LUNGS AT WORK
www.lungsatwork.org.uk

Ammonia tanker leak, Miramas, near Marseille, France. 2001

Acute inhalation injuries

SW Training Day
9th October 2018

Jo Feary

Definition

“An acute exposure to a potentially toxic agent that could cause respiratory illness”

Gas

Vapour (gaseous form of a substance that is in liquid or solid form at normal T and P)

‘Aerosols’ mix of potential states (liquid droplets or fine particulates dispersed in a gaseous medium); smoke is subset resulting from incomplete combustion

Fume (solid material, often metals, of small particle size [most <1.0µm and many <0.1µm] suspended in a gaseous medium that has condensed from a vaporized state)

Questions you might want to ask

Agent factors:

known or unknown?

solubility

highly soluble e.g. ammonia (agriculture), chlorine, sulphur dioxide

low water solubility e.g. Zinc oxide, Phosgene (carbonyl chloride)

quantity inhaled

duration of exposure

temperature of agent (thermal injury esp if particles in the inhaled material)

density of gas (Chlorine in WW1 trenches; displacement of oxygen if lighter than air)

Environment:

poorly ventilated or enclosed areas

elevation (low and high) can be important

others affected?

Host factors:

(age)

use of RPE

pre-existing respiratory disease

Acute management (1) : some principles [agent may be unknown]

Management is largely supportive:
Observe, treat symptoms as arise, recognise possibility of delayed onset of sx
If LOC or hypoxic then observe for 24h

Exclude carbon monoxide poisoning (oxygen saturations may be normal)
Nasal, oral or laryngeal burns, oedema or ulceration may indicate significant exposure
Radiological changes rare; suggest significant exposure

Measurement of **spirometry** if dyspnoea, wheeze or crackles

Acute management (2):

Progressive airway oedema, bronchorrhoea, bronchoconstriction, mucosal sloughing
Distal airways injury: mild interstitial oedema $\longleftrightarrow$ diffuse alveolar damage (hrs to 48h)
Acute complications: pneumothorax , pneumomediastinum

Consider pre-emptive intubation and invasive ventilation
- protect airway from progressive airway and pulmonary oedema
- treat hypoxemia

Management of ARDS/diffuse alveolar damage same as for other causes,
- e.g. protective ventilator strategies
- avoidance of high concentrations of oxygen

No evidence for corticosteroids acutely (although usually given)
Inhaled bronchodilators and ICS if airflow obstruction or ongoing dyspnoea

Avoid prolonged polypharmacy without objective assessment

Prognosis

Majority with acute inhalation injury have no long-term adverse health outcomes

Minority develop longer-term complications:

- case reports; little information on exposure measurement or pre-existing lung fn

- upper airways symptoms e.g. chronic rhinitis and VCD/ILO
- airflow obstruction / non-specific airway hyper-responsiveness (irritant-induced asthma)
- small airways disease e.g. Union Carbide explosion in pesticide factory (Bhopal 1984)
- “interstitial lung diseases”
- ARDS with assoc. complications of obliterative bronchiolitis and bronchiectasis
- PTSD (regardless of exposure and consequence)

51M Engineer and a marine plant specialist

Leisure centre
Exposed to sodium hypochlorite and trichloro-s-triazinetrione, dry
Undefined period of exposure 23rd and 24th August (whilst carrying out checks)
c.one hour on the 24th August at the time of the acute exposure (lid lifted on a tank)
Pulled colleague to safety

Productive cough with haemoptysis, dyspnoea, pleuritic chest pain, wheeze on inclines
Loss of sensation in his finger tips, brittle nails, significant weight loss (BMI 17) , poor memory, unsteadiness on his feet, yellow cataract and a metallic taste in his mouth…..

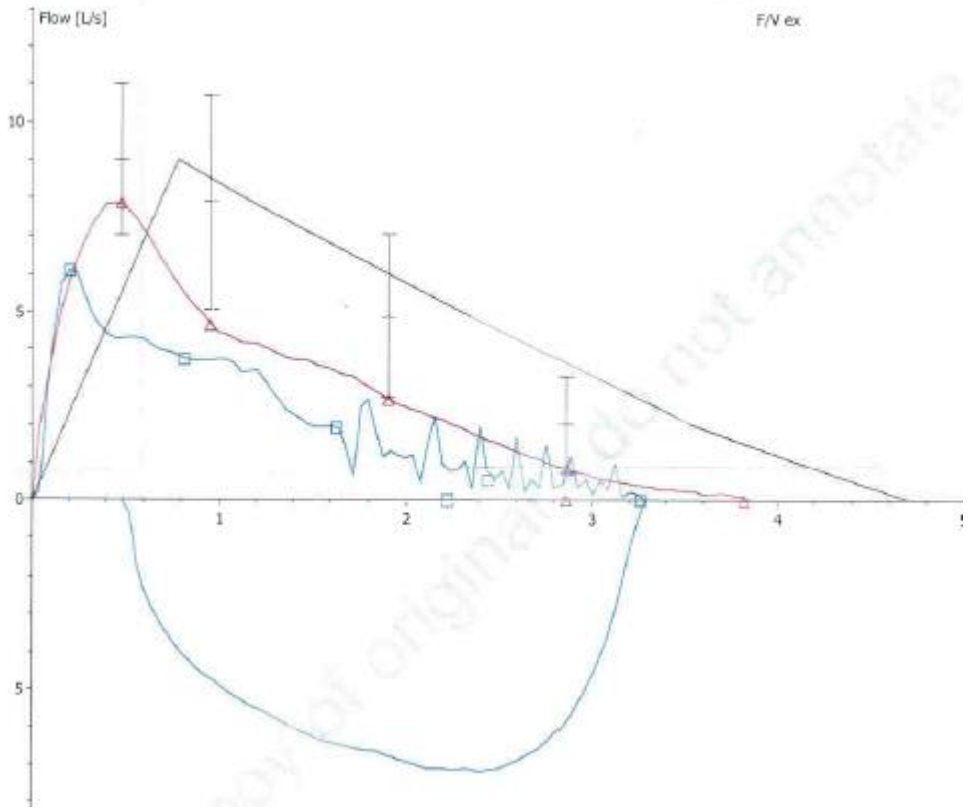
Has not been able to return to work; solicitor involved; angry +++

PMH: IHD, DM; no respiratory disease

DH: Ventolin x12/24h, Fostair 200/6 two puffs bd, beclomethasone, sertraline, tramadol, co-codamol, ibuprofen and aspirin

SH: Smoker of cigarettes and cannabis

	Pred	Pre Test 1	% PRED	SR	POST	% PRED	% CHANGE
Date		28/12/17			28/12/17		
FEV 1	3.77	2.22	59.0	-3.03	2.86	75.8	28.5
FVC	4.70	3.26	69.3	-2.37	3.82	81.2	17.1
VC MAX	4.90	3.26	66.5	-2.93	3.82	77.9	17.1
FEV1%M	78.03	68.27	87.5	-1.36	74.90	96.0	9.7
PEF	9.01	6.09	67.6	-2.42	7.87	87.3	29.2
MEF 25	2.03	0.55	27.1	-1.90	0.84	41.2	52.4
MEF 50	4.89	1.94	39.6	-2.24	2.67	54.6	37.9
MEF 75	7.88	3.74	47.5	-2.42	4.65	59.0	24.3
FET		4.70			6.42		36.5
PIF		7.17					
TLC-SB	7.30	5.57	76.3	-2.48			
RV-SB	2.25	2.41	106.9	0.38			
RV%TLC	33.85	43.20	127.6	1.71			
FRC	3.58	3.68	102.7	0.16			
DLCOc	10.60	6.75	63.6	-2.72			
VA	7.15	5.44	76.1				
KCO	1.45	1.24	85.4	-0.89			
KCOc	1.45	1.24	85.4	-0.89			
VIN	4.90	3.16	64.5	-3.11			
Hb		14.60					

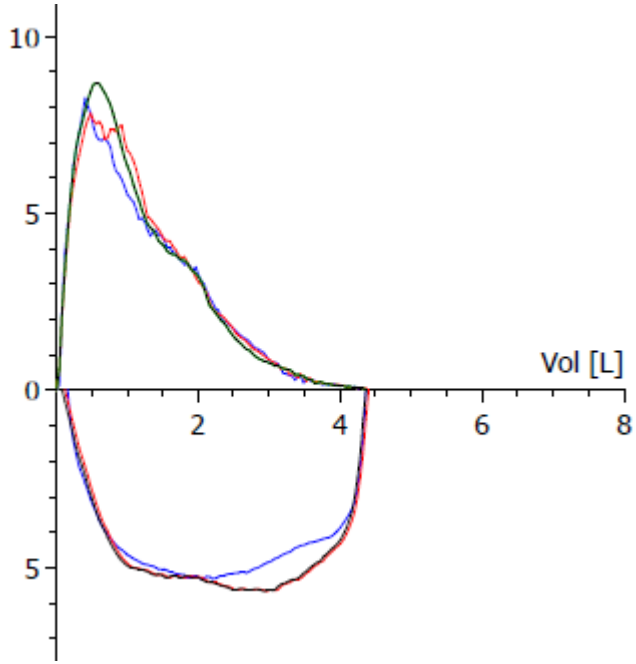


Bloods: **normal**

HRCT: **normal**

Mx: **physio**
analgesia

	Pred LI	Pred UI	Test1	% Pred	S.R.
Date			14/05/18		
FEV 1	2.78	4.45	3.00	83.0	-1.21
FVC	3.51	5.51	4.37	97.0	-0.22
VC MAX	3.78	5.62	4.37	93.1	-0.58
FEV1%M	66.05	89.65	68.61	88.1	-1.28
PEF	6.80	10.77	8.65	98.5	-0.11
MEF 75	4.88	10.50	5.58	72.5	-1.24
MEF 50	2.58	6.92	2.29	48.3	-1.86
MEF 25	1.93	1.93	0.58	29.9	
FET			8.94		
PIF	6.59	6.59	5.66	85.9	



Conclusions:

Irritant induced asthma (RADS)
Histamine challenge now normal
Slow withdrawal of medication
IIDB
Pursuing personal injury claim

Reactive airways dysfunction syndrome (RADS)

- **original criteria**

- documented absence of prior respiratory symptoms
- onset after a single, toxic exposure
- onset within 24 hours
- symptoms consistent with asthma
- NSBHR
- +/- airflow obstruction
- other pulmonary disease excluded

Brooks. Chest 1985;88:376-384

Irritant induced asthma (RADS)

Far less common than first thought

Very high levels of exposure

Hard to sustain diagnosis without evidence of BHR

Even more difficult to diagnose with pre-existing asthma as already evidence of BHR

No hypersensitivity so can tolerate same agent (in contrast with Occupational Asthma)

Often referred with a label and a legal case
Diagnosis may or may not be made appropriately
Consider if it helpful to undo diagnosis

Fear is often a reason not to return to work

52M Self employed sub-contractor for larger company

January 2014 A+E
reported exposure to “Lithofin StainStop” (LSS)
laying tiles in a bathroom and kitchen refurbishment
a small room with no ventilation in the area
operatives from the larger company used spraying equipment to administer LSS throughout the area he was working in.

“He has, he told me, previously used this product himself and reports that the data sheets state that it should be applied by cloth and not to be sprayed, as if inhaled can cause symptoms. He recalls that he became acutely unwell.”

Cough, choking, vomiting ++, dizziness, headache

2/52 inpatient stay, 24 hrs HDU
high dose steroids
lost 30 kg (previously weighed 123kg)

Feb 2015 RBH clinic
off steroids for some months
9 months to regain his fitness enough to return to work (lost business)
SOBOE on minimal exertion (prev. fit)
“prone to chest infections”

Clinical examination normal. Sats 98% on air. Non atopic.

06/14 (local)

FEV_1	2.81L (83%)
FVC	4.71L (112%)
Ratio	60%

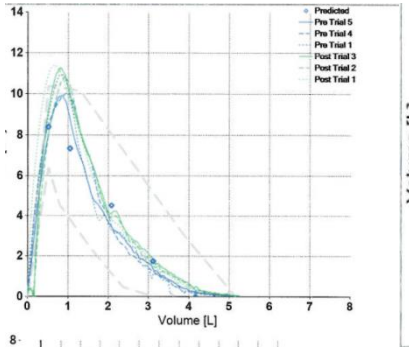
Gas transfer 65% predicted

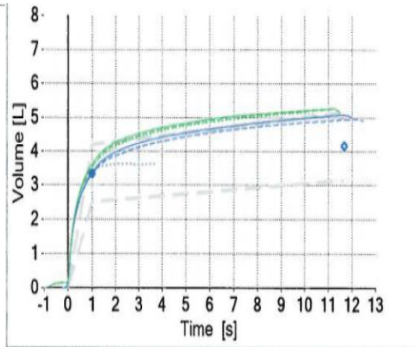
Lung function 02/15

FEV_1	3.38L (101%)
FVC	5.08L (122%)
Ratio	66%

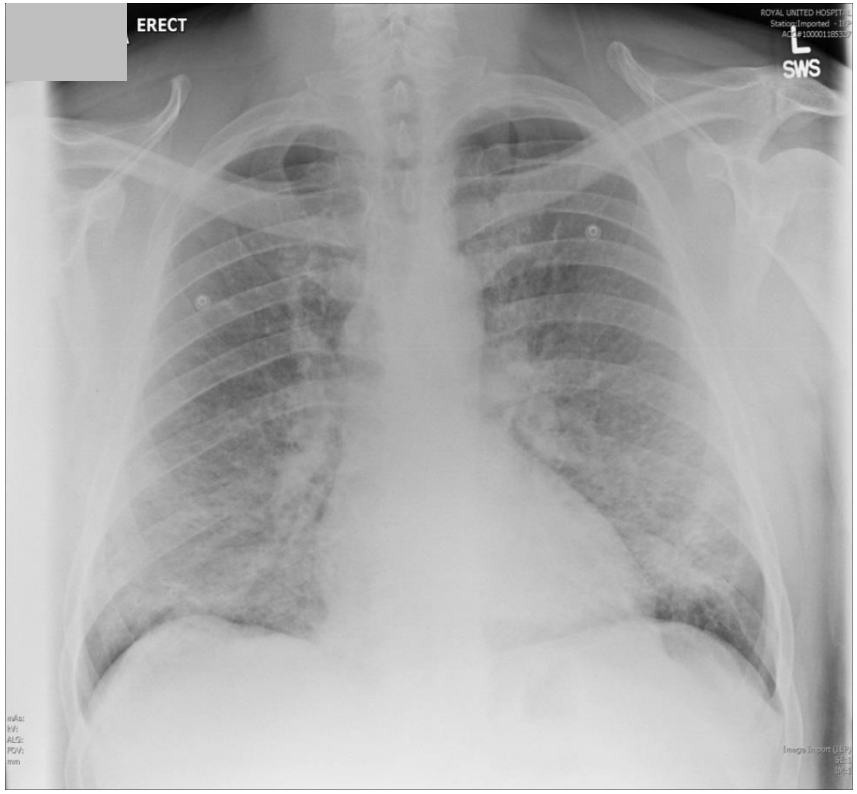
low FEFs, scalloping (ex-smoker)

No reversibility

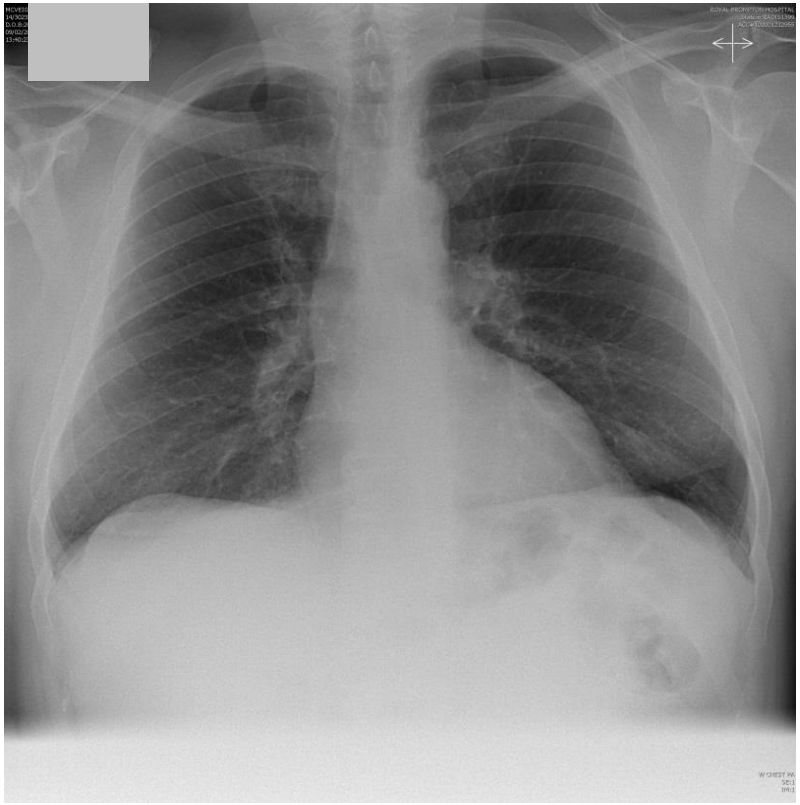




Jan 14



Feb 15



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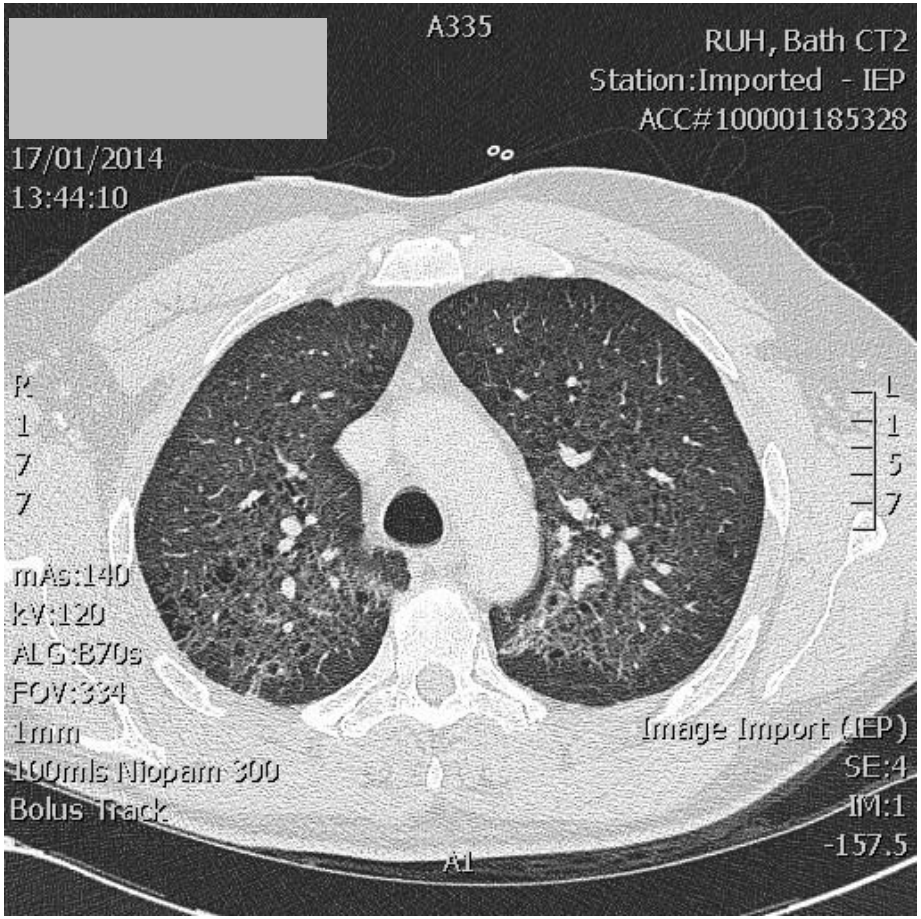
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ACC#100001185328
17/01/2014
13:44:10
mAs:140
kV:120
ALG:B70s
FOV:334
1mm
100mls Niopam 300
Bolus Track
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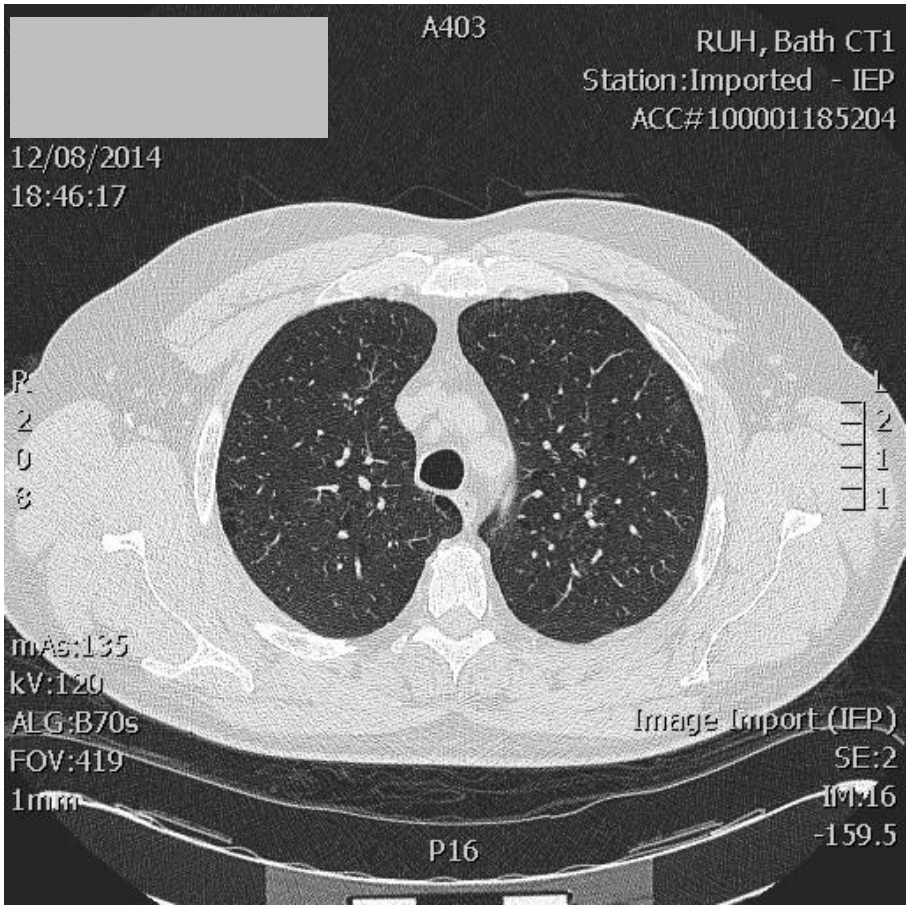
IM:1
-157.5
A1

Aug 14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RUH, Bath CT1
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Image Import (IEP)
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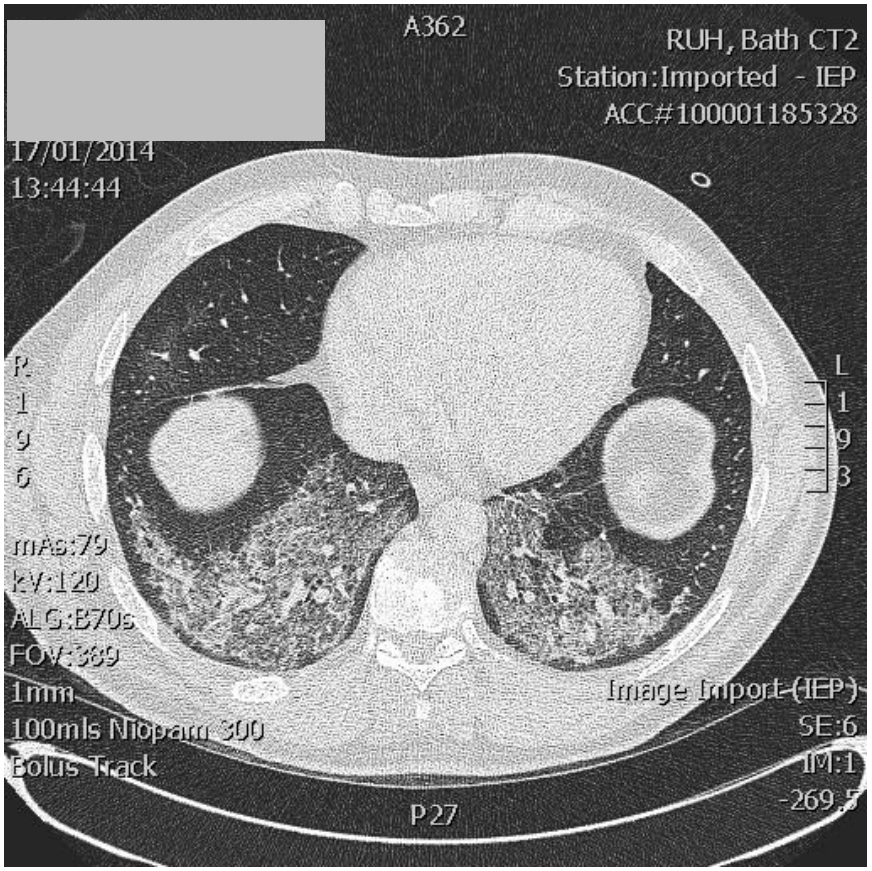
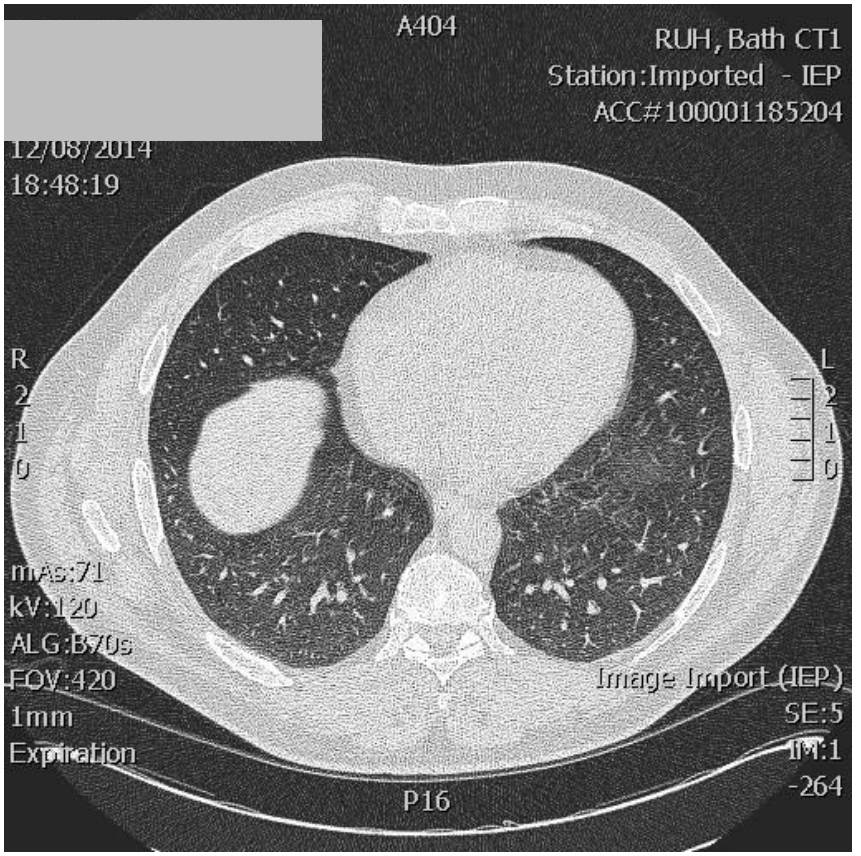
-159.5
P16

Jan 14



Aug 14



Imaging

His imported CT scans show an acute lung injury/diffuse alveolar damage, consistent with inhalation of toxic material

Changes resolved on the August scan and CXR today with no residual fibrosis.

material safety data sheet for Lithofin StainStop:

2.1. Classification of the substance or mixture

Classification according to 67/548/EEC or 1999/45/EC

R10
Xn; R65
R-phrases

10	Flammable.
65	Harmful: may cause lung damage if swallowed.

SECTION 7: Handling and storage

7.1. Precautions for safe handling
Advice on safe handling
While spraying wear respiratory protection.
Use only in thoroughly ventilated areas.
Use solvent-resistant equipment.
Take the usual precautions when handling with chemicals.

General protective measures
Avoid contact with eyes and skin
Do not inhale gases/vapours/aerosols.

8.2. Exposure controls
Respiratory protection
Breathing apparatus in the event of aerosol or mist formation.
Breathing apparatus in the event of high concentrations.
Short term: filter apparatus, filter A
Multi-purpose filter ABEK
(EN 14387, 133, 140, 149)

naphtha (petroleum) “hydrotreated heavy”
....a cause of <u>acute chemical pneumonitis</u> by inhalation

Data sheets inadequate

46F University Administrator

Breathlessness and chest tightness on exertion since pneumonia in 2014
Frequent attacks of 'brittle' asthma; please consider for novel biological therapy?

More history of attacks….

dyspnoea, throat closing, stridor and wheeze, chest tightness
triggers include perfumes (Lynx), strong smells (students), bonfires
"back-to-back" nebulisers and 3-5/7 prednisolone
paramedics every fortnight +/- admission; sickness absence ++
never happened when alone

Symbicort 400/12
Seretide 500
tiotropium 18mcg
salbutamol nebuliser

asthma treatment generally unhelpful

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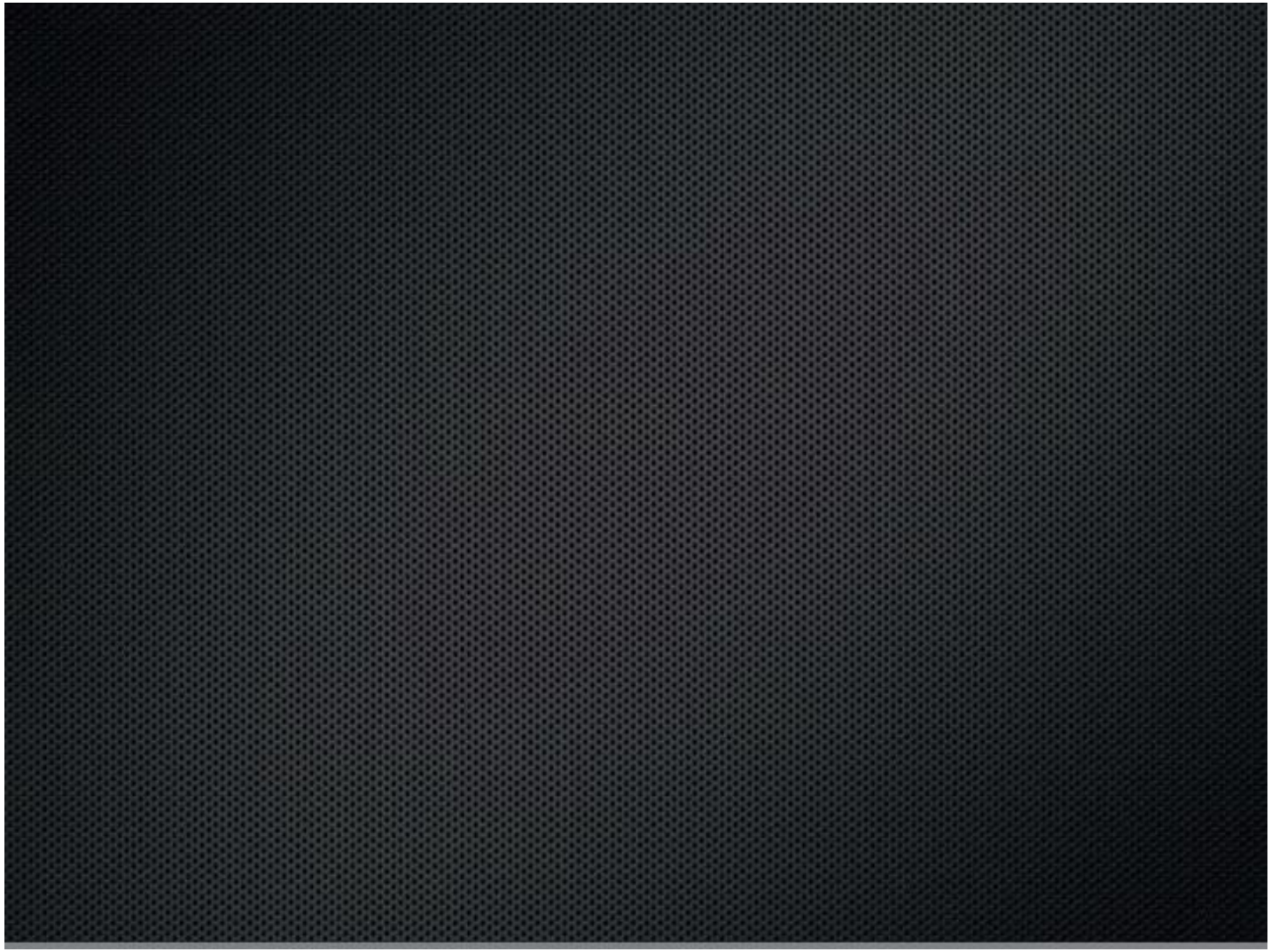
Investigations all normal: CT thorax, PFTs, histamine challenge

Continuous laryngeal endoscopy with provocation; classical appearances



Courtesy of Dr J Hull, RBH

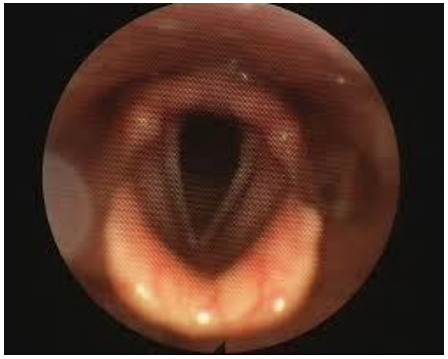
Inducible laryngeal obstruction (ILO): in the occupational setting

symptoms not typical for asthma (occupational or otherwise)

rapid onset of sx with exposure

"dying"; anaphylaxis, allergy, brittle asthma, "RADS"

variable (poor) response to asthma treatment



sickness absence, restricted working areas
significant disruption to general life

increasingly recognised condition
provocation challenge with real-time objective assessment to confirm diagnosis (RBHT)
NB: BPD, hyperventilation syndrome etc.

non-pharmaceutical management; "cure" is possible but support often needed

ERS/ELS/ACCP 2013 international consensus conference nomenclature on ILO. Christensen et al. European Resp Rev 2015 24 445-450

In the clinic and more commonly: "I've not been right since ……"

(1) establish disease, (2) consider attribution, (3) management

History:
a long time may have elapsed since exposure; beware recall bias
what is known and what is unknown
agent, duration of exposure, size of room, ventilation etc.
others affected?

feeling for the psychological impact (PTSD; legal involvement)

new symptoms or exacerbation of pre-existing disease?

Objective tests:
- lung function tests
- measure of BHR
- CT scans
- nasendscopy (ENT; NB timing)
- CPET
- breathing pattern review by specialist physiotherapists

Attribution

Consider: extent of exposure, biological plausibility

rarely possible: absence of comparator information
especially for common (non-specific) outcomes
may be easier in otherwise rare diseases

explore to what extent (and why) does it matter …. and to whom
a focus on attribution ('explanation') may be counterproductive to recovery

'is it safe for this person to return to their normal activities?'
'how can this best be 'managed'?'

unless it's clear, avoid the issue as far as you can
'we'll never really know'
shift the focus to management of the present and future

beware of attributions that you cannot robustly defend
(leave that to others)

recovery doesn't deny attribution

60M, mechanical technician at local hospital

Incident in the plant room of the hydrotherapy pool

Told to add two chemicals together (sodium hypochlorite and sodium bisphosphonate)

Flared up with significant production of fumes

Exposure lasted c.6 minutes

Developed watering eyes, cough with frothy sputum and significant breathlessness.

A&E: mildly low oxygen saturations; CXR & blood tests normal

Gave a statement

Panic symptoms and breathlessness on the tube

A&E (next day): SOB and nausea - nebs and inhalers

2 weeks off work; light duties

Resigned after 7 weeks

OccLD

Chest sx better: just occasional SOB with bleach and spices

Poor energy, blocked right ear, new blisters on lips, pains in wrists and hands
“Withdrawn, confidence issues, poor sleep due to dreams/panic attacks ”

No relevant PMH: salbutamol x6/24h; ex smoker
SH: ex-smoker

Examination: normal
Spirometry: normal
Histamine challenge: normal

Summary

1. He has clearly had a very **unpleasant experience** at work but his results today indicate that there is **no evidence of ongoing asthma** or **permanent damage** to his lungs from his exposure.

2. I would expect his ongoing symptoms of breathlessness to **improve over time** and I have encouraged him to **slowly reduce his inhalers**.

3. He has ongoing symptoms of **anxiety** associated with the event. He is going to contact his GP to see if there is any short term psychological support he can access to help him with practical strategies to overcome these.

4. I have left him with an **open appointment** for this clinic and we would be happy to see him again in the future should we be able to be of help in any way.

26M: BBC journalist working in foreign news

Exposure on 22nd October 2016 to toxic fume in Northern Iraq

2 hours outside in 'cloud'
6 hours inside in a tent with gas seeping through
US army wearing gas masks

Eyes and nose streaming;
Non-productive cough
Dyspnoea ++; chest tightness; wheeze

CXR: "cloudy"
Low oxygen levels
Not fit to fly for a week

Returned to London 28th October 2016
Ongoing chest tightness with cold air, exertion, talking

WORLDPOST 10/22/2016 08:45 am ET | **Updated** Oct 22, 2016

ISIS Sets Sulfur Plant Ablaze In Northern Iraq, Choking The Air With Deadly Chemicals

At least two civilians have died and more than 100 sought medical attention over the attack, a local medical worker said.

n=c.1000 treated

Patrick Osgood
@PatrickOsgood

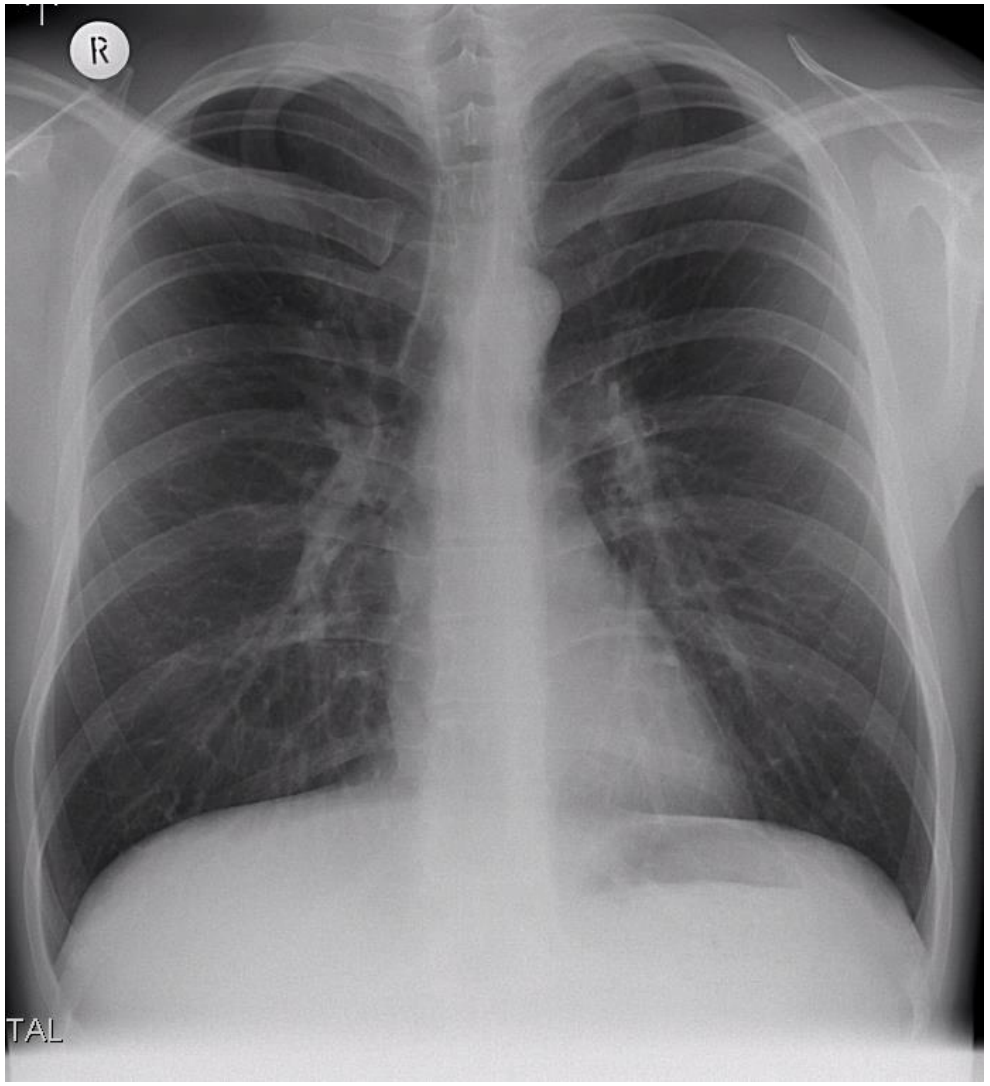
Follow

ISIS torched part of the Mishraq sulphur plant between Qayarah and Mosul this afternoon. Toxic fumes over area now much worse. (image: NASA)

CREDIT: THAIER AL-SUDANI/REUTERS

Smoke rises from a sulfur plant south of Mosul after ISIS militants set it on fire, releasing toxic smoke in the area Friday.

27th February 2017



	27/02/17	**27/02/17 Post BD**
FEV1	4.54 (97%)	5.39 (115%) *
FVC	6.55 (117%)	6.94 (124%)

*19% change but not reproducible

	27/02/17	**27/02/17 Post BD**	**20/03/17**
FEV1	4.54 (97%)	5.39 (115%) *	5.70 (124%)
FVC	6.55 (117%)	6.94 (124%)	7.17 (130%)

Unable to perform histamine challenge (spirometry not reproducible)

Twelve months on……

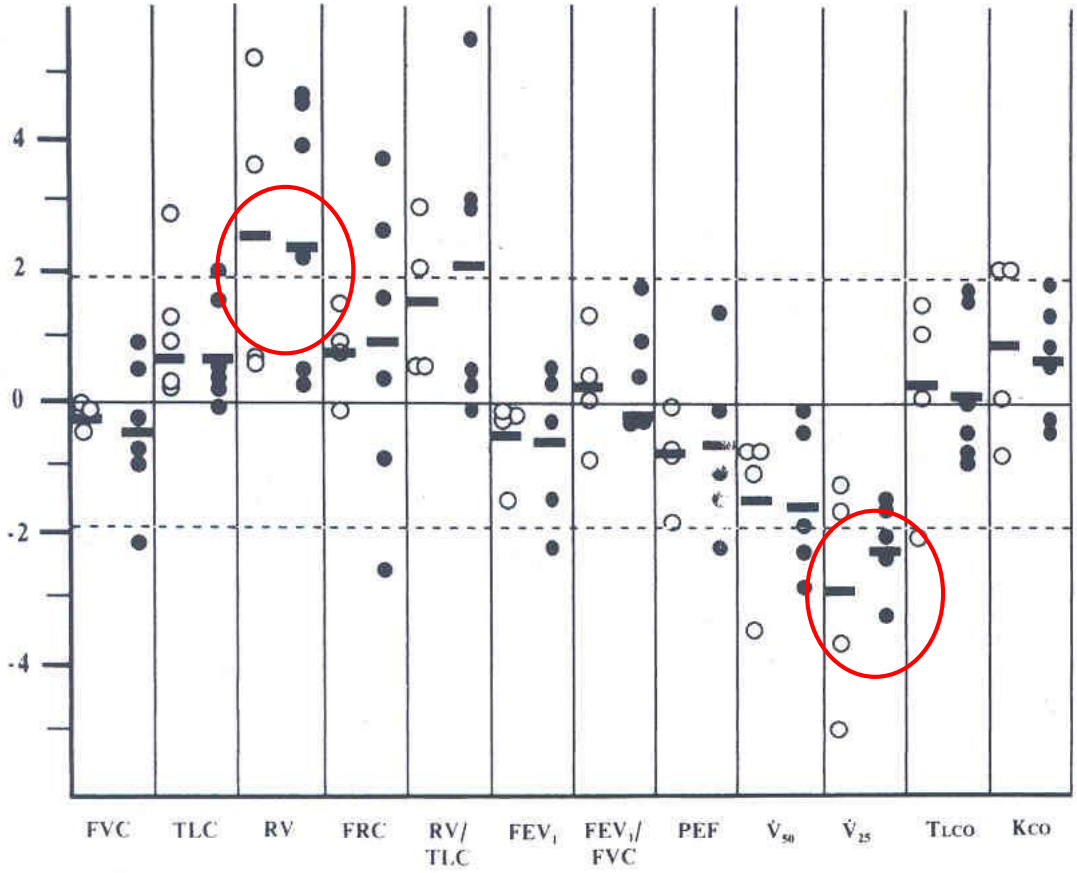
Much better, but breathlessness and “feeling like can’t get air in”
- Middle East
- South Korea terrorist attack simulation
- Grenfell tower fire

Partly relieved by becotide (?)
Still unable to perform reproducible spirometry
Suggestion of a 30% change/ >400mls in FEF25-75

- Reassured++
- Stopped all inhalers
- Physiotherapy
- Intervention for PTSD (EMDR)

Persistent disease after exposure to smoke

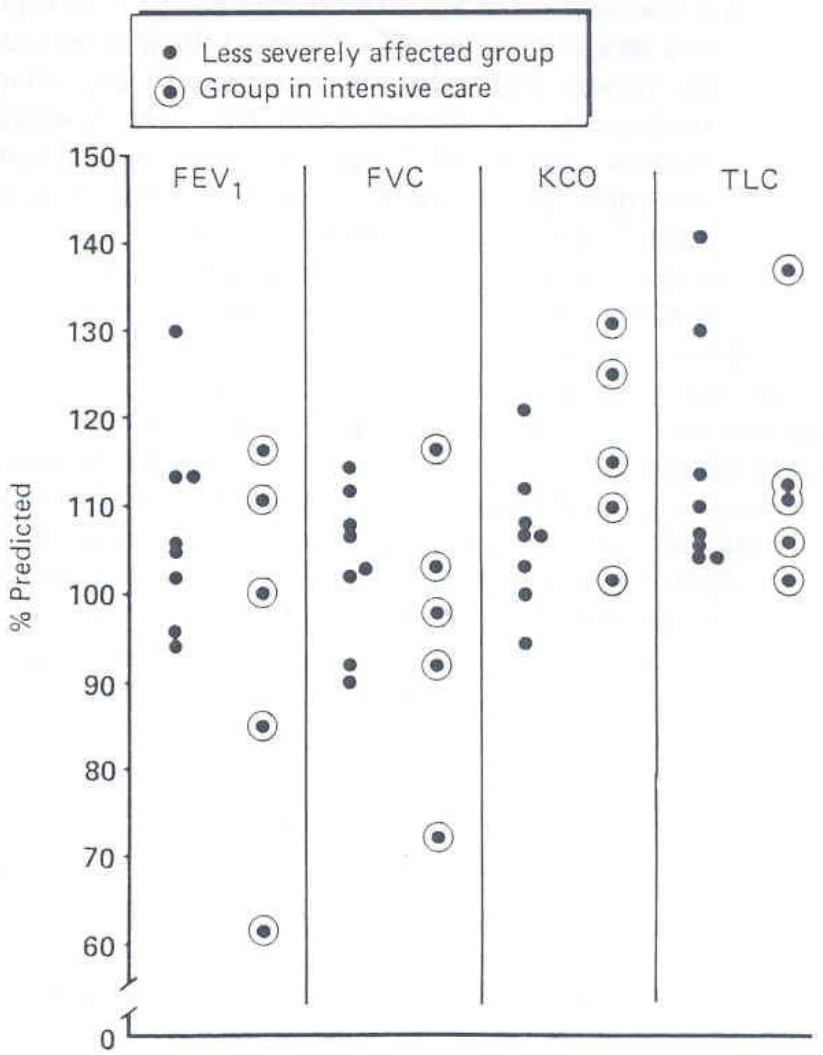
- Kings Cross fire 1987
- 31 fatalities at scene
- 27 injured – 15 had smoke inhalation; one died and 3 ventilated
- 10 survivors fire 1987
- lung function 2 years later



Fogarty. Thorax 1991;46:914-918

? persistent disease after exposure to smoke

- survivors of the Manchester air disaster
- engine explosion Boeing 737 1985
- 137 people on board
- 52 died
- lung function 3 months later (n=13)



"Suggested that long term physical problems associated with exposure to these fumes is rare."

O'Hickey. BMJ 1987;294:1663-1667

Summary

LUNGS AT WORK
www.lungsatwork.org.uk

Solubility of agent useful in predicting effects

Majority with acute inhalation injury have no long-term adverse health outcomes

Minority develop longer-term complications

Management is largely supportive

Consider ILO for those with upper airway symptoms

Irritant induced asthma (“RADS”) is probably over-diagnosed

Use objective tests to evaluate the present or absence of disease

Psychological factors may be important in recovery

Thank you!