David Purser

The assessment of fire hazards, tenability and human evacuation behaviour for fire safety engineering design
Erasmus Mundus Programme

Assessment of physiological and toxic hazards in fires
2: Asphyxiant gases and FED calculations

Prof. David Purser
Hartford Environmental Research,Hatfield UK.

Brandteknik och riskhantering
Lunds Tekniska Högskola, Sverige

Thursday 14th April 2011

David Purser

Toxic smoke products

Asphyxiant gases:

CARBON MONOXIDE

HYDROGEN CYANIDE

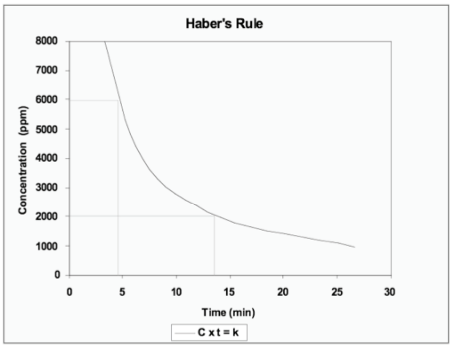
CARBON DIOXIDE

LOW OXYGEN

cause confusion and loss of consciousness followed by death from asphyxia when a sufficient dose has been inhaled

David Purser

Haber's Rule

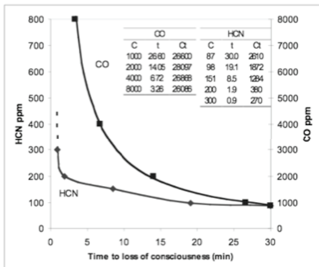


David Purser

Time to incapacitation for HCN and CO exposure

HCN present at 1000 ppm in domestic fires causes rapid incapacitation but dynamics of uptake and dispersal in blood result in low post-exposure blood CN, and CN unstable in blood, whereas COHb very stable

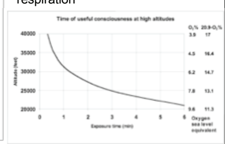
CO is probably the main ultimate cause of death by asphxiants but HCN may be a major cause of incapacitation



Upper layer oxygen concentration can be as low as 1% O_2 - one breath enough to cause collapse.

This effect has been reported in some fire incidents

CO_2 important because it increases the rate of uptake of other gases by stimulating respiration



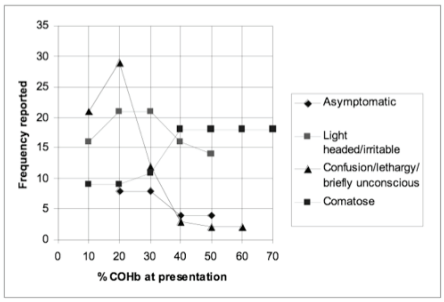
For HCN a small increase in concentration causes a large decrease in time to incapacitation

David Purser

CO symptoms

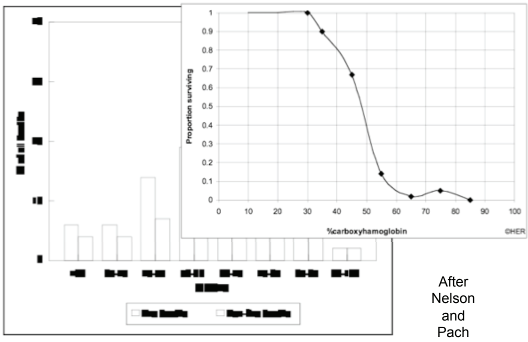
David Purser

CO symptoms clinical



David Purser

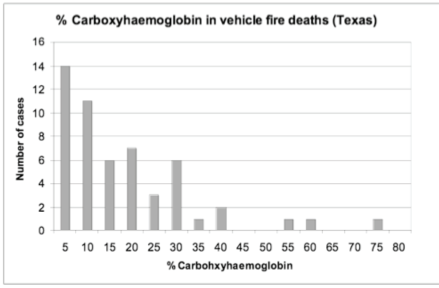
COHb post-mortem fire and non-fire



After Nelson and Pach

David Purser

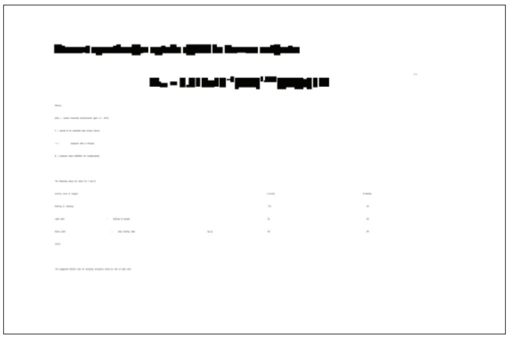
COHb post-mortem fire and non-fire



% carboxyhemoglobin in 53 vehicle fire fatalities from the Texas data for which fire could be identified as contributing to the causes of death (first set 0-5%, second 6-10% etc.)

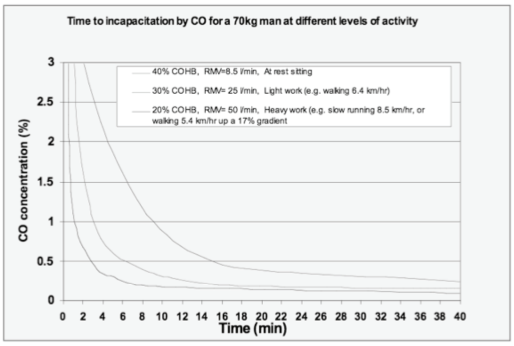
David Purser

Uptake of CO and exercise



David Purser

Uptake of CO and exercise



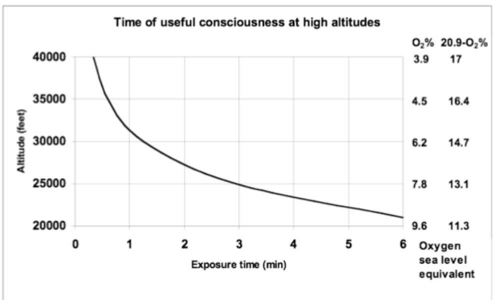
David Purser

Low oxygen hypoxia

- Effects depend upon:
  - Inhaled CO_2 concentration
  - Level of physical activity
  - Health status
- Up to a point can be tolerated due to compensatory mechanisms including:
  - Increased cerebral blood flow
  - More efficient unloading of oxygen from oxyhaemoglobin at low oxygen tensions
- A point is reached when compensatory mechanisms fail and cerebral depression occurs with symptoms consisting of lethargy and impaired consciousness
- This occurs at 10-12% oxygen

David Purser

Low oxygen hypoxia



$$FED_{lo} = t/\exp\,[8.13 - 0.54(20.9 - \%O_2)]$$

David Purser

Hydrogen cyanide

- The main mechanism of poisoning by hydrogen cyanide is prevention of oxygen metabolism in the mitochondrion by inhibition of cytochrome c reaction with oxygen
- This is a form of hypoxia, but in the presence of oxygen excess
- The blood is full of oxygen and bright red, because the oxygen cannot be used
- Cyanide does react with a form of haemoglobin called methaemoglobin to form cyanomethaemoglobin, it also combines weakly with haemoglobin and tends to be concentrated to some extent in red blood cells
- However while more than 80% of CO remains in the blood as COHb, most HCN is rapidly dispersed into the tissues, diffusing freely into the extracellular body fluid compartment and the intracellular fluids.
- Over an hour or so it is detoxified in the liver as well as being excreted from the lungs

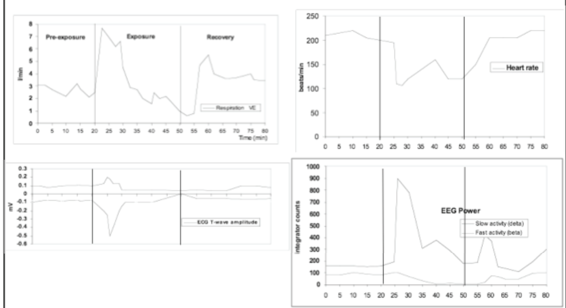
David Purser

Hydrogen cyanide

- Because CO almost all stays in the blood as COHb, and because it is very stable in cadavers and stored blood, carboxyhaemoglobin provides a very good record of the exposure of a victim to CO in a fire. In fact the COHb concentration can be used in combination with fire modelling to estimate the CO concentrations that were present in the fire.
- It is also possible to establish whether a victim had inhaled enough CO to incapacitate or cause death
- This is partly why CO is cited as the cause of death in many fires. Other agents like low oxygen or cyanide do not leave such clearly measurable traces.

David Purser

Physiological effects of HCN gas (147 ppm)



David Purser

Post-mortem blood cyanide

- In addition to the dynamics of volume of distribution, there are post-mortem changes
- The concentration halves in a cadaver over 24 hours
- In store samples of blood in a refrigerator cyanide is reasonably stable, but slowly decreases over a period of weeks and months
- This is due to the formation of thiocyanate
- Freezing or microbial action can reverse this process to some extent causing an apparent increase in concentration in stored samples

David Purser

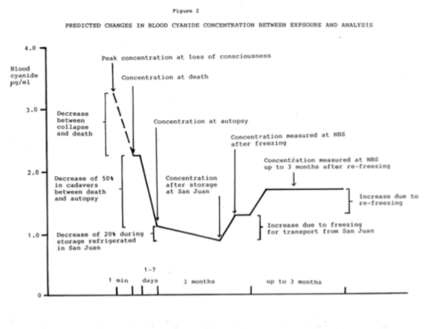
Post-mortem blood cyanide

- In primates loss of consciousness occurred at fresh blood cyanide concentrations of between approximately 2.5 and 3.4 µg/ml.
- In dogs incapacitation occurred at 2.15 µg/ml following i.v. injection of HCN and death at 3.6 µg/ml.
- Curry: blood cyanide concentration of 3.5 µg/ml in a human case at death, and approximately 0.75 µg/ml at autopsy the next day.
- Ballantyne found 2 µg/ml cyanide in blood following death by HCN inhalation in rabbits.
- Levels post-mortem in three fatal human cases of HCN inhalation 5, 2.4 and 1.7 µg/ml. Suggested that 1-2 ug/ml cyanide in whole blood is the lowest level compatible with death from acute cyanide poisoning.
- Hall et al. (13) suggest the following correlation between blood levels and toxic signs:

µg/ml	Signs
0.2-0.5	No signs
0.5-1.0	Flushing, tachycardia
1.0-2.5	Obtunded
2.5-3.0	Coma
3.0	Death

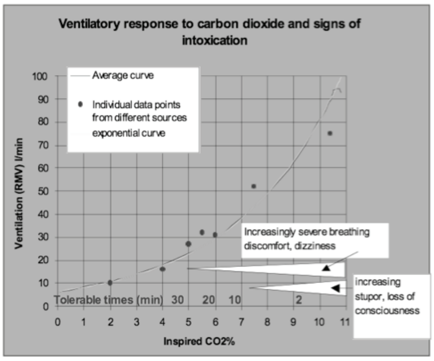
David Purser

Post-mortem changes in blood sample from the Dupont Plaza hotel fire



David Purser

Respiration and metabolism



David Purser

Hazard equations

Smoke: FEC_{smoke} = [OD/m]/0.2 for small enclosures or [OD/m]/0.1 for large enclosures

Irritants sensory:

$FIC = FIC_{HCl} + FIC_{HBr} + FIC_{HF} + FIC_{SO2} + FIC_{NO2} + FIC_{CH2CHO} + FIC_{CH2O} + \sum FIC_x$.

Irritants lethal:

$FLD_{irr} = FLD_{HCl} + FLD_{HBr} + FLD_{HF} + FLD_{SO2} + FLD_{NO2} + FLD_{CH2CHO} + FLD_{HCHO} + \sum FLD_x$

Asphyxiants:

$FED_{IN} = (FED_{Ico} + FED_{Icn} + FLD_{irr}) \times VCO_2 + FED_{Io}$

David Purser

Asphyxiant equations

FEDs for incapacitation for CO, HCN and low oxygen:

$FED_{Ico} = (8.2925 \times 10^{-4} \times \text{ppm } CO^{1.036}) \times t/30$

$FED_{Icn} = (\exp([CN]/43))t/220$

$FED_{Io} = t/\exp[8.13 - 0.54(20.9 - \%O_2)]$

Ventilatory stimulation by CO_2:

$VCO_2 = \exp([CO_2]/5)$

David Purser

FED equations asphyxiants

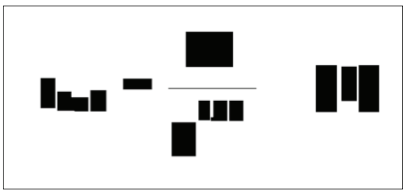
- For asphyxiants the effects of combinations are also approximately additive, but a number of interactions needs to be considered:
- The FED for CO and HCN are considered directly additive as has been demonstrated experimentally
- It is considered that the effects of irritants on lung function will also cause some hypoxia and so an additive term is included consisting of the FLD_{irr}
- All these will be increased according to VCO_2
- Low oxygen hypoxia will be additive with the overall effects, but is not increased by VCO_2 (in fact it is improved)
- The intoxicating effects of CO_2 are considered unlikely to occur before other effects so are normally ignored, but can be considered an “or” term in the equation

Asphyxiants:

$FED_{IN} = (FED_{Ico} + FED_{Icn} + FLD_{irr}) \times VCO_2 + FED_{Io}$

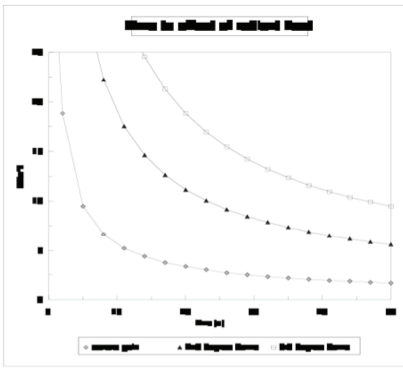
David Purser

Hazards from radiant heat



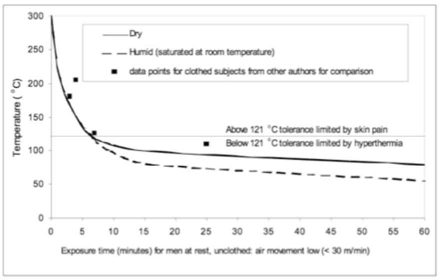
David Purser

Hazards from radiant heat



David Purser

Tolerance time for exposure to convected heat



David Purser

Incapacitation by heat

Time to incapacitation by convected heat

[redacted]

Summing FED for radiant and convected heat

[redacted]

David Purser

Total heat hazards

- Where a subject is exposed to both radiant and convected heat the effects are additive, so that some method for summation is required
- One method is to sum the fractional doses of heat acquired from the two sources to predict time to pain
- The algorithm for convected heat predicts time to pain but not to burns
- It would be useful if the same units could be used for both form of heat – I.e. expressed in terms of total heat flux

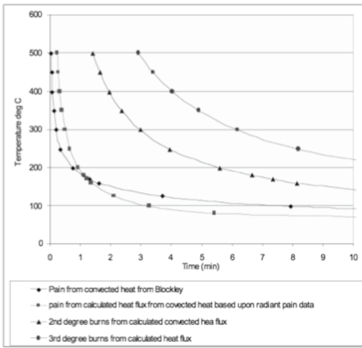
[redacted]

David Purser

Heat algorithms

Predicted times to pain and burns for convected heat comparing Blockley data for convected heat exposures with times calculated from convected heat flux using the radiant heat flux pain and burns data

(h taken as 8)

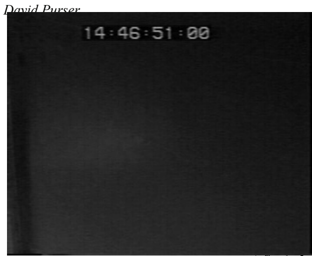


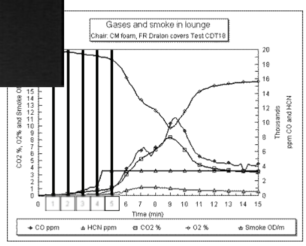
David Purser

Main fires types and hazard scenarios

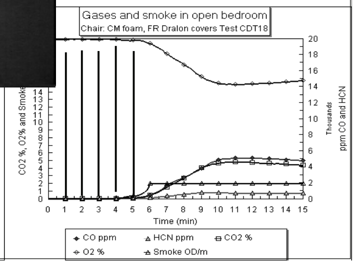
1. Non-flaming/smouldering fires
2. Early/well ventilated flaming fires
3. Ventilation controlled fires
   - •pre-flashover vitiated flaming fires
   - •post-flashover vitiated flaming fires

David Purser





David Purser

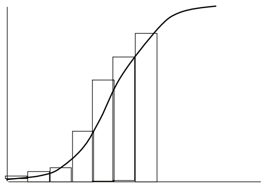


David Purser

Fully furnished house fire

David Purser

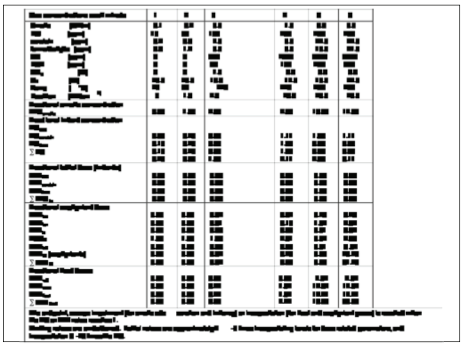
FED in fires



Σ C x Δt/Ct = FED

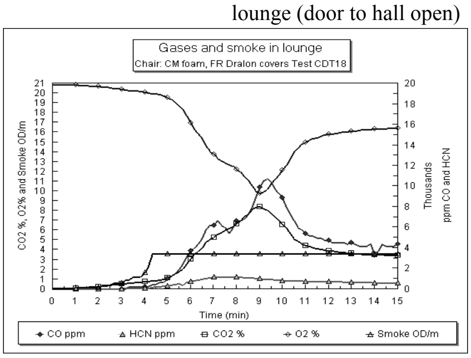
David Purser

FED analysis table for a fire



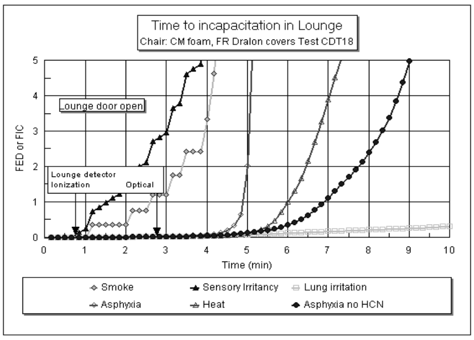
David Purser

Toxic gases and smoke in fire room for an armchair fire in a house lounge (door to hall open)



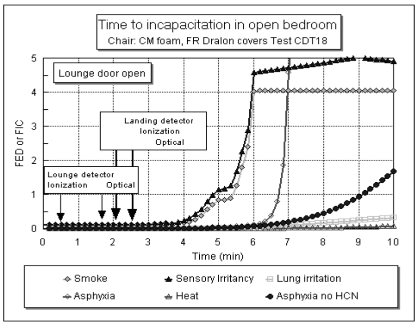
David Purser

Hazards in fire room from an armchair fire in a house lounge



David Purser

Hazards in open bedroom from an armchair fire in a house lounge



David Purser

Toxic effects of gas mixtures

- Fire gases are mixtures of irritants and asphyxiants
- Asphyxiants cause death during exposure due to brain hypoxia
- Irritants cause death after exposure due to lung inflammation
- Toxicity models assume different asphyxiants are basically additive with each other, and different irritants are also basically proportionally additive with each other – which more or less makes sense
- But in terms of overall lethality it is also assumed that all toxic products are basically proportionally additive. In practice it is to be expected that asphyxiants and irritants would act independently
- Rats have been exposed to a wide variety of toxic fire gas effluent mixtures to measure lethal exposure doses.
- By comparing the calculation models to the data it is possible to determine if they different components act additively or not

David Purser

Lethal toxic potency of mixtures in rats

The current version of the N-gas model for total deaths (during and after exposure) is as follows:

$$FED = \frac{m\,[CO]}{[CO_2] - b} + \frac{21-[O_2]}{21-LC_{50}\,O_2} + \left(\frac{[HCN]}{LC_{50}\,HCN} \times \frac{0.4[NO_2]}{LC_{50}\,NO_2}\right) + \frac{0.4[NO_2]}{LC_{50}\,NO_2} + \frac{[HCl]}{LC_{50}\,HCl} + \frac{[HBr]}{LC_{50}\,HBr} \qquad [4]$$

Fractional Effective Dose (FED) =

$$\left(\frac{[CO]}{LC_{50}\,CO} + \frac{[CN] - [NOx]}{LC_{50}\,HCN} + \frac{[\text{each acid gas}]}{LC_{50}\text{ each gas}} + \frac{[\text{each organic irritant}]}{LC_{50}\text{ each organic irritant}}\right) \times V_{CO2} + A + \frac{1}{\text{hypoxia function}} \qquad [5]$$

where:

VCO_2 multiplication factor for CO_2 driven hyperventilation = 1+ (exp (0.14 x $[CO_2]$)-1)/2

A is an acidosis factor = ($[CO_2]$ x 0.05) - 0.02

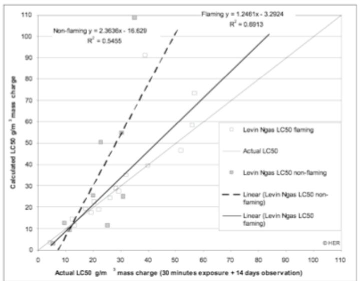
Hypoxia function = exp(8.13-0.54 x $[21-O_2]$)

[CN] represents the concentration of cyanide

[NOx] represents the summed concentration of NO and NO_2

David Purser

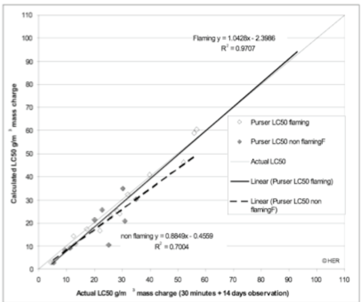
N-gas model against rat data



LC_{50} concentrations calculated using the Levin N-gas model compared with measured rat LC_{50} concentrations for different materials decomposed under non-flaming and flaming combustion conditions

David Purser

Purser LC_{50} model against rat data



LC_{50} concentrations calculated using the Purser LC_{50} model compared with measured rat LC_{50} concentrations for different materials decomposed under non-flaming and flaming combustion conditions.

- Flaming fire effluent mixtures contain mainly asphyxiant gases
- Non-flaming fire effluent mixtures also contain high yields of irritant organic chemical species

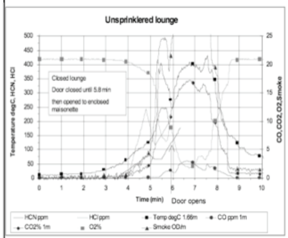
Conclusion:
Additive model is a good predict or of experimental results

David Purser

Cleckheaton domestic sprinkler fires

David Purser

Hazard analysis in sprinklered lounge fire



Sprinkelers triggered

David Purser

Hazard analysis in sprinklered lounge fire