Occupational Health and the Heart

John D Meyer MD MPH
West Virginia University
University of Manchester, UK
Occupational Health and the Heart

Two facets

Workplace exposures and their effects on the heart
- Primary causes of disease
- Exacerbations of underlying disease
- Attribution and workers’ compensation

Cardiovascular health and its effects on work
- Work capacity and abilities
- Workplace as focus for prevention efforts
Problems in identification of occupational etiologies of CVD

- Common in Western society:
  Increased risks superimposed on high baseline

- Multifactorial etiology:
  Work contributions difficult to tease out

- Long latency

- No accurate noninvasive tests for early disease

- Clinical expressions are similar whether the disease has an occupational or non-occupational cause
Agent and work effects on the heart

Agents can be grouped by main or major effects:

Angina
Atherogenesis
Dysrhythmias
Cardiomyopathy
Hypertension
Angina - Carbon monoxide

Sources of incomplete combustion: Furnaces, boilers
Internal combustion engine (warehouses, auto plants)
Hazards increased in cold weather with closed doors and windows
Carbon monoxide → carboxyhemoglobin

- Binds to hemoglobin more avidly than O₂ (CO has 200x oxygen’s affinity)
- Shifts oxygen dissociation curve to “left”: Tissue anoxia the result
- Binds mitochondrial enzymes and myoglobin
- Increases platelet stickiness
- Decreases arrhythmia threshold
CO and hemoglobin oxygen-dissociation curve
Reduced oxygen-carrying capacity of carboxyhemoglobin at high CO levels
Angina: Carbon monoxide

CarboxyHgb levels and symptoms:

- Cardiac compensatory effects seen at carboxyHgb levels 8 -10%: HA, lightheadedness, some chest pain
- EKG disturbances (extrasystoles, PVCs, atrial fibrillation) at higher levels (10-25%)

Dependent on previous cardiac status and susceptibility:

- Cigarette smokers chronically at ~5%
- Individuals with pre-existing CAD may develop angina with moderate activity at carboxyHgb levels as low as 3 - 5%
Carbon monoxide: Exposure limits

NIOSH REL: 35 ppm for 10-hour TWA
Equivalent to 5% COHgb level
Uptake will increase with physical exertion: Exposure should be correspondingly limited in jobs with high physical demands

OSHA STANDARD: 50 ppm /TWA

ACGIH: TLV®: 25 ppm/ TWA
BEI®: 3.5% COHgb
More protective of sensitive groups. BEI may be useful in documentation of significant exposure.
Carbon monoxide

Methylene Chloride $\text{CH}_2\text{Cl}_2$

- Solvent: degreasing, paint stripping
- Absorption through respiratory route or through skin
- Metabolized in bloodstream to CO
Methylene Chloride

May elevate carboxyhemoglobin to 10% or more especially in poorly ventilated space

Probably not significant to healthy person; may become mildly symptomatic

Cigarette smokers, those with angina or current CHD a concern: excess CO may trigger symptoms
Methylene Chloride

OSHA Standard: 25 ppm/ TWA8: STEL 125 ppm

NIOSH: As low as can be achieved (carcinogen)

Because of metabolic conversion to CO, the biological life of COHgb from methylene chloride is longer than that from direct CO exposure
Chronic exposure to CO associated with cardiovascular mortality:
NYC bridge and tunnel officers
Carbon monoxide: Long-term exposure effects

SMRs for death from cardiovascular disease of bridge (low-CO-exposure) and tunnel (high) officers in NYC:

<table>
<thead>
<tr>
<th>Duration of Employment</th>
<th>Bridge Officers</th>
<th>Tunnel Officers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;10 years</td>
<td>≥10 years</td>
</tr>
<tr>
<td>Bridge Officers</td>
<td>0.87 (0.70-1.07)</td>
<td>0.81 (0.56-1.15)</td>
</tr>
<tr>
<td>Tunnel Officers</td>
<td>1.07 (0.77-1.44)</td>
<td>1.88 (1.36-2.56)</td>
</tr>
</tbody>
</table>

Angina: Nitrates

Noted to have vasodilatory effects in explosives workers.

Tolerance to absorbed nitrate symptoms (headaches, tachycardia, diastolic HTN) develops quickly.
Dynamite and other explosives manufacture

Ethylene glycol

Glycerin

Nitrator

Liquid NTG / EGDN storage and supply

“Dope”

Dynamite Mix House

Dynamite Hand-Packing House

Machine Packing (Cartridge-filling) Houses

Dynamite Case Houses

Magazine and Shipping

Ethylene glycol dinitrate

H₂C-O-NO₂

H₂C-O-NO₂

H₂C-O-NO₂

nitroglycerin

ethylene glycol dinitrate
Acute effects in workers noted in early 1960s:

Sudden death:
- 24-96 hours after exposure ceased (weekends/holidays)

“Monday Morning Angina”:
- Relieved by RTW, nitrate meds: coronary spasm in absence of CAD

Three-fold increase in acute deaths in younger men from ischemic CHD
Angina: Nitrates

- Mechanism of acute effects not clear: Rebound vasospasm vs. arrhythmias (VF) triggered by re-exposure

- CAD risk increased 2-3x after 20 years exposure: persists after removal

- Possible HTN after cessation of exposure
Atherogenesis

Carbon disulfide (CS$_2$)

- Cellulose-derived materials
  - Rayon
  - Cellophane
- Solvent for rubber, oils
- Pesticides
- Fumigant for grain, books
- Microelectronics industry
Viscose process for Rayon manufacture

Wood Flakes → Raw Cellulose → Cellulose Xanthate → Filtered Viscose → "Ripening" → Spinning → Rayon Filaments

Chemical Reagents:
- Lye
- Zn
- H$_2$SO$_4$
- CS$_2$
- H$_2$SO$_4$
Cellulose flakes after lye treatment

Viscose emerging from spinneret. $\text{CS}_2$ is given off when viscose cross-links to form rayon.
Deaths among operatives and staff aged 45-64 with > 10 yr employment in rayon factories

<table>
<thead>
<tr>
<th>Occupation</th>
<th>P-Y at risk</th>
<th>Coronary Heart Disease</th>
<th>Other CV Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Obs</td>
<td>Exp</td>
</tr>
<tr>
<td>Operatives</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Viscose Making</td>
<td>2221</td>
<td>5</td>
<td>7.2</td>
</tr>
<tr>
<td>Viscose Spinning</td>
<td>4585</td>
<td>28*</td>
<td>14.6</td>
</tr>
<tr>
<td>Non-process</td>
<td>1997</td>
<td>6</td>
<td>8.0</td>
</tr>
<tr>
<td>Staff</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spinning</td>
<td>1502</td>
<td>9**</td>
<td>4.3</td>
</tr>
<tr>
<td>Non-process</td>
<td>752</td>
<td>3</td>
<td>2.3</td>
</tr>
</tbody>
</table>

* $\chi^2 = 12.2$ p<0.001
** $\chi^2 = 5.2$ p<0.05

Carbon Disulfide and Atherogenesis

RR of 2 to 5x for death from CAD

Epidemiologic evidence suggests a direct role in atherogenesis in blood vessels:

Enzyme inhibition by metabolites of CS$_2$

- React with amino acids to form dithiocarbamates: these chelate trace metals and react with enzyme cofactors
- May interfere to increase elastase activity, disrupting blood vessel walls
- May decrease fibrinolytic activity and enhance thrombosis
Japanese CS$_2$ workers:
Retinal microaneurysms
Japanese CS$_2$ workers:
Retinal hemorrhages
Carbon Disulfide

OSHA Standard:  20 ppm TWA
MAC:  100 ppm/30 minutes

NIOSH REL:  1 ppm TWA
STEL:  15 ppm/15 minutes

ACGIH BEI®:  Urine TTCA:  5mg/g creatinine
End of workshift urine sample.
Dysrhythmias

- Chlorofluorocarbons (Freon® etc)
  - Refrigeration, air conditioning, propellants
  - May sensitize myocardium to catechol effects

- Other solvents implicated in sudden death:
  - Trichloroethylene, toluene, benzene

- Findings at autopsy usually unremarkable: c/w sudden death from arrhythmias
Cardiomyopathy

- Cobalt: used to stabilize beer foam (1960’s: Canada, Belgium)
- Cardiomyopathy reported in beer drinkers several months afterward
Cardiomyopathy: Cobalt

Dose-related: seen in heavy drinkers
greatest risk in those drinking $\geq 10$L/day (!)
22 - 50% mortality in some series

Why this group?

- CM not seen in cobalt therapy for anemia
Probable synergistic effect with alcohol, poor diet
Hypertension

Associations with several occupational exposures and agents

Mechanisms are varied and depend on action of agent
Hypertension

Lead

- Probable mechanism is via renal injury
- May also increase vascular tone and resistance
- Chelation may improve HTN in acute Pb intoxication, but will not reverse if longstanding renal damage is present

Cadmium possibly associated with HTN; noted to occur at levels below nephrotoxic dose
Hypertension

Carbon disulfide
- Vascular nephropathy and accelerated atherogenesis appear to be mechanisms

Noise, shiftwork
- Postulated effects mediated by stress response (increase sympathetic and hormonal mediator release)
Job Strain and Cardiovascular Disease

Body of evidence suggests relationship between job strain and cardiovascular mortality.

Main associations are with exposure to high psychological demands and low control over job.

Professional drivers (especially urban transport) have the most consistent evidence of increased risk.
Pioneering work of Marmot showed increased CHD mortality related to social status.

Unskilled manual workers (Class V) have considerably increased risk when compared with professionals (Class I)
Cardiovascular effects on work

Some figures on heart disease in US:

- 1.5 million MI each year
- Nearly 200,000 CABG per year
- Over 80% of workers are generally able to return to work after initial MI or CABG
Cardiovascular effects: Return-to-Work after MI

Medical Factors
Major predictors of RTW:
• LV dysfunction
• persistent ischemia / angina after treatment

Non-Medical Factors
• Coping styles
• Perception of work (demands, satisfaction)
• Age, gender, education
• Benefits/incentives
Cardiovascular effects of work

- Reinfarction and death NOT more frequent at work
- Many workers older (>50) and have moved into sedentary roles even pre-infarction

Longshoremen study: Lowest rates of CAD mortality linked to heaviest jobs:

<table>
<thead>
<tr>
<th>Activity level</th>
<th>CV Mortality</th>
<th>Sudden Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>26.9</td>
<td>5.6</td>
</tr>
<tr>
<td>Medium</td>
<td>46.3</td>
<td>19.9</td>
</tr>
<tr>
<td>Low</td>
<td>49.0</td>
<td>15.7</td>
</tr>
</tbody>
</table>
Assessing work capacity / capabilities

History:
  Review of prior and current symptoms
    • CP, dyspnea, orthopnea, etc
  Evidence of improvement with treatment? Descriptions of exertional tolerance (routine activities, work simulations)
  Current medicines

Physical:
  Signs on examination
    • arrhythmias
    • JVD
    • edema
    • chest exam

Review previous and current records
Assessing work capacity / capabilities

- **Exercise EKG**
  - May help refine judgement about RTW, in jobs requiring high exertion
  - Ability to reach Bruce Stage 4 on treadmill (12 minutes; ~8-9 METs) indicates low risk of subsequent cardiac event
  - Most individuals after single uncomplicated MI can generate 8+ METs before fatigue or discomfort
Exercise EKG

- Better for assessing isotonic exercise/work (walking, running etc)
  - Results in ↑ cardiac output, BP remains stable through ↓ peripheral vascular resistance,

- Exercise testing may not yield good estimate of capabilities for isometric work (lifting, static exertion)
  - BP elevates without reduction in PVR
Assessing work capacity: Some numbers

3.5 METs: Bartending, frequent walking with 10lb objects (many office jobs)

4 - 5 METs: Painting, masonry work, light carpentry

5 - 6 METs: Lighter digging, shoveling

6 - 7 METs: Heavier or more frequent shoveling

7 - 8 METs: Carrying 50-60 lbs; sawing hardwood
Assessing work capacity /capabilities

- **Job description:**
  - Always request
  - Assess *static* vs *dynamic* work
  - Other stressors (temperature, psych)
  - Other exposures (CO, cigarette smoke)

- Simulated work (+/- exercise EKG) may be better in judgment of capabilities than testing in lab setting

- Specialist opinion: but beware of conservatism
Work capacity: Some guidance

- **Average** energy demands of job can safely be \( \leq 40\% \) of peak workload

- **Peak** energy demands of job should be \(< \) maximum workload achieved on testing

Thus individual generating 8+ METs can be reasonably asked to work at light-medium physical demand level
Consider in the disabled individual:

- Inadequate treatment
- Depression
- Whether accommodation or changing non-essential requirements of job will allow return
- Socio-economic explanations

Over half of post-CABG patients considered “totally disabled” could have safely performed their normal duties or equivalent work, based on exercise testing results.

What about exercise-testing of asymptomatic workers??

- Predictive value of positive test is low in younger asymptomatic individuals: High false-positive rate requires additional work-up in many cases

- May have better predictive value in > 40yo with other risk factors (smoking, obesity, +FH, hypercholesterolemia, etc)
Fitness-for-Duty Evaluations

Many safety-sensitive jobs (fire, police) have qualification requirements based on exercise testing or physical fitness standards.

Principles outlined in last slide apply: predictive value may be low in younger/healthier workers.

Be careful not to exclude asymptomatic workers on basis of positive exercise test only.

ADA conflicts: May not be limited in performance of job.
Other issues in job assessment

Statutory / Regulatory

- Dept. of Transportation (DOT)
  - Commercial Driver’s License (CDL) exams
    - Exclusionary criteria: “Current” CAD accompanied (or likely to be) by angina, syncope, collapse or congestive heart failure
- Federal Aviation Administration (FAA) more stringent

ADA

- Accommodations
- Direct threat
Attribution and Workers’ Compensation

- Heart disease multifactorial: risk from work exposures is superimposed on a high baseline

- Firefighters, Police: Often a statutory presumption that CAD arose from work, if worker has required years of service
Acknowledgments

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