunotherapy
Module 5: Symptoms and Treatment of Asthma
Module 6: Food Allergy

World Allergy Forum (WAF)
Symposia are held at major international allergy meetings. Developed by international expert advisory panels, the symposia provide up-to-the-minute presentations on scientific and clinical developments in the field of allergic disease.

PAAA: Prevention of Allergy and Allergic Asthma
Prevention of Allergy and Allergic Asthma (PAAA) is a collaborative project with the World Health Organization providing guidelines and recommendations for prevention of the allergen-specific immunological sensitization necessary for disease.

Emerging Societies Meetings
WAO offers advice on initiating and developing allergy societies throughout the world. This proactive initiative aims to expand and improve the specialty of allergy by supporting colleagues working in the field of allergy worldwide. Through sharing practical experiences and alerting new societies to the criteria required for WAO membership, ESM creates relationships with future World Allergy Organization member societies, and educates WAO’s leadership about the challenges and opportunities faced by colleagues in developing countries.

WAO Journals
ACI-International – Journal of the World Allergy Organization (ACII - JWAO) and International Archives of Allergy and Immunology
Read the latest in global allergy and asthma news and research through subscriptions to WAO’s journal partners: ACI International - Journal of the World Allergy Organization (ACII - JWAO) and International Archives of Allergy and Immunology.
WAO Member Societies*

National Member Societies

Albanian Society of Allergology and Clinical Immunology
American Academy of Allergy, Asthma and Immunology
American College of Allergy, Asthma and Immunology
Argentine Association of Allergy and Immunology
Argentine Society of Allergy and Immunopathology
Australasian Society of Clinical Immunology and Allergy
Austrian Society of Allergology and Immunology
Azerbaijan Society for Asthma, Allergy and Clinical Immunology
Bangladesh Society of Allergy and Immunology
Belgian Society of Allergology and Immunology
Brazilian Society of Allergy and Immunopathology
British Society for Allergy and Clinical Immunology
Bulgarian National Society of Allergology
Canadian Society of Allergy and Clinical Immunology
Chilean Society of Allergy and Immunology
China Allergology Society and Chinese Allergists
Chinese Hong Kong Institute of Allergy
Colombian Allergy, Asthma and Immunology Association
Croatian Society of Allergology and Clinical Immunology
Cuban Society of Allergology
Danish Society of Allergology
Ecuadorian Society of Allergy and Immunology
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Finnish Society of Allergology and Clinical Immunology
French Society of Allergology and Clinical Immunology
German Society for Allergy and Clinical Immunology
Georgian Association of Allergology and Clinical Immunology
Hellenic Society of Allergology and Clinical Immunology
Hungarian Society of Allergology and Clinical Immunology
Icelandic Society of Allergy and Clinical Immunology
Indian College of Allergy, Asthma and Applied Immunology
Indonesian Society for Allergy and Immunology
Israel Society of Allergy and Clinical Immunology
Italian Society for Allergology and Clinical Immunology
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Mexican College of Pediatricians Specialized in Allergy and Clinical Immunology
Mexican College of Allergy, Asthma and Clinical Immunology
Mongolian Society of Allergology
Netherlands Society of Allergology
Norwegian Society of Allergology and Immunopathology
Paraguayan Society of Immunology and Allergy
Peruvian Society of Allergy and Immunology
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Spanish Society of Allergology and Clinical Immunology
Swedish Association for Allergology
Swiss Society for Allergology and Immunology
Allergy and Immunology Society of Thailand
Turkish National Society of Allergy and Clinical Immunology
Ukrainian Association of Allergologists and Immunology
Uruguayan Society of Allergology
Venezuelan Society of Allergy and Immunology
Vietnam Association of Allergy, Asthma and Clinical Immunology
Zimbabwe Allergy Society

Associate Member Societies

Czech Society of Allergology and Clinical Immunology
Ecuadorian Society of Allergology and Affiliated Sciences
Egyptian Society of Pediatric Allergy and Immunology
Italian Association of Territorial and Hospital Allergists
Laotian Association of Allergists
Panamanian Association of Allergology and Clinical Immunology
Association of Allergy and Clinical Immunology of Serbia and Montenegro

Regional Organizations

The Asian Pacific Association of Allergology and Clinical Immunology
CIS Society of Immunology and Allergology
European Academy of Allergology and Clinical Immunology
Latin American Society of Allergy, Asthma and Immunology

Affiliate Organizations

International Association of Asthmology

*As of June 2006

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Web site: www.worldallergy.org
Dear Colleagues,

Welcome to the 29th Symposium in the World Allergy Forum Series, “Life-Threatening Allergy – An Homage to Von Pirquet”. Today’s program focuses on anaphylaxis and its treatment. We believe that Dr. Von Pirquet would have enjoyed hearing our three superlative speakers and the current state of anaphylaxis. World Allergy Forum is the longest running educational program of World Allergy Organization, and we are grateful for the unrestricted educational grant from Novartis, our longest running pharmaceutical partner, which enables us to bring you this program today.

I am lucky to preside over WAO at this time; the Organization has become very active in a wide range of activities, the leadership is extremely dynamic, and our position in the world of allergy is firmly established. Over the next two years, we plan a global agenda to strengthen allergy both for patients and allergists; we plan to make WAO more visible, of greater service to our member societies and establish strong and broad partnerships with our national and regional societies. Where we already have strong allergy societies, we will cooperate with them while always giving recognition to their skills and strengths. Where we have newer or less established societies, we will partner with and strengthen these fledgling groups through our Emerging Societies Program. In areas where there are no societies, WAO will help local allergists to make the governing bodies aware of the need for allergy and work to create an environment where allergy and asthma sufferers can get access to well-trained physicians.

As part of our goal to promote our specialty, in January 2006 the WAO Specialty and Training Council published the results of a survey of allergy needs and practices in member countries in our two journals, the Journal of the World Allergy Organization (JWAO), and the International Archives of Allergy and Immunology. This important document will be followed by the publication of a provisional WAO position statement entitled Requirements for Physician Training in Allergy: Key Clinical Competencies Appropriate for the Care of Patients with Allergic or Immunologic Diseases then later this year we will publish a third statement defining What is an Allergist.

We are actively planning the next World Allergy Congress, which will take place 2-6 December 2007 in Bangkok, Thailand. The meeting will have several unique features. It will begin with an international symposium on Immunotherapy cosponsored by WAO, EAACI and AAAAI. The meeting will run for 3 1/2 days thereafter, ending with an overlapping international symposium on Food Allergy, cosponsored by the ACAAI and WAO.

A global organization will only be as good as its ability to communicate. WAO is fortunate to have Richard F. Lockey as Editor-in-Chief of the WAO Web site, and Johannes Ring as Editor of JWAO. The monthly email newsletter, WAO News and Notes, is designed to keep everyone informed of clinical advances in the field and to provide a ready means of rapid communications. If you are not receiving this free of charge communication, please contact us at www.worldallergy.org and share your email information.

As we look to the future, we recognize that allergy is a rapidly developing and expanding field, but that recognition of the importance of allergy is still underappreciated, and that the time when allergy is accepted as a subspecialty of medicine and pediatrics akin to cardiology and gastroenterology is still on the horizon. WAO is committed to strengthening allergy through active educational and research partnerships with our 74 member societies. In the end, the many millions of patients with asthma and allergy will benefit as the importance of allergic diseases is recognized and taught more widely.

With my best regards,

Michael A. Kaliner
President, WAO
Dear Colleagues,

Anaphylaxis is something of a Cinderella among the various conditions that allergists see and treat. As a potentially life-threatening condition, it is clearly important and should attract our attention both for research and management. And yet it seems to have a lower place in our thoughts and congresses than other allergic conditions such as asthma and rhinitis. Today’s symposium is part of an attempt to address that gap between clinical importance and congress coverage.

We know a lot about mast cell biology and the various mediators that are released when mast cells are activated, but surprisingly little is known about why some patients develop generalized reactions after local exposure, while others just have local reactions. Sensitizing IgE antibodies are certainly necessary for most forms of anaphylaxis, but if that were all that was needed, everyone with hay fever would develop anaphylaxis during the pollen season. It seems clear that there must be other mechanisms that act as transducers, taking an initial local allergic reaction, and triggering a systemic response.

We know quite a lot about how to investigate patients who have sustained anaphylaxis but our management strategy remains based on accurate analysis of trigger factors, careful avoidance of these and provision of first aid, especially injectable epinephrine. These measures are vital and potentially life-saving, but wouldn’t it be good if we could find ways of switching off the response altogether? We can do this for insect venom hypersensitivity, in most cases, so perhaps we should look more closely at ways of desensitizing for food allergies and drug allergies.

Today’s World Allergy Forum symposium attempts to address these challenges, with the intention of raising awareness of this important condition, its mechanisms, treatment and epidemiology. The organizers hope that you find something useful here, and that you will join the discussion at the end of the presentations.

Anthony J. Frew
President, EAACI
Epidemiology of Anaphylaxis

Aziz Sheikh

Aziz Sheikh, BSc, MBBS, MSc, MD, MRCP, DCH, DRCOG, DFFP, FRCGP
The University of Edinburgh
Edinburgh, Scotland

Aziz Sheikh is Professor of Primary Care Research and Development, Department of General Practice, University of Edinburgh. Prof. Sheikh is a member of the Working Party on Epidemiology of Anaphylaxis of the American Academy of Allergy, Asthma and Immunology, and a member of the British Thoracic Society Research Committee. He holds a Masters degree in Epidemiology. Prof. Sheikh’s research interests include evaluating the clinical effectiveness of established and new treatments for use in primary care, and investigating the epidemiology and clinical management of asthma and other allergic disorders.

Abstract

Characterizing the epidemiology of conditions is difficult in the absence of clearly defined and agreed diagnostic criteria; it is also challenging for conditions that are uncommon and where clinical features are short-lived. Each of these three considerations applies to anaphylaxis, this to a large extent explaining why progress in describing the epidemiology of anaphylaxis has been so slow. But despite the inherent challenges in studying anaphylaxis, there have been a number of important studies published over the last decade, in particular from Europe, North America, and Australasia, which have employed a range of innovative methodological approaches to progressing understanding of this potentially fatal disorder. This paper will summarize some of the key studies conducted to-date, critically appraise their strengths and limitations, and reflect on the clinical implications of their findings.

Key references

1. Sampson HA et al. JACI 2005; 115: 584-91
2. Sampson HA et al. JACI 2006; 117: 391-97
9. Stewart A et al. QJM 1996; 89: 859-64
Epidemiology of Anaphylaxis

Aziz Sheikh
Professor of Primary Care Research & Development
University of Edinburgh

Overview

• Challenges of studying uncommon disorders
• Populations studied
• Anaphylaxis:
  – Incidence
  – Trends over time
  – Age, gender, geographical & socio-economic variations
  – Triggers
  – Outcomes
• Clinical implications

Studying anaphylaxis

• Epidemiology is the “study of the distribution and determinants of health-related states or events in specified populations and the application of this study to the control of health problems” (Last)
• Fundamental assumption is that disease distribution is not random
• Depends crucially on clear definitions of case status and population under study
• Studying the epidemiology of disorders is difficult if:
  – No clear/agreed diagnostic criteria
  – Uncommon
  – Rapid onset, short-lived and reversible
UK anaphylaxis death register

- Objective
  - To understand the circumstances leading to fatal anaphylaxis
- Methods
  - Running since 1992; ONS mortality data coded for anaphylaxis since 1993
  - Detailed information obtained from medical records, medical staff, coroners officers and mast cell serum tryptase
- Main findings
  - ~20 recorded deaths/year i.e. ~1.2.8 million
  - 50% iatrogenic; 25% food and 25% venom
  - ~50% died from asphyxia (food) and 50% from shock (iatrogenic and venom)
  - Median time to death: 5mins if iatrogenic; 15mins venom; and 30 mins food
  - Adrenaline rarely used before cardiac arrest

International Collaborative Study of Severe Anaphylaxis

- Objective
  - To quantify the risk of anaphylaxis due to drugs and other exposures in hospital patients
- Methods
  - Hospitals in Sweden, Hungary, India and Spain
  - Incident cases 1992-1995
  - Clinical diagnosis using a priori agreed criteria, independent of presumed trigger
- Main findings
  - 123,481,752 i.e. risk of 15-20/100,000 admissions
  - 33% males
  - Median age ~53
  - 79% respiratory symptoms; 70% cardiovascular symptoms; 49% both
  - Death in 2% of cases
Hospital discharge data

- Almost all emergency care in the UK is provided by the National Health Service
- Anaphylaxis frequently necessitates hospital admission
- Studying hospital admissions data can offer a window onto the epidemiology of anaphylaxis

Objectives
- To describe patterns of hospital admissions for anaphylaxis in England during 1991/2 – 1994/5:
  - time trends
  - aetiology
  - age and sex variations
  - socio-economic and geographical variations

International Classification of Diseases codes

<table>
<thead>
<tr>
<th>ICD-9</th>
<th>ICD-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-1995</td>
<td>Post-1995</td>
</tr>
<tr>
<td>995.0 Anaphylactic shock</td>
<td>T78.0 Anaphylactic shock due to adverse food reaction</td>
</tr>
<tr>
<td>999.4 Anaphylactic shock due to serum</td>
<td>T78.2 Anaphylactic shock, unspecified</td>
</tr>
<tr>
<td></td>
<td>T80.5 Anaphylaxis due to serum</td>
</tr>
<tr>
<td></td>
<td>T88.8 Anaphylactic shock due to adverse effect of correctly administered medication</td>
</tr>
</tbody>
</table>


[Bar chart showing the number of discharges for anaphylaxis by cause and year]
Anaphylaxis discharge rates by socio-economic status

<table>
<thead>
<tr>
<th>Deprivation</th>
<th>Anaphylaxis discharges (n)</th>
<th>% Pop</th>
<th>Anaphylaxis discharge rate (95% CI), per 100,000</th>
<th>Rate ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UPA &lt; -7</td>
<td>556</td>
<td>26</td>
<td>19.9 (19.0 to 22.4)</td>
<td>1</td>
</tr>
<tr>
<td>UPA -7 to &lt;4</td>
<td>949</td>
<td>24</td>
<td>19.3 (17.8 to 21.0)</td>
<td>0.94 (0.83, 1.06)</td>
</tr>
<tr>
<td>UPA 4 to 18</td>
<td>601</td>
<td>25</td>
<td>17.6 (16.3 to 19.1)</td>
<td>0.86 (0.76, 0.96)</td>
</tr>
<tr>
<td>UPA &gt;18</td>
<td>541</td>
<td>25</td>
<td>13.8 (12.7 to 15.0)</td>
<td>0.67 (0.59, 0.75)</td>
</tr>
</tbody>
</table>


![Graph showing trends in discharge rates for systemic allergic disorders](image)

**English A&E**

- **Objective**
  - To describe the incidence, aetiology and management of anaphylaxis in A&E

- **Methods**
  - Two retrospective case note analyses in 1993 & 1994 in one A&E department in Cambridge

- **Main findings**
  - Anaphylactic shock in 1/1500 – 1/6000 attendances
  - Does not include episodes without collapse
  - 24% gave a history of previous allergic reaction to the suspected cause
  - Very poor access to self-administered adrenaline
  - Only 1/3 treated with adrenaline in A&E

**Stewart A et al. QJM 1996; 89: 659-64**

**Hong Kong ED**

- **Objective**
  - To describe the epidemiology, clinical features and management of anaphylaxis

- **Methods**
  - Retrospective review of all age patients in one ED, 1999-2003
  - Clinical diagnosis of anaphylaxis

- **Main findings**
  - ~1/2600 attendances for anaphylaxis
  - Median age of 28 years; 59% male
  - 19% of patients had history of asthma
  - Trigger: food in 50% (mainly sea food); drugs (41%); venom (7%)
  - 67% treated with epinephrine
  - Biphasic reactions reported in 15% (mean time 8 hours; range 1-23h)

Australian children’s ED

- Objective
  - To describe the epidemiology, aetiology, clinical features and management of anaphylaxis in Australia
- Methods
  - Retrospective case note review <16 in one ED over 3 years (1998-2001)
  - ICD/ASCIA criteria
- Main findings
  - 1/1000 attendances for anaphylaxis
  - Median age 4.1 (range 0.2-14.1)
  - Males: Females 1.7:1
  - Most common triggers: food (56%); drug (5%); venom (5%)
  - Reaction to previously known trigger in 21%
  - Adrenaline used in 16% pre-hospital and in 13% in-hospital (total=39% of severe cases); no fatalities


Swiss Canton Bern

- Objective
  - To estimate the incidence and causes of severe anaphylaxis with circulatory signs
- Methods
  - Study of Canton Bern area (pop 940,000), over 3 years 1996-1998
  - Assessment of clinical records of clinics, EDs and hospitals
- Main findings
  - 246 episodes
  - Incidence rate of 7.9-9.6 per 100,000 person-years
  - Triggers: stings (59%); drugs (18%) and food (10%)
  - Deaths in 3/246 (1.2%)


Australian Cape Territory

- Objective
  - To examine the incidence of anaphylaxis and risk factors for recurrence
- Methods
  - 432 patients referred to allergy clinic 1995-2000
  - 674 person years follow-up
- Main findings
  - Incidence of 9.9 per 100,000 person-years
  - Mean age 27.4 (SD 19.5)
  - 73% atopic; atopy was a significant risk factor for food anaphylaxis, but not other forms
  - 1/12 experience recurrence in a year
  - Compliance with carrying and using adrenaline is poor

**HMO in Washington US**

- **Objective**
  - To estimate incidence and clinical features of anaphylaxis
- **Methods**
  - Secondary analysis of HMO data of children and adolescents from 1991-1997
  - ICD codes
- **Main findings**
  - Incidence rate of 10.5 per 100,000 person-years
  - 71% treated in ED
  - 11% resulted in hospital admission

  Banhe K. et al. JACI 12004;113:536-42.

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**UK GP Research Database**

- **Objective**
  - To describe the incidence, cause and severity of anaphylaxis
- **Methods**
  - Secondary analysis of 8 million records from 1994-1999
  - Random selection of case records reviewed to verify diagnosis
  - Further clarification from GP sought in a sample of cases
- **Main findings**
  - 675 cases; ~8 million person years follow-up
  - Crude incidence rate estimated at 8.4 per 100,000 person-years
  - Most common causes were insect stings (32%), drugs (30%) and food (22%)
  - 65-70% subsequently seen in A&E or hospitalised
  - 10% associated with hypotension/shock requiring resuscitation
  - 1 death (0.1%)


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**Epinephrine dispensing in Canada**

- **Objective**
  - To use epinephrine dispensing patterns to understand the epidemiology of anaphylaxis
- **Methods**
  - Administrative claims pharmaceutical database
  - Epinephrine prescribing over 5 years
- **Main findings**
  - 0.95% of the general population had injectable epinephrine dispensed
  - Rates highest in younger people
  - Males > females in younger age groups, but from 15-64 higher in females; no difference in elderly people

Conclusions

- Anaphylaxis is uncommon, but appears to be increasing
- Significant independent age, gender, socioeconomic and geographical variations in anaphylaxis incidence are likely
- Most common triggers are: drugs, food and venom, and these vary with age
- Drug and venom induced anaphylaxis typically cause cardiovascular compromise and progress rapidly
- Food induced anaphylaxis will typically cause respiratory difficulties
- Risk of recurrence is high (1/12 year) and most people are ill-prepared
- Death is an infrequent outcome, but is often associated with poor self- and professional-management
- In summary, these findings suggest that environmental factors are important in determining risk and outcomes of anaphylaxis and these may therefore be modifiable
Mechanisms of Anaphylaxis

Richard F. Lockey, M.D.
Professor of Medicine, Pediatrics and Public Health
Director, Division of Allergy and Immunology
Joy McCann Culverhouse Chair in Allergy and Immunology
Department of Internal Medicine
University of South Florida College of Medicine
and James H. Haley Veterans’ Hospital

Dr. Lockey is Director of the Division of Allergy and Immunology and Professor of Medicine and Pediatrics, and Joy McCann Culverhouse Chair of Allergy and Immunology at the University of South Florida College of Medicine in Tampa, Florida. He is also a Professor of Public Health at the University of South Florida School of Public Health and Chief of the Section of Allergy and Immunology at the James A. Haley Veterans’ Hospital. Dr. Lockey has authored, co-authored or edited over 500 scientific publications. He is Treasurer of the World Allergy Organization and is the current editor of the World Allergy Organization Web site and the Organization’s e-letter, WAO News and Notes. Among his many distinctions, Dr. Lockey is a Past-President of the American Academy of Allergy, Asthma and Immunology.

Abstract
Anaphylaxis is a syndrome with varied mechanisms, clinical presentations, and severity and is an acute life-threatening reaction, mediated by an immunologic mechanism (allergic, IgE and non-IgE), but not always (non-allergic), that results from the sudden systemic release of mast cells and basophile mediators. These mediators result in some or all of the following signs and symptoms: diffuse erythema, pruritus, urticaria and/or angioedema, bronchospasm, laryngeal edema, hyperperistalsis, hypotension, and/or cardiac arrhythmias. Other symptoms can occur, such as nausea, vomiting, lightheadedness, headache, feeling of impending doom, uterine cramps, and unconsciousness. Usually reactions are uniphasic but also can be biphasic or prolonged.

IgE allergic mechanisms account for most anaphylaxis; however, non-IgE-mediated allergic anaphylaxis also occurs, such as with cytotoxic and immune complex reactions. The causes for non-allergic anaphylaxis, as occurs in radio contrast media reactions, remain unknown.

The end result is that when acute anaphylaxis occurs; it is potentially a life-threatening reaction. Understanding the allergic and non-allergic mechanisms which trigger anaphylaxis is important. So, too, is understanding the pathophysiologic consequences of these reactions in order to assure appropriate treatment and duration of treatment.

References:


Mechanisms of Anaphylaxis

Richard F. Lockey, M.D.

University of South Florida
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James A. Haley Veterans’ Medical Center
Tampa, Florida
U.S.A.

Discovery of Anaphylaxis

- Charles R. Richet and Paul Jules Portier
- Purpose: Discovery of antitoxin to venom of Portuguese man-of-war
- Instead of protection, it triggered anaphylaxis
- Instead of *physaxis* (protection) added *ana* or *anaphylaxis*
- Received Nobel prizes
Definition of Anaphylaxis

- **Anaphylaxis** – an acute systemic allergic reaction resulting from the activation of mast cells and basophils with the release of chemical mediators which cause an acute response. The response can be severe enough to cause death.

- Usually mediated by an allergic or immunologic mechanism, **allergic anaphylaxis**.

- Includes **non-allergic anaphylaxis** (formerly referred to as an anaphylactoid reaction).

  *WAO Nomenclature Review Committee JACI 2004*

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Revised Nomenclature for Anaphylaxis

- **Anaphylaxis**
  - Allergic anaphylaxis
  - Non-allergic anaphylaxis (previous anaphylactoid)
    - IgE mediated Anaphylaxis
    - Non-IgE mediated anaphylaxis

  *WAO Nomenclature Review Committee JACI 2004*

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Mechanisms and Pathophysiology of Anaphylaxis
Shock Organs in Anaphylaxis

a) Guinea pig – bronchial smooth muscle constriction.
b) Rabbit – fatal pulmonary artery vasoconstriction with right ventricular failure.
c) Dog – venous system of liver contracts producing hepatic congestion.
d) Mouse – could be the ideal immunologic model. What about organ response?
e) Human – shock organs are the cardiovascular system, respiratory tract, skin, and gastrointestinal tract. Laryngeal edema, respiratory failure, and circulatory collapse are common. Asthma is an important risk factor for death from anaphylaxis.


Gell and Coombs Hypersensitivity (Immunopathologic Reactions)

- Type I Immediate hypersensitivity - IgE Allergic Anaphylaxis
- Type II Cytotoxic reactions – non-IgE Anaphylaxis
- Type III Immune Complex reactions – non-IgE
- Non-allergic anaphylaxis

Types I, II and III can result in immunologically-induced or allergic anaphylaxis

Kemp and Lockey JACI 2002

IgE-Mediated Anaphylaxis

- Allergen
- Mast cell granules
- IgE antibody
- Immediate reaction
  - Wheal
  - Urticaria
  - Hypotension
  - Abdominal cramping

Phil Lebovics: Anaphylaxis, a clinician’s manual (modified)
The Late Phase Reaction in Anaphylaxis

Protracted Anaphylaxis

- Initial Symptoms
- Time
- Antigen Exposure
- Possibly >24 hours

Involvement of Histamine Receptors in Anaphylaxis and Anaphylactoid Reactions

- H1 receptor-mediated effects
  - Vascular permeability
  - Smooth muscle contraction
  - Vasodilatation
    - Endothelial cell relaxing factor (nitric oxide)
    - Direct effect
  - Cardiac effects
    - Increased rate of depolarization of sinoatrial node
    - Coronary artery vasospasm
  - Stimulation of nerve endings
    - Neuropeptide release
    - Pruritus
    - Vagal irritant receptors
  - Increased mucous gland secretion (viscosity)

Lieberman PL. Melscape General Medicine 1999;5(1)
Involvement of Histamine Receptors in Anaphylaxis and Anaphylactoid Reactions

- \(H_2\) receptor-mediated effects
  - Cardiac effects
  - Positive inotropic effects
  - Positive chronotropic effects
  - Decreased fibrillation threshold
  - Vasodilation
  - Mucus glycoprotein secretion from goblet cells and bronchial glands

- Requires both \(H_1\) and \(H_2\) receptors for maximum effect
  - Vasodilation
  - Hypotension
  - Flush
  - Headache
  - Increased mucous gland secretion (amount)

Liebman PL. Medscape General Medicine 1996;1(1)

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Effects of Pretreatment with Histamine Receptor Antagonists on Widening of Pulse Pressure in Response to Increasing Concentrations of Plasma Histamine

![Graph showing effects of histamine receptor antagonists on pulse pressure](image1)


---

Mast Cell and Basophil Mediators

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Pathophysiology</th>
<th>Clinical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukotrienes</td>
<td>Smooth muscle contraction and vascular permeability</td>
<td>Wheeze and hypotension</td>
</tr>
<tr>
<td>Prostaglandins</td>
<td>Increased vascular permeability and smooth muscle contraction</td>
<td>Wheeze and hypotension</td>
</tr>
<tr>
<td>Platelet-activating Factor</td>
<td>Increase in vascular permeability</td>
<td>Hypotension, activation of clothing</td>
</tr>
</tbody>
</table>
### Mast Cell and Basophil Mediators

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Pathophysiology</th>
<th>Clinical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kallikrein</td>
<td>Activate contact system</td>
<td>Hypotension, angioedema</td>
</tr>
<tr>
<td>Tryptase</td>
<td>Activate contact system, complement, clotting</td>
<td>Hypotension, angioedema, disseminated intravascular coagulation</td>
</tr>
<tr>
<td>Heparin</td>
<td>Inhibits clotting, anticomplementary</td>
<td>Anti-inflammatory</td>
</tr>
<tr>
<td>Chymase</td>
<td>Genesal of angiotensin II</td>
<td>Modulates hypotension</td>
</tr>
</tbody>
</table>

### Pathway Activation During Anaphylaxis

![Pathway Diagram]

- Clotting
- Factor XII
- Kallikrein
- Contact system

### Formation of Nitric Oxide

- Bradykinin
- Histamine
- Leukotriene
- PAF
- Substance P

![Nitric Oxide Formation Diagram]

- L-arginine
- NO
- L-citrulline
### Endogenous Compensatory Mechanisms for Hypotension

![Diagram showing endogenous mechanisms for hypotension]

*Phil Leberman. Anaphylaxis, a clinician's manual (modified)*

### Table: Changes in Hemodynamic Variables During the Development of Shock

<table>
<thead>
<tr>
<th>Hemodynamic Variable</th>
<th>Onset of Shock</th>
<th>After a Few Minutes Without Treatment</th>
<th>Prolonged Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic vascular resistance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac output</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic volume</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac rate</td>
<td>±</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central venous pressure (CVP)</td>
<td>Stable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary capillary wall pressure (PCWP)</td>
<td>Stable</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


### Bradycardia May Be More Common Than Appreciated

- Sting challenge
- 19 subjects
- All 8 subjects who experienced hypotension had an initial tachycardia followed by bradycardia

Gell and Coombs Hypersensitivity (Immunopathologic Reactions)

- Type I Immediate hypersensitivity - IgE
- Type II Cytotoxic reactions – non-IgE
- Type III Immune Complex reactions – non-IgE
- Non-allergic anaphylaxis

Types I, II and III can result in immunologically-induced or allergic anaphylaxis

Kemp and Lockey JACI 2002

Agents that Cause Non-IgE-Mediated Anaphylaxis

Cytotoxic (Type II) – Non-IgE

- Transfusion reactions to cellular elements (IgG, IgM)

Kemp Immunol Allergy Clin N Am 2001
Gell and Coombs Hypersensitivity
(Immunopathologic Reactions)

- Type I Immediate hypersensitivity - IgE
- Type II Cytotoxic reactions – non-IgE
- Type III Immune Complex reactions – non-IgE

- Non-allergic anaphylaxis

Types I, II and III can result in immunologically-induced or allergic anaphylaxis

Kemp and Lockey JACI 2002

Agents that Cause Non-IgE-Mediated Anaphylaxis

Immune aggregates (Type III) – Non-IgE

- Intravenous immunoglobulin
- Dextran (possibly)

Kemp Immunol Allergy Clin N Am 2001
Gell and Coombs Hypersensitivity (Immunopathologic Reactions)

- Type I Immediate hypersensitivity - IgE
- Type II Cytotoxic reactions – non-IgE
- Type III Immune Complex reactions – non-IgE
- Non-allergic anaphylaxis

Types I, II and III can result in immunologically-induced or allergic anaphylaxis

*Kemp and Lockey JACI 2002*

---

Agents that Cause Non-Allergic Anaphylaxis

*Multimediator complement activation/activation of contact system:*

**Radioccontrast media**
- Ethylene oxide gas on dialysis tubing
- Protamine (possibly)
- ACE-inhibitor administered during renal dialysis with sulfonated polyacrylonitrile, cuprophane, or polymethylmethacrylate dialysis membranes

*Kemp Immunol Allergy Clin N Am 2001*

---

Agents that Cause Non-Allergic Anaphylaxis

*Disturbances in arachidonic acid metabolism*

Aspirin and other non-steroidal anti-inflammatory drugs

*Kemp Immunol Allergy Clin N Am 2001*
Agents that Cause Non-Allergic Anaphylaxis

Nonspecific degranulation of mast cells and basophils

- Opiates
- Physical factors:
  - Exercise
  - Temperature (cold, heat)
- Catamenial (menstrual)
- Idiopathic causes

*Kemp Immunol Allergy Clin N Am 2001*

---

Laboratory Tests in the Diagnosis of Anaphylaxis

![Graph showing plasma histamine, serum tryptase, and 24-hour urinary histamine metabolites over time.](image)

---

### Mouse Anaphylaxis

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>IgE-dependent anaphylaxis</th>
<th>IgE-independent anaphylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Requirement for IgE</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Requirement for IgG</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Requirement for FcεRI</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Requirement for FcyRII</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Mediated by histamine</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Mediated by PAF</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Mediated by mast cells</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Mediated by macrophages</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Requires high concentrations of antibody</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Requires large amounts of antigen</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Inhibited by blocking antibody</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

### Mouse and Human Immune Systems

Anaphylaxis-relevant similarities and differences between the mouse and human immune systems:

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mouse</th>
<th>Human</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of IgE</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Presence of FcεRI on mast cells and basophils</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Presence of FcεRI on macrophages and Langhans cells</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Presence of FcγRIib on mast cells</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Presence of FcγRIII on macrophages</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Presence of FcγRIIA, FcγRIIB, and FcγRIIC</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Mast cell production of histamine</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Macrophage production of PAF</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Activation of mast cells by IgG</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>(total)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IgE binding to FcγRIib and FcγRIII</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>


---

### Of Mice and Men

1. Anaphylaxis in the mouse can occur through 2 independent pathways: one that involves IgE, FcεRI, mast cells, PAF, and histamine and one that involves IgG, FcγRIII, macrophages, and PAF.

2. Considerably less antibody and antigen are required to trigger IgE-dependent anaphylaxis than IgG-mediated anaphylaxis.

3. Most human anaphylaxis is probably IgE-dependent because it involves small quantities of antigen.


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### Of Mice and Men (cont'd)

4. IgG and IgA blocking antibodies inhibit the ability of small quantities of antigen to induce IgE-dependent anaphylaxis by neutralizing antigen before it can cross-link mast cell-associated IgE.

5. IgG antibodies also inhibit IgE-dependent anaphylaxis by mediating an interaction between FcεRI and FcγRIIb on mast cells.

6. Gastrointestinal anaphylaxis is induced by an IgE/FcεRI/mast cell/PAF plus serotonin pathway and can cause systemic symptoms if levels of blocking antibodies are low.

7. Pulmonary involvement in anaphylaxis requires pre-existing pulmonary allergic inflammation.

Management of Anaphylaxis

F. Estelle R. Simons, MD, FRCPC
University of Manitoba
Winnipeg, Manitoba, Canada

Dr. Estelle Simons is a Professor in the Department of Pediatrics & Child Health and the Department of Immunology in the Faculty of Medicine at the University of Manitoba. She served as the 2005-2006 President of the American Academy of Allergy, Asthma, and Immunology. She is a member of the Board of Directors of the World Allergy Organization. She is a past-President of the Canadian Society of Allergy & Clinical Immunology. She has authored or co-authored more than 400 original peer-reviewed publications and has edited and co-edited several textbooks, including Middleton’s Allergy Principles & Practice, Sixth Edition (2003).

Abstract
Physicians play a critically important role in the acute management of anaphylaxis. This role involves: assessing the patient’s airway, breathing, circulation, and mentation, injecting epinephrine, maintaining the airway, placing the patient supine, and administering oxygen and large volumes of intravenous fluids. Additional measures may be needed.

Allergy/immunology specialists should also be cognizant of their responsibilities in the long-term management of individuals who have experienced anaphylaxis. Their unique role involves risk assessment: verification of the trigger by obtaining a comprehensive history of the anaphylaxis episode and performing relevant investigations, as well as determining co-morbidities and concomitant medications. Risk assessment should be followed by long-term, personalized, trigger-specific, risk-reduction strategies. These may include: allergen avoidance for prevention of food-, medication-, or latex-induced anaphylaxis, immunotherapy for prevention of venom-induced anaphylaxis, and prophylactic medications for idiopathic anaphylaxis.

Despite vigilant avoidance of the trigger and good compliance with treatment, at-risk individuals may experience anaphylaxis again. They therefore need to be trained to use self-injectable epinephrine, reminded that they cannot depend on an oral antihistamine or an asthma puffer in anaphylaxis, and equipped with accurate medical identification and an Anaphylaxis Emergency Action Plan.

Anaphylaxis education should be provided for individuals at risk, their families and caregivers, healthcare professionals, and the general public.

References
Joint Task Force on Practice Parameters, American Academy of Allergy, Asthma, and Immunology, American College of Allergy, Asthma, and Immunology, Joint Council of Allergy Asthma and Immunology. The diagnosis and management of anaphylaxis: an updated practice parameter. J Allergy Clin Immunol 2005;115(Suppl.):S483-S523.
MANAGEMENT OF ANAPHYLAXIS

F. Estelle R. Simons, MD FRCPC

Department of Pediatrics & Child Health
Department of Immunology
CIHR National Training Program in Allergy & Asthma
The University of Manitoba

OBJECTIVES

...to describe the dual role of the allergy/immunology specialist:

• in acute anaphylaxis (traditional role)
  - assessment
  - treatment

• in long term anaphylaxis management (unique role)
  - assess risk (confirm diagnosis and trigger)
  - reduce risk (develop an individualized plan)
  - educate (patients, health care professionals, public)

ANAPHYLAXIS: A MODERN DISEASE

TIMELINES

Simone FEIR, J Allergy Clin Immunol 2006;117:247-77
ANAPHYLAXIS 2006

- no universally accepted definition
- true prevalence unknown
- many episodes occur in the community
- under-recognized, under-diagnosed, and under-treated

Simons FE. J Allergy Clin Immunol 2006;117:367-77

ANAPHYLAXIS MANAGEMENT 2006

- for acute anaphylaxis episodes, there are limited treatment options
- randomized double-blind, placebo-controlled trials are not feasible
- treatment of episodes depends on prompt epinephrine injection
- risk assessment and long-term risk reduction are critical

Simons FE. J Allergy Clin Immunol 2006;117:367-77
PHYSICIAN-SUPERVISED MANAGEMENT

- assess airway, breathing, circulation and mentation
- inject epinephrine* IM (into the muscle of the anterolateral thigh)
- place patient in recumbent position and elevate lower extremities
- maintain airway (laryngeal mask airway, endotracheal tube, cricothyrotomy)
- administer oxygen, 6-8 L/min
- give normal saline IV (volume expanders/collodids for severe hypotension)

*0.3 - 0.5 mg (0.01 mg/kg in children)

MEASURES FOR SPECIFIC CLINICAL SITUATIONS

- urticaria
  - diphenhydramine 50 mg IV (1 mg/kg in children)
  - ranitidine 50 mg (1 mg/kg in children), diluted, injected IV over 5 min
- bronchospasm
  - nebulized albuterol (salbutamol) 2.5 - 5 mg in 3 ml 0.9% NaCl
- refractory hypotension
  - dopamine 400 mg in 500 ml normal saline, IV 2 - 20 µg/kg/min
  - glucagon 1 - 5 mg (20 - 30 µg/kg, max 1 mg in children), IV over 5 min followed with continuous IV infusion 5-15 µg/min
- general supportive treatment
  - methylprednisolone 1 mg/kg per 24 hr
LONG-TERM MANAGEMENT

- assess risk: confirm diagnosis and trigger
- reduce risk: develop an individualized plan
- educate: patients, health care professionals, public

RISK ASSESSMENT

- confirm the diagnosis
- identify the trigger(s)
- consider the possibility of a novel trigger
- identify relevant co-morbidities
- identify relevant concomitant medications
- consider the possibility of a novel mechanism
ANAPHYLAXIS: RISK REDUCTION

- prepare for recurrence
  - coach: epinephrine autoinjector use
  - provide accurate medical identification
  - develop or update Emergency Action Plan

- prevent recurrence
  - review trigger-specific avoidance strategies
  - start trigger-specific preventive treatment, if available

EPINEPHRINE: ESSENTIAL LIFE-SAVING MEDICATION

![Graph showing plasma epinephrine levels](image)

Simons FER. J Allergy Clin Immunol. 2003;111:397-77

SELF-INJECTABLE EPINEPHRINE (SIE) IN ANAPHYLAXIS

- SIE dispensed for 0.96% of a general population (range 0.32% - 1.44%) (Simons FER et al. J Allergy Clin Immunol 2009;124:447-51)

- inadequate range of doses in autoinjectors: 0.3 mg prescribed from 20 mo – 17 yr; 0.15 mg from 2 mo – 17 yr. (Simons FER et al. Ann Allergy Asthma Immunol 2019;122:525-40)

- life-saving SIE available in only ≤ 50% of countries (Simons FER, Ann Allergy Asthma Immunol 2015;115:531-4)
LIFE-SAVING EPINEPHRINE® OUT OF REACH FOR MANY IN NEED

ADULTS

56.4% no country

43.6% no country

CHILDREN

INFANTS

no country

* Epipen, Epipen Jr, FastiPen, Anapen, Anadren

Saito RN, Annals of Allergy 2013; 110: 124-8

AUTOINJECTOR DOSE FOR INFANT/CHILD

<table>
<thead>
<tr>
<th>Body weight</th>
<th>≤5 kg</th>
<th>10 kg</th>
<th>15 kg</th>
<th>20 kg</th>
<th>25 kg</th>
<th>≥30 kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>50th percentile</td>
<td>2 mo</td>
<td>14 mo</td>
<td>3 yr</td>
<td>6 yr</td>
<td>9 yr</td>
<td>12 yr</td>
</tr>
<tr>
<td>Optimal dose (mg)</td>
<td>0.05</td>
<td>0.1</td>
<td>0.15</td>
<td>0.2</td>
<td>0.25</td>
<td>0.3</td>
</tr>
<tr>
<td>Autoinjector 0.15 mg</td>
<td>3× OD</td>
<td>1.5× OD</td>
<td>optimal</td>
<td>1.3× UD 1.7× UD</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Autoinjector 0.3 mg</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1.5× OD</td>
<td>1.2× OD</td>
<td>optimal</td>
</tr>
</tbody>
</table>

OD = overdose; UD = underdose

Saito RN, J Allergy Clin Immunol 2004; 113: 827-844

BODY WEIGHT 22 kg: EPINEPHRINE® 0.15 mg or 0.3 mg?

Decide on 0.3 mg autoinjector if one or more of the following apply:

- peanut, tree nut, milk, egg, seafood, fish or insect sting trigger
- concurrent diagnosis of asthma
- history of a previous life-threatening reaction
- poor access to emergency medical services
- dysfunctional or chaotic family situation

* autoinjector 0.15 mg = 1.5-fold underdose; autoinjector 0.3 mg = 1.3-fold overdose

Saito RN, J Allergy Clin Immunol 2004; 113: 827-844
EPINEPHRINE (EPI) PROSPECTIVE CLINICAL PHARMACOLOGY STUDIES

- In adults, EPI IM, thigh is absorbed faster than EPI IM or SC, arm (Gomesa FGK et al. J. Allergy Clin. Immunol. 2008;122:871-7)

- In children, EPI IM, thigh is absorbed faster than EPI SC, deltoid (Gomesa FGK et al. J. Allergy Clin. Immunol. 2008;121:33-7)

- Sublingual EPI is absorbed rapidly and holds considerable promise (Gomesa FGK et al. J. Allergy Clin. Immunol. 2008;121:10)

IM = intramuscular, SC = subcutaneous

SUBLINGUAL EPINEPHRINE ABSORPTION

![Graph showing sublingual epinephrine absorption](image)


EPINEPHRINE (EPI): IN VITRO STUDIES

- Lay individuals can't draw up an EPI dose rapidly/accurately from an ampule (Simon AFS et al. J. Allergy Clin. Immunol. 2008;121:984-9)

- EPI degrades over time in auto-injectors; available dose ↓ as months ↑ past expiry date (Simon AFS, J. Allergy Clin. Immunol. 2008;121:1025-36)
WHY PATIENTS/CAREGIVERS DO NOT INJECT EPINEPHRINE

- failure to recognize the allergic reaction
- reaction seems to be mild; previous spontaneous recovery
- epinephrine auto-injector not available or affordable
- concern about epinephrine adverse effects
- fear of pain/needle/injection
- reliance on asthma “puffers” e.g. salbutamol, or on an oral antihistamine


ORAL H₁-ANTIHISTAMINES

- no randomized, blinded, controlled studies in anaphylaxis
- decrease itching and hives
- no effect on respiratory or GI symptoms, or shock
- slow onset of action: 0.8-3 hours, slow absorption: t₁/₂ = 1-3 hours
- symptoms after oral H₁-antihistamine = spontaneous improvement
- sedating antihistamines impair recognition of CNS symptoms


ANAPHYLAXIS WALLET IDENTIFICATION CARD

PERSONAL IDENTIFICATION
Name: ____________________________
Allergy to: ______________________
Food, drug, insect, venom, exercise, cold? ______________________
Other medical problems: ______________________

WHAT TO DO
- IMMEDIATE ACTION
  • Call 911 or local emergency number
  • Have others call 911
  • Tell them about allergies
  • Give epinephrine
  • Call 911 or local emergency
  • Emergency contact: ______________________
  • Medication allergies: ______________________
  • Other medical problems: ______________________

- IMMEDIATE ACTION
  • Use an auto-injector if available
  • Blood pressure: ______________________
  • Heart rate: ______________________
  • Pulse oximetry: ______________________
  • Respiratory rate: ______________________
  • Skin color: ______________________

Exterior surfaces

Interior surfaces

Sawyer PDR, J Allergy Clin Immunol, 2006;117:362-77
ANAPHYLAXIS EMERGENCY ACTION PLAN

Who is at risk?

How do we know?

What to do?

When to call?

Prevention of Anaphylaxis

- Avoidance

- Avoidance of co-triggers, warm-up, pre-medication

- Immunotherapy

Anaphylaxis From Food: Prevention

- Complete avoidance of food trigger: easier said than done
- Labelling may be absent or hard to read: “difficult” words, small print
- Many labels state “may contain” or “possibly exposed during manufacture”
- Constant vigilance, and lifestyle changes, required every day year-round
- Quality of life for patient and family may be significantly impaired
**PREVENTION OF ANAPHYLAXIS: LONG TERM**

- engineered allergen protein immunotherapy
- plasmid DNA-based immunotherapy
- peptide immunotherapy
- allergen immunostimulatory sequence immunotherapy
- chimeric human-allergen fusion protein

**EDUCATION**

Keep it simple....

- recognize anaphylaxis
- inject epinephrine

**KILLER ALLERGY: ANAPHYLAXIS**

A sudden severe allergic reaction....

- who is at risk?
- how do we know?
- when can it happen?
- where can it happen?
- what should we do?
- why is follow-up needed?
ANAPHYLAXIS EDUCATION: TEACHING THE TEACHERS

Anaphylaxis Simulation

Anaphylaxis Education Tool Kit

ANAPHYLAXIS: QUANDARIES IN FIRST-AID TREATMENT

- under-recognized and under-treated by health-care professionals
- yet, we expect patients/caregivers to recognize and treat it
- anaphylaxis versus: asthma, urticaria, choking, anxiety attack
- physicians: ?anaphylaxis? prescribe epinephrine

Schein Srl, Sieranski R. J Allergy Clin Immunol 2005;115:575-583

EDUCATION IS THE KEY

- patients and their families
- physicians (ED,PCP) and all other health care providers
- others
  - teachers, coaches, camp directors, child care workers
  - food service industry, grocery manufacturers
  - legislators (Food Allergy Labeling and Consumer Protection Act; Sabine’s Law)

American Academy of Allergy, Asthma, and Immunology: www.aaaai.org
Food Allergy and Anaphylaxis Network: www.foodallergy.org
American Latex Allergy Association: www.latexallergyresources.org
SUMMARY

...we've described the dual role of the allergy/immunology specialist:

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  - assessment
  - treatment

- in long term anaphylaxis management (unique role)
  - assess risk (confirm diagnosis and trigger)
  - reduce risk (develop an individualized plan)
  - educate (patients, health care professionals, public)
Bangkok - the cosmopolitan city of delightful contrasts

We look forward to welcoming you to Bangkok at the
World Allergy Congress 2007
2-6 December

WAO
WORLD ALLERGY ORGANIZATION

Asia Pacific Association of Allergology and Clinical Immunology

The Allergy and Immunology Society of Thailand