



Toxic Trauma

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Objectives of the presentation

- Definition of the concept of 'toxic trauma' and its relationship with physical trauma
- Definition of the key properties of toxic agents
- How toxic agents cause trauma to various somatic systems with respiratory failure as a terminal event
- Diagnosis and management of toxic trauma
- A re – examination of the validity of the chemical, biological, radiological and nuclear (CBRN) grouping of hazards from a medical viewpoint.

Potential Conflict of Interest

- Consultant Medical Adviser to Pneupac, Smiths Medical International, UK who manufacture specialized portable ventilators for use in chemically – contaminated zones

The classification of trauma

- Trauma may be defined as the effects of harmful external forces that disrupt both the structure and function of the body
- Usually classified in terms of the various forms of physical trauma
 - **blunt**
 - **penetrating**
 - **blast**
 - **burns**
- The past 50 years have seen the development of integrated systems of trauma management (eg the ATLS programme of the American College of Surgeons) with emphasis on early life support

What is toxic trauma?

- There is a need to integrate the somatic effects of exposure to harmful chemical agents into the overall management of trauma, particularly in emphasising the importance of early and continuing advance life support
- ‘Poisoning’ does not adequately describe the effects of exposure to the many thousands of toxic agents that exist in war and in peace
- ‘**Toxic trauma**’ : may be defined as the disruption of somatic systems and their function by exposure to a toxic agent. This goes beyond the conventional concept of poisoning

Pathways to toxic trauma

- Inhalational
 - Toxic industrial chemicals
 - CW oedemagens eg chlorine, phosgene
 - Nerve agents
 - Incapacitating agents
- Dermal
 - Vesicant CW agents
 - Nerve agents
 - Toxins
- Ingestion
 - Classic route for conventional poisoning
 - Less common for CW agents

Key properties of agents causing toxic trauma

- Physical form
 - Persistency
 - Toxicity
 - Latency
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- Physical form and persistency relate to the management of the toxic release and the transmission hazard
 - Toxicity and latency relate to the pathophysiology following exposure and the management of the patient

Physical form

- Agents causing toxic trauma may be:
- Solid
- Liquid
- Vapour
- Gas
- Aerosol

Persistency

- Very important property of toxic agents
- Determines how long the toxic release remains a hazard and the requirement for decontamination
- CW agent persistency
 - Short: hydrogen cyanide, chlorine, sarin
 - Long: sulphur mustard, VX
- Toxic industrial chemical persistency
 - Short: sulphur dioxide, nitrogen oxides
 - Long: sulphuric acid, toluene di - isocyanate

Toxicity

- Conventional expressions eg LD₅₀, LC₅₀
- For inhaled toxic agents: Haber principle
 - Concentration x time = constant
 - Not always valid
 - Original expression now modified to $C^n t = W$
where C is a constant between 1 and 7

Latency

- Toxic trauma appears at variable times following exposure
- No proper expression of latency has been developed (eg L_{50} - the time taken for signs and symptoms to appear in 50 % of an exposed population)
- Short and long latency agents recognised
- CW :
 - **Short** : sarin, HCN, early effects of lung damaging agents
 - **Long**: lung damaging agents (producing pulmonary oedema), vesicants

Recognition of toxic trauma

- Toxic exposure affects many somatic systems with variable toxicity and latency
- Military chemical exposure management relies on early detection and identification using physical means
- In civil exposure physical detection systems are usually absent
 - Exposure initially revealed by **characteristic signs and symptoms**
- Importance of the early recognition of **toxidromes**
 - Eg - the muscarinic cholinergic syndrome of OP exposure
 - **S**alivation
 - L**achrymation
 - U**rinary incontinence
 - D**efaecation with diarrhoea
 - G**astrointestinal - cramps
 - E**mesis

Toxidromes and somatic systems

- Primary effects on the CNS and PNS
 - Anticholinesterase agents (eg OP CW agents, pesticides)
 - Incapacitating agents (eg BZ)
 - 'Knockdown' agents (eg fentanyl compounds)
- Primary effects on internal and external respiration
 - Lung damaging agents (eg chlorine, phosgene)
 - Active chemical asphyxiants (eg HCN)
 - Passive asphyxiants (eg nitrogen)
- Primary effects on the skin and mucous membranes
 - Sulphur Mustard Gas



Organophosphate cholinergic syndromes: important points

- Classic syndromes such as SLUDGE may not always be apparent
- Importance of a rapid **primary assessment** (as with physical trauma) and to treat any early indications of life – threatening airway blockage and respiratory failure.
- **Advanced life support** must have at least equal priority with antidote treatment.
- In severe cases the use of atropine and oximes will not be sufficient to treat respiratory failure. No effect on neuromuscular paralysis

Respiratory failure following toxic trauma

- Short latency – effects on airway, CNS and PNS.
 - Organophosphates
 - Conventional antidote treatment (atropine, oximes, benzodiazepines) will have little effect on developing respiratory failure. No effect on established NM paralysis
 - **Ventilatory support essential** (cf conventional physical trauma with acute respiratory failure)
- Longer latency
 - Lung damaging agents: onset of pulmonary oedema after a 12 – 18 hour latent period
 - **Intermittent positive pressure ventilation with positive end expiratory pressure (PEEP)** required for severe cases.

Moscow theatre siege 2002: 129 hostages died from respiratory failure



First World War: mass fatalities from toxic pulmonary oedema



Syrian Civil War: short latency civilian fatalities from respiratory failure



Management of toxic trauma: key points

- **Treat** the presenting signs and symptoms and apply early life support measures if necessary
- **Do not** apply inappropriate antidote treatment
 - Eg use of atropine after an attack with vesicant agents
- **Remember** that toxic trauma is an evolving process. Observe patients carefully for 24 hours if possible. Triage and re – triage of great importance
- **Apply** ABCD as for physical trauma
 - **A** = airway and antidotes
 - **B** = breathing and artificial ventilation
 - **C** = circulatory support (hypotension in muscarinic cholinergic syndrome, OP dysrhythmias)
 - **D** = assess disability (respiratory and neurological); supportive therapy (eg steroids)

Artificial ventilation in a contaminated zone



Toxic trauma and the CBRN classification

- Military and civil Hazmat responders have developed a unified approach to chemical, biological, radiological and nuclear injury
- But the nature and latency of trauma produced is very different
- Different approaches required for protection
- CBRN response teams often wear protective equipment that is inappropriate for the potential hazard
- Eg: Level 1 protection essential for highly caustic and toxic materials but inappropriate for a 'white powder' incident



Application of key properties of agents causing toxic trauma to the CBRN classification

- **Chemical**: toxic, short to medium latency, variable persistency
- **Biological**: toxic via secondary mechanisms (eg bacterial toxins) , long latency (incubation period) usually unstable and non - persistent in the atmosphere (exception: spore forming bacteria such as anthrax)
- **Radiological**. Isotopes are stable and persistent (half life up to thousands of years) Absorbed radiation affects many systems of the body with final pathways parallel to toxic trauma
- **Nuclear** : very short latency with release of physical energy causing conventional trauma (physical and burns), immediate release of radiation and radioisotopes. Longer latency effects from fallout.

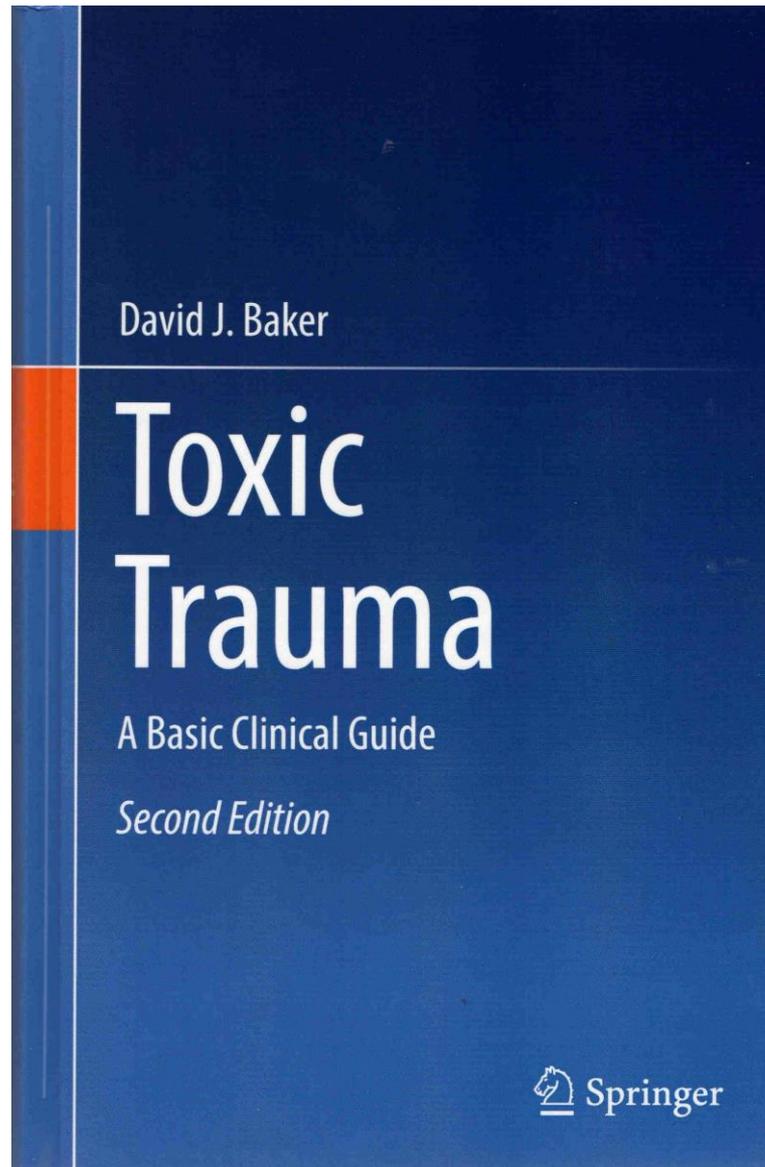
Should we continue to use the CBRN classification in medical management?

- The nature of trauma produced by CBRN agents is very different
- Similarities of approach to the management of CBRN casualties exist in protection, decontamination and emergency life support management
- The idea of a single collective 'CBRN' medical response may cause confusion among responders and is not appropriate to management
- Particularly important to understand the essential differences between chemical and biological agents and how toxic trauma is produced in each case.

Conclusions

- Exposure to toxic chemicals can affect many somatic systems and cause life threatening injury with variable latency: this injury may be termed **'toxic trauma'**
- **Respiratory failure** and cardiac arrest is a common final pathway and must be treated quickly by airway and ventilation support in conjunction with antidotes
- The four **key properties** of toxic chemical agents can be applied to other hazards in the CBRN classification
- Although the CBRN grouping remains a part of military and civil hazmat responses, for medical responders it is essential to understand the differences in the trauma caused by each hazard in the classification and the appropriate management, particularly **advanced respiratory life support**.

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Toxic Trauma

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