

*OCCUPATIONAL
CARDIOVASCULAR
DISEASE*

Leading causes of death in Iran



- **Cardiovascular diseases % 45**
- **Accidents % 17.5**
- **Cancers %14**
- **Neonatal disease %6**
- **Respiratory disease %6**

Problems in identification of occupational etiologies of CVD

- Common in society
- Multifactorial etiology
- Long latency
- No accurate noninvasive tests for early disease
- Clinical expressions are similar whether the disease has an occupational or non-occupational cause

Cardiovascular risk factors

Modifiable risk factors

- **Hypertension**
- **Smoking**
- **Hypercholesterolemia**
- **Diabetes Mellitus**
- **Overweight & Obesity**
- **Physical Inactivity**
- **Nutritional habits**

Non – Modifiable risk factors

- **Family history**
- **Increasing age**
- **Male sex**

OCCUPATIONAL TOXICOLOGY

- Cardiac arrhythmia
- Coronary artery disease
- Hypertension
- Non atheromatous ischemic heart disease
- Myocardial injury
- Peripheral arterial occlusive disease
- Arsenic, CFC, Solvents
- CS₂ , CO, Lead
- Cadmium,CS₂, Lead
- Organic nitrate, ethylene glycol dinitrate
- Antimony , Arsine , Cobalt , Arsenic , Lead
- Arsenic , Lead

OCCUPATIONAL HEART DISEASE

- **CARBON MONOXIDE(CO)**
- **CARBON DISULFIDE(CS₂)**
- **NITRATES**
- **SOLVANTS**
- **HEAVY METALS**

Carbon monoxide (CO)

Sources of incomplete combustion:

Furnaces, boilers

Internal combustion engine
(warehouses, auto plants)

Hazards increased in cold weather
with closed doors and windows



Carbon monoxide Acute Poisoning

- Binds to hemoglobin more avidly than O₂ (CO has 200x oxygen's affinity)
- Shifts oxygen dissociation curve to “left”: Tissue anoxia the result



CARBON MONOXIDE(CO)

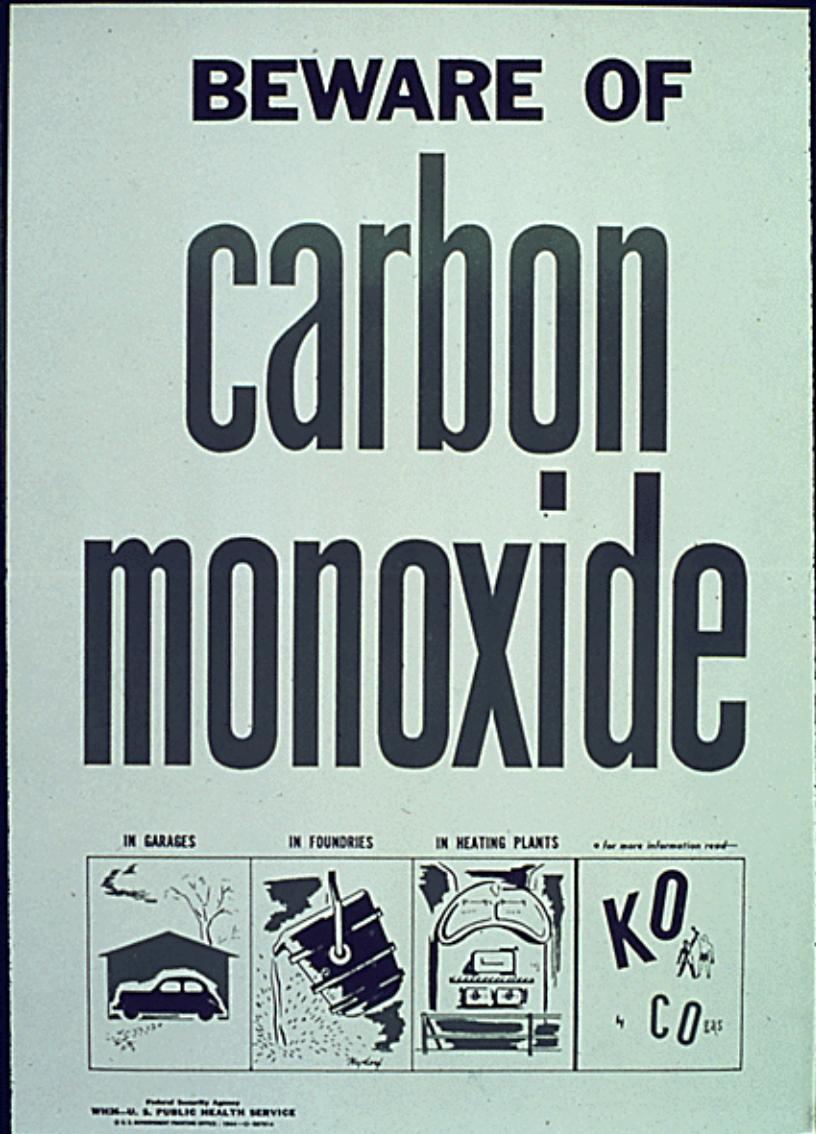
Chronic Exposure

Chronic exposure
to CO associated
with
cardiovascular
mortality



CARBON MONOXIDE(CO)

- Binds mitochondrial enzymes and myoglobin
- Increases platelet stickiness
- Decreases arrhythmia threshold



Carbon disulfide (CS₂)



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

Carbon Disulfide and Atherogenesis

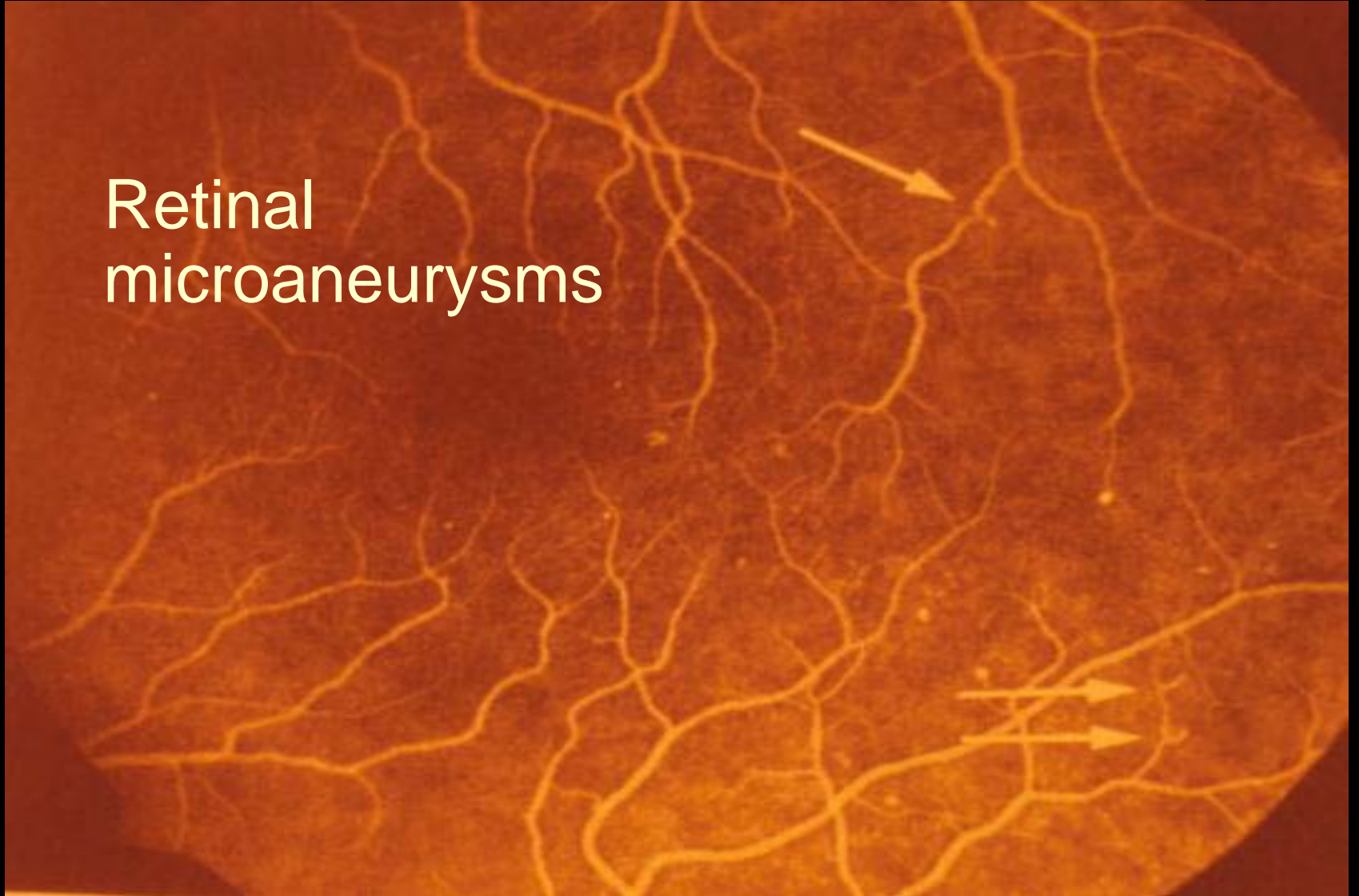


RR of 2 to 5x for death from
CAD

Epidemiologic evidence suggests a direct
role in atherogenesis in blood vessels

CS₂

**Retinal
microaneurysms**



CS₂

**Retinal
hemorrhages**



Angina: Nitrates



- Noted to have vasodilatory effects in explosives workers
- Tolerance to absorbed nitrate symptoms (headaches, tachycardia, diastolic HTN) develops quickly

NITRATES

Acute effects in workers

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

Dysrhythmias



- **Chlorofluorocarbons** (Freon[®] etc)
 - ✓ Refrigeration, air conditioning, propellants
 - ✓ May sensitize myocardium to catechol effects
- Other **solvents** implicated in sudden death:
 - ✓ Trichloroethylene, toluene, benzene

Cardiomyopathy



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

OCCUPATIONAL HEART DISEASE



- **NOISE**
- **HOT & COLD**
- **VIBRATION**
- **PSYCHOSOCIAL FACTORS**
- **PHYSICAL INACTIVITY**

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, Shift work

- 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





Return-to-Work
After MI,
CABG ,PTCA

Patients impact of being out of work

- **Have reduced confidence and self esteem**
- **Have increased morbidity and mortality - particularly mental health**
- **Have disability greater than underlying impairment**

Return-to-Work after MI

- **Over 80% of workers are generally able to return to work after initial MI or CABG**
- **Reinfarction and death NOT more frequent at work**

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



Any Question?