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INDUSTRIAL HYGIENE AND TOXICOLOGY

In Two Volumes

VOLUME I

INDUSTRIAL HYGIENE AND TOXICOLOGY

FRANK A. PATTY, *Editor*

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CONTENTS
Volume I

Preface v
List of Contributors xi
Table of Atomic Weights xiii
Conversion Table for Gases and Vapors xiv
Table of Useful Equivalents and Conversion Factors xvi

I. Industrial Hygiene—Retrospect and Prospect. By FRANK A. PATTY 3
I. Historical Résumé 3
A. Medical and Industrial Hygiene Literature 3
B. Labor Legislation 5
C. Significant Events in the Progress of Industrial Hygiene in the United States 7
II. World War II and Industrial Hygiene Service 10
A. Official Agencies 10
B. Insurance and Industrial Groups 10
C. Educational Institutions 11
D. Research Organizations 12
III. Industrial Hygiene in Foreign Countries 12
A. England 12
B. Russia 13
C. Germany 14
D. Other Countries 14
IV. Industrial Hygiene in the United States 15
V. Prospective Roles of Industrial Hygiene 15

II. Industrial Hygiene Records and Reports. By JOHN B. LITTLEFIELD 19
I. Introduction 19
II. Records of the Environment 19
III. Records of Biological Specimens 22
IV. Interdependence of Medical and Industrial Hygiene Records 23
V. The Nurse's Records 26
VI. Safety Records 26
VII. Personnel Records 26
VIII. Compensation Claim Records 27
IX. Industrial Hygiene Reports 27

III. The Industrial Hygiene Survey and Personnel. By FRANK A. PATTY 29
I. Introduction 2
II. Types of Surveys 3
A. The Inspection Survey 3
B. The Preliminary Industrial Hygiene Survey 31
C. The Investigational Industrial Hygiene Survey 32
1. Surveying the Plant 32
2. Developing Control of Harmful Situations 36
3. Records and Reports 38
D. The Combined Industrial Hygiene and Medical Survey 39

III. The Industrial Hygiene Unit ... 40
IV. Qualifications and Training of Personnel ... 41
A. Administrative Industrial Hygienist ... 41
B. Industrial Hygienist ... 42
C. Other Personnel ... 42

IV. Personal Factors in Competence and Fatigue. By JOSEF BROZEK ... 45
I. General Principles ... 45
II. Problems of Selection ... 48
A. Physical Factors ... 49
B. Psychological Factors ... 52
1. Interview ... 54
2. Tests ... 55
C. Limitations—Physical Handicaps ... 62
D. Limitations—Old Age ... 65
III. Problems of Fatigue ... 69
A. Approach to the Study of Fatigue ... 69
1. Subjective Reports ... 69
2. Measured Functional Changes ... 70
3. Industrial Output Records ... 71
B. Types of Fatigue ... 72
1. Exhaustion-Fatigue ... 72
2. Tiredness-Fatigue ... 73
3. Boredom-Fatigue ... 74
C. Reduction of Fatigue ... 74
1. Occupational Fitness ... 75
2. Motion Economy ... 76
3. Time Relationships ... 77
4. Between-Meal Feeding ... 80
5. Music in Industry ... 83
IV. Problems of Maintenance ... 88
A. Nutrition ... 88
1. Caloric Requirements ... 88
2. Special Dietary Requirements ... 88
3. In-Plant Feeding ... 91
B. Personal Adjustment ... 91
C. Morale ... 95
V. Comment ... 103

V. Environmental Factors in Fatigue and Competence. By W. N. WITHERIDGE 105
I. Air Conditioning ... 107
A. Comfort and Efficiency ... 107
B. Physiological Response ... 107
C. Effective Temperature Index ... 108
D. Temperature Differentials in Hot Weather ... 112
E. Excessive Heat in Industry ... 112
F. Loss of Salt from the Body ... 113
G. Air-Conditioned Crane Cabs ... 114
H. Low Temperature ... 114
I. High and Low Humidity ... 115
J. Air Movement and Drafts ... 115
K. Acclimatization in Air Conditioning ... 116

L. Psychology of Air Conditioning ... 117
M. Air-Conditioning Complaints ... 117
N. Measurement of Air Conditions ... 118
II. Light Conditioning ... 119
A. Requirements for Comfortable and Efficient Seeing ... 120
B. Natural Illumination or Daylighting ... 124
C. Artificial Illumination ... 125
D. Measurement of Illumination ... 126
III. Sound Conditioning ... 127
A. Effect of Noise on Human Beings ... 127
B. Prevention of Noise and Vibration ... 128
C. Measurement and Analysis of Noise ... 129
IV. Sanitary Conditioning ... 130

VI. Physiological Effects of Abnormal Atmospheric Pressure. By HEINZ SPECHT 135
I. Introduction—Historical Background ... 135
II. Properties of the Atmosphere ... 137
A. Composition ... 137
B. Physical Attributes of Atmospheric Gases, and Fundamental Physiological Aspects ... 138
1. Mass and Weight ... 138
2. Density ... 138
3. Compressibility ... 138
4. Partial Pressures ... 142
5. Solubility or Absorption ... 142
6. Temperature of the Atmosphere, Its Origin and Variation ... 145
C. Chemical Activity ... 146
1. Oxygen and Carbon Dioxide ... 147
2. "Inert" Gases ... 148
III. Effects of Increased Atmospheric Pressure on the Body ... 149
A. Charging of the Body with Gases ... 149
1. Mechanical Effects ... 149
2. Solution of Gases in the Body ... 151
3. Rate of Change of Pressure ... 152
B. Effects of Maintained Positive Atmospheric Pressure ... 152
1. State of Saturation of the Several Body Tissues ... 152
2. Effects on the Circulation ... 153
3. Oxygen Poisoning ... 154
4. Inert Gas Effects ... 154
5. Effects of Temperature and Humidity ... 155
6. Other Effects and Subjective Responses ... 155
C. Discharge of Gases from the Body Following Exposure to Increased Atmospheric Pressure: Decompression ... 156
1. Mechanical Effects ... 156
2. Bubble Formation ... 157
3. Rate of Decompression ... 159
IV. Effects of Reduced Atmospheric Pressure on the Body ... 161
A. Discharge of Gases from the Body: Decompression ... 161
1. Rate of Ascent ... 161
2. Evolution of Gases from Solution in Body Tissues during Ascent ... 163
3. Mechanical Effects ... 164

B. The Effects of Maintained Low Atmospheric Pressure 165
1. Hypoxia 165
2. Hypocapnia 166
3. Water Vapor of the Lungs 167
4. Critical Altitudes 168
5. Decompression Sickness 168
6. Acclimatization 171
C. Charging of the Body with Gas in Descent: Recompression 171
1. Re-solution of Gases. Relief of Decompression Sickness 171
2. Mechanical Effects of Recompression 172
3. Rate of Recompression 172
D. Comparison of Effects of High and Low Atmospheric Pressure 173

VII. The Mode of Entry and Action of Toxic Materials. By FRANK A. PATTY ... 175
I. Classification of Contaminants 175
A. Physical Classifications 175
1. Gases and Vapors 175
2. Particulate Matter—Dispersoids 176
3. Particulate Matter—Condensoids 176
4. Other Physical Classifications and Definitions 176
B. Chemical Classifications 176
C. Physiological Classifications 176
1. Irritants 177
2. Asphyxiants 177
3. Anesthetics and Narcotics 177
4. Systemic Poisons 178
5. Particulate Matter Other Than Systemic Poisons 178
II. Respiration 178
A. Mechanics of Respiration 178
B. Lung Structure, Vital Capacity, and the Dead Space 179
C. Regulation and Control of Respiration 180
D. Function of Hemoglobin 180
E. Circulation as a Factor, and Its Regulation 181
III. Absorption, Distribution, and Elimination 182
A. Modes of Expressing Concentrations 182
B. Volume–Pressure–Temperature Relations 183
1. Partial Pressures 183
C. Role of Solubility in Absorption of Gases and Vapors 184
1. Solubility Coefficient 184
2. Body Saturation 185
3. Effect of Intermittent Exposures 189
4. Relative Concentrations of Vapor in Blood, Tissues, and Expired Air .. 189
D. Dusts and Fumes 190
1. The Absorption of Particulate Matter 190
2. Action of Suspended Particulate Matter 191
E. Mists 191
F. Other Means of Absorption 192
1. Ingestion 192
2. Absorption through the Skin 192
IV. Standards of Physiological Response 193
V. Standards for Permissible Atmospheric Contamination and How They Are Set. 194

VIII. Sampling and Analysis of Atmospheric Contaminants. By FRANK A. PATTY 199
Gases and Vapors 200
I. Methods Giving Quantitative Results in the Field 200
A. Evaluating the Intensity of Odor and Irritation 200
B. Portable Rapidly Indicating Devices 203
1. Interferometer 204
2. Portable Orsat 206
3. Combustion Devices 206
4. Ultraviolet Absorption Devices 208
C. Collection in Indicator Medium 208
D. Hand Piston Pump, Luer Syringe, and Rubber-Bulb Collection Devices 210
II. Methods Requiring Laboratory Analysis 210
A. Halogenated Hydrocarbons Combustion Apparatus 210
B. Adsorption for Evaluation by Weight 212
C. Condensation at Low Temperatures 213
D. Collection by Absorption or Adsorption for Laboratory Analysis 213
E. Sampling in Evacuated Bottles 213
1. Vacuum Bottle Samples 213
2. Samples in Partially Evacuated Bottles 214
F. Spectrometry 214
1. Infrared 214
2. Ultraviolet 215
3. Light Absorption 215
Dusts, Fumes, and Smokes 215
I. Sampling 215
A. Impingement 215
1. Greenburg-Smith Apparatus 216
2. The Midget Impinger 216
3. The Konimeter 217
4. Owen's Jet 217
B. Electrostatic Precipitation 218
C. Filtration 219
D. Thermal Precipitation 219
E. Sedimentation 220
II. Evaluation of Dust Samples 220
A. Making a Dust Count 220
1. Counting Impinger Samples 220
2. Counting Konimeter Samples 224
3. Counting Owen's Jet Samples 224
B. Determining Particle-Size Distribution 225
C. Weighing the Sample 227
D. Analyzing the Dust 228
1. Chemical Analysis for Toxic Materials 228
2. Determination of Quarts 228
Bacteria 232

IX. Radiant Energy and Radium. By LEON F. CURTISS 235
I. Fundamental Concepts of the Production of Injury by Radiation 235
A. Ionization as Cause of Injury 235
B. Target Theory 235
C. Injuries in Higher Forms of Life 236
D. Genetic Effects of Radiation 236
E. Visible Evidence of Injury from Radiation 238

II. Penetrating Ionizing Radiation ... 239
A. Types of Exposure and Injuries ... 239
1. Latent Period ... 239
2. Tolerance Dose ... 240
3. Methods of Measuring Exposure ... 241
4. Blood Counts as Index of Injury ... 244
B. Protection from Gamma Rays ... 245
1. Pre-employment Examination ... 245
2. Protection of Personnel ... 245
3. Storage of Radium ... 246
4. Manipulation ... 247
5. Exposure Table for Gamma Rays ... 248
6. Protection of Messengers ... 249
7. Transportation by Common Carrier ... 249
C. Protection from X-Rays ... 250
1. Special Features ... 250
2. Protection from Electric Shock ... 251
3. Shielding Materials for Protection against X-Rays ... 252
4. Shield of the X-Ray Tube ... 253
5. Screening Complete Rooms ... 253
6. Ventilation ... 254
7. Special Conditions ... 254
8. Unnecessary Hazards ... 254
III. Infrared and Ultraviolet Radiation ... 255
A. Infrared ... 255
1. General Effect ... 255
2. Protective Measures ... 255
B. Ultraviolet ... 255
1. General Effect ... 255
2. Protective Measures ... 256
3. Tolerance ... 257
IV. Corpuscular Radiation ... 257
Neutrons ... 257
1. Types and Effects ... 257
2. Measurement ... 258
3. Shielding ... 259
V. Poisoning from Radium or Thorium ... 259
A. Radium Poisoning ... 259
1. Historical Summary ... 259
2. Radioactive Nature of Radium ... 261
3. Injuries and Symptoms in Radium Poisoning ... 263
4. Tolerances ... 264
5. Detection of Unsafe Conditions ... 265
6. Protective Rules for Radium Dial Painting ... 269
B. Thorium Poisoning ... 272
1. Industrial Use of Thorium ... 272
2. Tolerances ... 273
3. Methods for Measurement of Thoron ... 273
4. Protective Rules for Handling Thorium ... 274

X. Ventilation. By W. N. WITHERIDGE ... 275
I. Human Ventilation Requirements ... 276
A. Air Composition ... 276

B. Carbon Dioxide as an Index of Ventilation Requirements ... 277
C. Effect of Room Size on Per Capita Ventilation Requirements 278
D. Mines, Tunnels, and Underground Spaces ... 279
II. General Industrial Ventilation ... 280
A. Classification of General Industrial Ventilation ... 280
1. Natural General Ventilation ... 280
2. Mechanical General Ventilation ... 280
B. General Ventilation Specifications ... 281
1. General Ventilation Rates in Terms of Air Changes ... 281
2. General Ventilation Rates Based on Floor Area ... 282
3. Dilution Ventilation (General and Local) ... 283
4. General Ventilation for Control of Solvent Vapors ... 284
5. Vapor Equivalents of Liquid or Solvents ... 285
C. Dispersion of Air Contaminants by General Ventilation ... 286
D. Provisions for Make-Up Air ... 286
1. Airbound Rooms and Buildings ... 287
2. Estimating Infiltration by the "Crack Method" ... 288
3. Cost of Heating Make-Up Air ... 288
4. Heat Loss Analysis ... 290
5. The Degree-Day ... 291
6. Heat Loss Analysis Using Degree-Day Data ... 291
7. Heating Values of Various Fuels ... 291
E. Air-Supply Inlets and Exhaust Outlets ... 292
1. Air Inlets ... 292
2. Air Outlets ... 292
F. Short-Circuiting the Air Flow ... 293
G. Successive Ventilation ... 293
H. General Ventilation vs. Local Exhaust Ventilation ... 294
III. Industrial Process Ventilation ... 294
A. Industrial Process Enclosures ... 295
1. Paint-Spraying Rooms or Booths ... 295
2. Welding Booths or Rooms ... 295
3. Abrasive-Blasting Rooms ... 296
4. Metalizing or Metal-Spraying Rooms ... 296
5. Grinding Booths ... 297
B. Local Exhaust Ventilation ... 297
1. Exhausting vs. Blowing ... 299
2. DallaValle's Equation for Unobstructed Hoods ... 299
3. Flanges and Baffles Increase the Range of Exhaust Hoods ... 300
4. Air Velocities for Control of Air Contaminants ... 301
5. Specific Gravity of Gases and Vapors ... 303
6. Canopy Hood Ventilation ... 303
7. Sidedraft or Backdraft Hoods ... 304
8. Downdraft Hoods ... 306
9. Face Velocity ... 306
10. Slot Exhaust Ventilation for Tank Processes ... 306
11. Tailpipe Exhaust Systems for Internal-Combustion Engines ... 308
12. Special or Irregular-Shaped Hoods ... 310
C. Ventilation in the Munitions Industry ... 314
IV. Air-Cleaning Methods and Equipment ... 314
A. Selection and Performance of Air Cleaners ... 314
B. Types of Air Cleaners ... 316

1. Centrifugal and Dynamic Collectors: Cyclones and Rotary Cones . . . 316
2. Wet Collectors: Spray Washers, Scrubbers, and Condensers . . . 317
3. Filters: Cloth, Felt, Paper, Asbestos, Glass, and Metal . . . 318
4. Electrostatic Precipitators . . . 319
C. Recirculation from Air Cleaners . . . 320
D. Air Bacteria Control . . . 320
V. Aeromotive Methods and Equipment . . . 321
A. Natural Aeromotive Forces . . . 321
1. Wind Direction, Velocity, and Pressure . . . 322
2. Roof Ventilators . . . 323
3. High-Temperature Stacks . . . 324
B. Mechanical Aeromotive Forces . . . 324
1. Fans: Blowers and Exhausters . . . 324
2. Ejectors, Venturi Ejectors, or Syphon Jets . . . 328
VI. Design of Ducts for Exhaust Systems . . . 328
A. Procedure for Design of Exhaust Systems . . . 329
B. Design Example . . . 336
C. Relation between Air Velocity and Velocity Pressure . . . 336
D. The Wright Friction Chart . . . 336
E. Reynolds Number . . . 338
F. Flexible Metal, Canvas, or Rubber Ducts . . . 338
G. Adjustable Dampers or Flow Regulators . . . 338
H. Pneumatic Conveying Systems . . . 339
I. Construction Specifications for Ventilating Systems . . . 339
J. Central vs. Unit Exhaust Systems . . . 339
K. Maintenance of Ventilating Equipment . . . 340
VII. Air Flow Observation and Measurement . . . 340
A. Visual Methods of Observing and Measuring Air Flow . . . 341
1. Smoke Clouds . . . 341
2. Floating Objects . . . 342
B. Static and Dynamic Pressure Indicators . . . 343
1. The Pitot Tube or Pitot-Static Tube . . . 343
2. Suction at Exhaust Hood Throat . . . 343
3. Venturi and Orifice Meters, Nozzles . . . 344
4. Propeller or Revolving-Vane Anemometer . . . 345
5. Deflecting-Vane Anemometer . . . 345
C. Chemical Methods of Measuring Ventilation . . . 346
D. Thermal and Electrothermal Measurements of Air Flow . . . 346
1. Kata Thermometer (Standard and Electric) . . . 346
2. Electrically Heated Thermometer Anemometer . . . 347
3. Electrically Heated Wire Anemometer . . . 347
4. Electrically Heated Thermocouple Anemometer . . . 347
E. Air Movement in Unconfined Spaces . . . 348

XI. Occupational Dermatoses. By LOUIS SCHWARTZ, M.D. . . . 349
I. Historical Data . . . 349
II. Incidence . . . 350
III. Causes of Occupational Dermatoses and Their Classification . . . 351
A. Predisposing Causes . . . 351
1. Race . . . 352
2. Age . . . 352
3. Sex . . . 352

4. Season of the Year . . . 352
5. Perspiration . . . 352
6. Presence of Skin Disease . . . 353
7. Uncleanliness . . . 353
8. Allergy . . . 353
B. Actual Causes . . . 354
1. Mechanical . . . 354
2. Physical . . . 354
3. Chemical . . . 354
4. Biologic Agents . . . 358
IV. Clinical Types of Occupational Dermatitis . . . 359
V. Diagnosis of Occupational Dermatoses . . . 359
The Patch Test in Industry . . . 360
1. Technique . . . 361
2. Interpretation and Reading of Patch Tests . . . 363
3. Complications of Patch Tests . . . 364
VI. Prevention of Occupational Dermatoses . . . 365
1. Pre-employment Examinations . . . 365
2. Ventilation . . . 366
3. Protective Clothing . . . 366
4. Cleanliness . . . 368
5. Protective Ointments . . . 368
VII. Treatment . . . 370
VIII. Occupational Cancer . . . 370
Prevention of Occupational Cancer . . . 371
IX. Methods of Investigation . . . 372
X. Skin Hazards According to Types of Workers or Occupations . . . 374

XII. The Visible Marks of Occupation and Occupational Diseases. By CAREY P. McCORD, M.D. . . . 381
I. Introduction . . . 381
II. Stigmata of Degeneration . . . 383
III. Marks from Work and Work Diseases . . . 385
IV. Summary . . . 386
V. Common External Marks of Occupation or Occupational Diseases or Other Diseases (Table) . . . 387

XIII. *Section One.* Fire and Explosion Hazards of Combustible Gases and Vapors. By G. W. JONES . . . 409
I. Limits of Inflammability . . . 409
A. Factors Affecting the Limits of Inflammability . . . 410
B. Limits of Inflammability of Gases and Vapors in Air . . . 410
C. Calculation of Limits of Inflammability . . . 415
1. One Combustible . . . 415
2. Two or More Combustibles . . . 417
3. Complex Gas Mixtures . . . 418
D. Limits of Inflammability of Gases and Vapors in Oxygen . . . 418
II. Ignition Temperatures . . . 419
A. Factors Affecting Ignition Temperature . . . 420
B. Minimum Ignition Temperatures of Gases and Vapors . . . 420
C. Ignition Temperatures in Air and Oxygen . . . 426
III. Flash Points . . . 426
IV. Temperature Range of Inflammability . . . 426

V. Methods of Minimizing Explosions 429
A. Control of the Oxygen Content of the Atmosphere 429
B. Operating Outside the Range of Inflammability 432
C. Use of Less Inflammable Materials 433
D. Elimination of Ignition Sources 434
E. Segregation of Hazardous Operations 435
F. Provision of Adequate Ventilation 436
G. Release Diaphragms and Vents 436
H. Combustible Gas Indicators for Inflammable Atmospheres 438

XIII. ***Section Two.*** **Explosion and Fire Hazards of Combustible Dusts.** By IRVING HARTMANN 439
I. The Explosion Hazard 439
A. Factors Affecting Explosibility of Dusts 440
1. Composition of Dust 440
2. Fineness and Physical Structure of Dust 441
3. Concentration of Dust Cloud 442
4. Composition of Atmosphere 443
5. Type of Ignition Source 444
6. Characteristics of Explosion Space 444
II. Laboratory Data on Dust Explosibility 444
A. Data on Ignition Temperatures 445
B. Minimum Explosive Concentration 447
C. Minimum Energy Required for Ignition 448
D. Maximum Pressure and Rates of Pressure Rise in Dust Explosions 448
E. Relative Inflammability of Dusts 450
III. The Fire Hazard 450
Prevention of Dust Explosions and Fires 451

XIV. Respirators and Respiratory Protective Devices. By FRANK A. PATTY 455
I. Historical Résumé and Present Approval Procedure 455
II. Types and Uses of Respiratory Protective Devices 456
A. Air-Purifying Respirators 456
1. Gas Masks (Canister Type) 456
2. Chemical Cartridge Respirators 458
3. Mechanical Filter Respirators (for Dust, Fume, Fog, or Mist) 459
B. Atmosphere-Supplying Respirators 459
1. Supplied-Air Respirators 460
2. Self-Contained Air- or Oxygen-Supplying Equipment 461
3. Newly Developed Air- or Oxygen-Supplying Devices 461
C. Unapproved Equipment 462
1. Filtering Efficiency against Paint Mist 462
2. Unreliable Carbon Dioxide Absorbents 463
III. Selection of a Respirator 463
IV. Cleaning and Sterilizing 465
V. Examining a Respirator 465

XV. Dust and Its Role in the Causation of Occupational Disease. By EDWARD E. DART, M.D. 467
I. Introduction 467
A. Properties of Dust 468
B. Atmospheric Dust Concentrations and Particle Size 469
II. Classification of Dust Based on Its Effect in the Body 470

III. Anatomical Factors of Importance in Injury by Dust ... 471
IV. Physiological Factors of Importance in Injury by Dust ... 475
V. Dust Causing Extensive Pulmonary Fibrosis (Silicosis and Asbestosis) ... 476
A. History ... 476
B. Exposure to Silica in Industry ... 476
C. Etiology of Silicosis ... 478
1. Composition of Dust in Relation to Production of Fibrosis ... 479
2. Number of Particles Inhaled in Relation to Production of Fibrosis ... 484
3. Particle Size in Relation to Development of Fibrosis ... 485
4. Individual Predisposition ... 486
D. Pathological Anatomy and X-Ray Findings ... 486
1. Nonspecific Pneumoconiosis ... 486
2. Discrete Nodular Silicosis ... 487
3. Modified Silicosis ... 495
4. Nodular Silicosis with Localized Conglomerate Lesions ... 496
E. Tuberculosilicosis ... 496
F. Diagnosis of Silicosis ... 500
G. Evaluation of Disability in Silicosis ... 500
1. Statement of the Problem ... 500
2. Pathological Physiology and General Principles ... 501
3. Terminology ... 503
4. Tests for Ventilatory Efficiency ... 504
5. Tests for Respiratory Insufficiency ... 507
6. Exercise Tests in General ... 508
7. Summary ... 508
H. Control of Silicosis in Industry ... 509
1. Engineering Control ... 509
2. Medical Control ... 509
3. Aluminum Prophylaxis and Therapy ... 510
I. Asbestosis ... 511
VI. Dust Causing Minimal Fibrosis or No Fibrosis ... 513
A. Silicates ... 513
B. Nonsiliceous Dust ... 514
VII. Dust Causing Chemical Irritation ... 515
VIII. Dust Causing Systemic Poisoning ... 515
IX. Dust Causing Allergic Manifestations such as Dermatitis, Hay Fever, and Asthma ... 516
X. Dust Causing a Febrile Reaction (Acting in an Unknown Manner, Possibly as an Allergen) ... 516
XI. Summary ... 517

Subject Index ... 519

CHAPTER FIFTEEN

Dust and Its Role in the Causation of Occupational Disease

EDWARD E. DART, M.D.

I. Introduction

The industrial hygienist and the industrial physician are concerned with dust because of its almost constant dispersion in air, where it may be inhaled and in some instances cause disease. This suspension of finely divided particles in the air may not ordinarily attract attention in rural areas but the smokiness of large cities is immediately apparent. Clean country air may contain as much as 0.2 mg. dust per cubic meter of air, whereas the average amount in city air during the winter months is approximately 0.5 mg. per cubic meter of air.[1] Thus, the cloud of dust that arises from a stonecutter's chisel is an excessive amount superimposed on that which is present in the already dust-laden air.

In addition to the medical problems, the legal entanglements which may revolve around dustiness are often of sufficient complexity that months of careful study are required to determine or fix responsibilities. In cities where general industrial smoke contamination is a perennial problem individual firms may be selected each year to bear the brunt of civic ire, and each year certain luckless corporations are forced to defend themselves in civil suits that are based almost entirely upon claims having no factual evidence for support. Medical-legal situations may be even more complex than the nonmedical problems and are complicated by numerous intangible factors. Not only does the exact nature of the disease and its relation to dust require proof, but the actual source of exposure must be determined so that a nondusty industry will not be held responsible for dust disease that may have resulted from exposure many years prior to the onset of the disease.

This chapter is written as an attempt to bring together facts necessary for a general understanding of dust and dust diseases. The presentation in the first portion is somewhat general so that the contents can be readily grasped by physicians with-

[1] J. M. DallaValle, *Micromeritics. The Technology of Fine Particles*, 2nd ed., Pitman, New York, 1948.

[illegible] extensive background in engineering or physics, and by engineers and industrial hygienists without medical training. In the later sections an attempt is made to review pneumoconioses in sufficient detail to aid the physician called upon to examine employees whose work exposes them to dust.

A. PROPERTIES OF DUST

The important physical and chemical properties of dust have been summarized by Drinker and Hatch,[2] and much of the material presented here was obtained from that source.

(*a*) *Settling rates.* Suspended microscopic dust particles are attracted to the earth by gravity just as is any other free body. However, because of their small mass and the relatively great resistance of the air, they do not fall according to the usual laws of gravity. Settling rates are much greater for large than for small particles. A settled dust, accordingly, may contain a proportionately greater percentage of large particles than the contaminated air from which it settled and in which the fines predominate. Ore as it is found in mines may contain a certain amount of quartz, which is frequently harder than the other minerals present. Because of this relative hardness the quartz may be ground less finely in drilling operations. Much of this coarser quartz dust may settle out from the air in the working area with comparative rapidity, leaving a smaller percentage of quartz in the atmospheric dust than may be present either in the parent material or in the settled rafter dust.

(*b*) *Flocculation.* Dust produced by crushing or grinding a material such as quartz differs from fume such as magnesium oxide in respect to its tendency to flocculate. Freshly formed fume particles are usually well below 0.3 μ and accordingly exhibit marked Brownian movement. Because of this and perhaps other factors, they tend to collide and form flocs which, because of their larger size, settle out relatively quickly. Such masses tend to adhere to vertical walls and projecting surfaces.

True dust is much less apt to flocculate than is fume, and any flocs formed are much more loosely bound together. Settling of dust clouds is therefore much less pronounced than is settling of fume clouds. As a dust cloud clears, the size of the particles remaining in suspension decreases and the degree of dispersion increases, i.e., the ratio of flocculated to discrete particles becomes less. On the contrary, in a metallic fume both the percentage of flocs and their size increase with the age of the cloud.

Turbulence in the air increases floc formation in fume clouds by increasing the collision frequency, but it does not improve the settling rate of dust.

Humidity below saturation is said to have little effect on flocculation time. However, humidity to the point of supersaturation is an important factor, as the dust particles function as nuclei on which water may condense, thus increasing particulate size.

(*c*) *Wetting.* Wetting is primarily an adsorption phenomenon in which the

[2] P. Drinker and T. Hatch, *Industrial Dusts.* McGraw-Hill, New York, 1936.

surfaces of the particles become covered with a film of water. Most liquids tend to spread on plane surfaces, but great force must often be used to wet dust, probably because the particles are already surrounded by a film of air. Wetting is of importance in dust sampling and in control of dust.

Three factors have been described by Drinker and Hatch as of importance in the wetting of dust. First, it is necessary to maintain intimate and vigorous contact of long duration between the dust and liquid. Second, it is advantageous to apply the liquid immediately at the dust source, because the heat usually generated in producing dust by drilling or grinding hampers air adsorption and because continuous flooding at the source excludes air. Third, wetting agents may be used to increase the wetting power of water.

(*d*) *Electrical properties.* Dust particles are electrically charged and accordingly are attracted to oppositely charged particles. If, therefore, particles in a dust suspension are given an electrical charge, the tendency for floc formation should be increased. Attempts have been made to utilize electrically induced flocculation as a method of air purification but without great success. Electrostatic precipitation, on the other hand, has been used quite successfully for collection of dust and fume, but such precipitation depends upon the attraction of the electrically charged particles to an oppositely charged plate rather than upon flocculation.

(*e*) *Optical properties.* Dust or moisture particles reflect light and it is this light, reflected from dust particles otherwise too small to be seen, that makes them visible when a beam of light enters a darkened room. The scattering of a beam of light by dust or mist is known as the Tyndall phenomenon. The optical properties of suspended particles in air vary with their shape, their transparency, and especially their size. With particles larger than the wave length of light (about 0.7 μ), the strength of the Tyndall beam varies directly with particle surface area and concentration; thus, for a given weight concentration it varies inversely with the particle size. Attempts have been made to utilize these optical properties in the measurement of dust concentration, but variation in the size of the particles makes such determinations unreliable.

B. ATMOSPHERIC DUST CONCENTRATIONS AND PARTICLE SIZE

Whenever a solid or liquid is ground or broken into particles as small as dust, the surface area is greatly increased. Thus if a cube of mineral 1 cm. in each of its dimensions is ground into small cubes 1 cu. μ in size, there will be 10^{12} particles with a total surface area of 6 sq. meters as compared with 6 sq. cm. for the original cube. This great increase in surface area is largely responsible for the increased chemical activity of finely ground materials and may be an important factor in the production of silicosis.

Industrial dust concentrations may range from the amounts present in "pure air," in operations where no dust is produced or where there is adequate dust control, to as high as 2 billion particles per cubic foot of air, in rock drilling. DallaValle[1] has pointed out that more than 50 per cent of industrial dusts are greater than 1 μ in

size were in atmospheric dust the median size is approximately 0.5 μ. Bloomfield found that only 20 per cent of the particles found in fire-clay dust were smaller than 1 μ and that the median size was 1.3 μ; he suggested that most of the particles of smaller size found in industrial atmospheres were lost in the mouth parts in air. In certain industries, however, such as pneumatic rock drilling, large numbers of particles well below 1 μ in size are produced.[3]

II. Classification of Dust Based on Its Effect in the Body

The industrial hygienist is interested in dust because of its effect on the human body. Therefore, a limited classification of dust from this viewpoint may form a basis for relating the chemical composition of dust to the anatomical and physiological reactions which occur in injury from dust.

(*1*) *Dust causing extensive pulmonary fibrosis.* This group includes all dust containing free silica or asbestos. Free silica is encountered in nature in the following forms: *quartz*—a crystalline form found in granite, schist, quartzite, sandstone, and sand; *opal*—an amorphous colloidal hydrate found in diatomaceous earth; *flint*—a crystalline form found free or in association with chalcedony; *chalcedony*—a mixed form consisting of fibers of quartz and an *interstitium* of opal. Other less common forms are tridymite, cristobalite, and siliceous or vitreous glass. *Asbestos* is hydrated magnesium silicate. It is mined in Canada as the mineral chrysotile.

(*2*) *Dust causing minimal pulmonary fibrosis or no fibrosis.* This group includes almost all inorganic dust except that containing free silica or asbestos. To the industrial hygienists the silicates, carbon, iron, and barium are the most important forms of dust in this group—the silicates because of their close chemical relationship to free silica, carbon because of its frequent occurrence in industrial atmospheres and its tendency to accumulate in the lungs, and iron and barium because of the unusual roentgenographic findings produced.

(*3*) *Dust causing chemical irritation.* This group includes obvious chemical irritants such as acids, alkalies, fluorides, and chromates. (Fluorides may also be included in group 4.)

(*4*) *Dust causing systemic poisoning.* This group includes dust of certain metals such as lead, arsenic, drugs, and all systemic poisons that may occur in dusty form.

(*5*) *Dust causing allergic manifestations such as dermatitis, hay fever, and asthma.* This group includes almost innumerable organic dusts such as pollens, synthetic resins, plastics, felt, fur, gums, kapok, leather, spices, tobacco, paper, rags, rayon, rosin, rubber, wood dust, starch, and wool. In addition to causing dermatitis and other allergic manifestations, these dusts may irritate the skin and mucous membranes by purely mechanical means.

(*6*) *Dust causing a febrile reaction (acting in an unknown manner, possibly as an*

[3] J. J. Bloomfield, *U.S. Pub. Health Repts.*, **48**, 961 (1933).
[4] T. Hatch and C. L. Pool, *J. Ind. Hyg.*, **16**, 177 (1934).

although . The two outstanding members of this category are metal fume (especially zinc oxide) and cotton dust.

III. Anatomical Factors of Importance in Injury by Dust

The fate of inhaled dust in the body can be understood only by knowing something of the anatomical structures and physiological reactions involved in respiration.

The air passageway consists of the nose, pharynx, trachea, bronchi, bronchioles, and alveoli. The relationship of these various structures is shown in Figure 1.

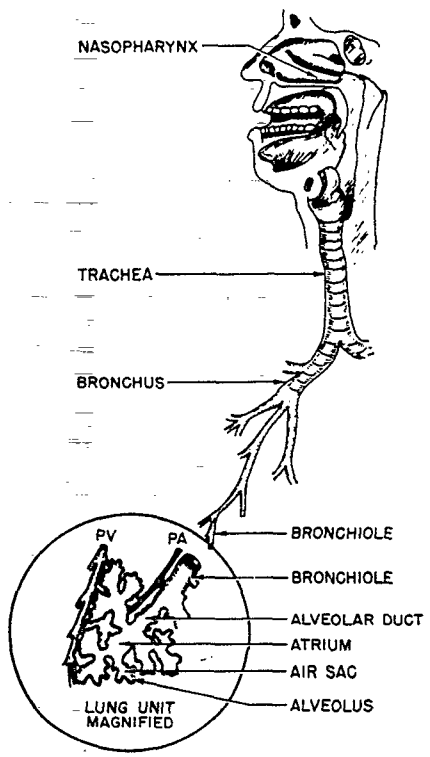

FIG. 1. Diagrammatic representation of the respiratory tract.[5]

(1) *Nose.* The portions of the nasal cavities just within the external nares are lined with skin containing hairs. The remaining parts of the nasal cavities are lined with mucous membrane, composed on its surface of ciliated and mucus-secreting cells. This tissue is highly vascular and contains freely anastomosing venous channels. The nasal passages are connected directly with the air sinuses in the skull.

The hairs in the entrance of the nose and the mucous material secreted by the lining membrane collect many of the larger inhaled particles. The cilia of the entire respiratory tract tend to move material toward the mouth, those of the nose tending to force the mucus and particles in it toward the pharynx. An indication of the importance of the nose as a protective mechanism has been shown by G. Lehmann.[6] This investigator studied 426 miners, of whom 241 had silicosis and 181 were normal, and found the median efficiency of nasal filtering in the silicotics to be 27.5 per cent and in the normals 45 per cent. He concluded from this that persons who develop silicosis do so, in part at least, because of a faulty filtering mechanism in the nose.

(2) *Pharynx.* The pharynx is a common pathway for food and air, connecting

[5] L. E. Hamlin, *Rocky Mt. Med. J.*, **41**, 391 (June, 1944).
[6] G. Lehmann, *Arbeitsphysiol.*, **7**, 147 (1933); see also *J. Ind. Hyg.*, **17**, 37 (1935).

Fig. 2. The two lungs with the trachea and the branching of the bronchi exposed by removing portions of the lung

the nasal passage with the oral pathway and with the trachea. Its nasal portion is lined with ciliated epithelium.

(3) *Larynx and trachea.* The larynx lies between the pharynx and trachea. It is strengthened by strong cartilages, contains the vocal mechanism, and has at the opening to the oral passage a valvelike structure—the epiglottis—which closes the tracheal opening during swallowing to prevent food from entering the respiratory pathway. The trachea is a tube 1 to 2 cm. in diameter, strengthened by cartilage rings and, like the nasal pharynx, lined with ciliated and mucus-producing cells. The relationship of the trachea and bronchi to the lungs as a whole is shown in Figure 2.

(4) *Bronchioles and alveoli.* Macklin[8] divides the bronchial tree into two parts that may be compared to the trunk and branches of a tree. The first part, which extends from the trachea to the *terminal bronchioles* inclusive, serves simply as an air conduit, and, like the branches and twigs of a tree, has no respiratory function. The terminal bronchioles are the last of a series of subdivisions of these nonrespiratory bronchioles. The muscular tissue in their walls is more highly developed than that in any other part of the bronchial tree and when fully contracted exerts a sphincterlike action which can completely shut off the air supply to the chambers beyond.

The structures lying distal to the terminal bronchioles are the "leaves" of the bronchial tree. They have a respiratory function: the interchange of gases between air and blood occurs through their walls. The respiratory portion consists of the respiratory bronchioles, alveolar ducts, alveolar sacs, and pulmonary alveoli. The cluster formed by a related group of these structures constitutes a *lung unit* or *primary lobule*. This is the distensible or bellows part of the lung.

The *respiratory bronchiole* as described in Best and Taylor[9] has the same diameter as the terminal bronchiole, of which it appears as a branch or a continuation. Five or six *alveolar ducts* arise from each respiratory bronchiole. Each alveolar duct after a variable number of rebranchings gives rise to from three to six dilatations, the *alveolar sacs*. The bays in the walls of the latter constitute the *pulmonary alveoli*, which are lined by a single layer of flattened epithelial cells cemented together. The alveolar walls contain elastic fibers and a rich network of capillaries. Frequently a single capillary channel alone intervenes between the walls of adjacent alveoli. The blood in the capillaries is therefore separated from the air in the alveoli by only two membranes of the utmost delicacy—the alveolar and capillary walls, so the greatest freedom is afforded for the diffusion of gases from the blood to the alveolar air and from the alveolar air to the blood.

The bronchioles, as they approach the periphery of the lung, branch and rebranch repeatedly, diminishing in length with each subdivision. The first branchings

[7] J. Sobotta and J. P. McMurrich, *Atlas of Human Anatomy*, Vol. II. Stechert, New York, 1933.

[8] C. C. Macklin, *Am. Rev. Tuberc.*, **25**, 363 (1932).

[9] C. H. Best and N. B. Taylor, *The Physiological Basis of Medical Practice*. 3rd ed., Williams & Wilkins, Baltimore, 1943.

are about 1.5 mm. in length and from 0.3 to 0.4 mm. in diameter. The terminal respiratory bronchioles are from 0.2 to 0.5 mm. in length, but of [illegible] the same diameter as the more central subdivisions. That is, the bronchioles, though progressively smaller, show practically no decrease in diameter as they pass toward the periphery. The alveolar sac, however, is considerably wider than [illegible] the respiratory bronchiole or the alveolar duct from which it arises. The pulmonary alveoli are semiglobular and have diameters ranging from 0.075 to 0.125 mm.; the total number in the lungs has been estimated by Zuntz at 750 million. Willson estimates the total epithelial surface of the lungs at 70 sq. meters; of this, probably 55 sq. meters, over 25 times the surface area of the skin, is respiratory.

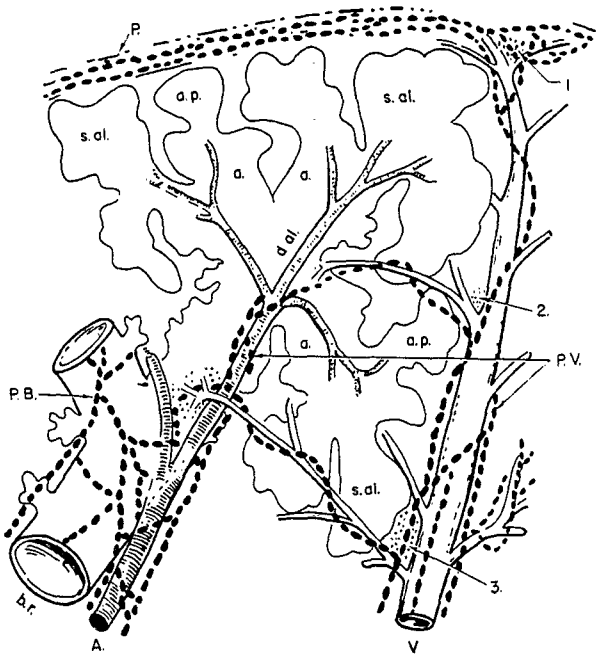

FIG. 3. Composite diagram (modified from Miller[11]) of the primary lobule lymphatic system, indicating the primary distributions or accumulation points for dust which will lead to predominant phases of pneumoconiosis: *b.r.*, respiratory bronchiole; *d.al.*, alveolar duct; *a.*, atria; *s.al.*, sacculi alveolare; *a.p.*, alveoli opening into respiratory bronchioles and alveolar ducts in close relation with the origins of peribronchial and perivascular lymphatics; *A.*, branch of pulmonary artery, accompanying the air passages; *V.*, branch of pulmonary vein in interlobular septum; *P.*, pleura; *P.B.*, peribronchial lymphatics; *P.V.*, perivascular lymphatics; 1, 2, 3, and other dotted areas, lymphoid deposits. (By previous permission of Dr. W. S. Miller.)

[10] H. G. Willson. *Am. J. Anat.*, **30**, 267 (1922).

[11] W. S. Miller, as reproduced by Pendergrass in A. J. Lanza, *Roentgen Diagnosis, Silicosis and Asbestosis*, Oxford Univ. Press, New York, 1938.

From these facts it is apparent that lung tissue offers by far the best medium of any in the body for the absorption of materials that may come in contact with it.

(5) *Blood vessels and lymphatics.* Except in the alveolar areas the blood vessels and lymphatics follow rather closely the path of the bronchial tree. The relationship in the periphery of the lung is shown clearly in Figure 3.

The lymphatic system consists of vessels that carry fluid similar to the blood plasma and nodules composed largely of protective and phagocytic cells. The lymphatic vessels and lymphoid tissue are important in connection with a study of dust because most of the dust particles finally lodge in the peribronchial and interstitial lymphoid tissues, carried there by wandering phagocytic cells.

(6) *Phagocytes.* In the alveolar spaces there are large cells capable of ingesting foreign material and having the power of independent motion. A large number of these ameboid forms appear wherever foreign material enters the lungs. After ingesting the exogenous particles, some of these cells pass into the blood stream and finally lodge in the spleen and liver or move elsewhere in the lung tissues; the majority, however, make their way to the lymph vessels and eventually to accumulations of lymph tissue, where they lodge. This resting place may be anywhere in the lung. Some phagocytes migrate peripherally to the pleura and others centrally to the hilus. In the lymph tissue the foreign material may be absorbed, may remain in an inert state, or may, by some chemical reaction, initiate pathological changes such as the formation of silicotic nodules.

IV. Physiological Factors of Importance in Injury by Dust

(1) *Volume of air inhaled.* Just as we are interested in the amounts of dust suspended in air, so are we also interested in the amount of air inhaled by men under different conditions. This has been discussed in Chapter 7.

(2) *Dust retention.* Before we can arrive at a logical basis for determining maximum permissible concentrations, it is necessary to know what proportion of inhaled dust is retained. Since most dust-collecting apparatus imposes considerable resistance to the passage of air from the lungs, satisfactory results were not obtained in such studies until Baumberger[12] used an electrostatic precipitator with tubes large enough to reduce the resistance to a negligible factor. His studies on tobacco smoke yielded results considerably higher than those obtained later by Sayers,[13] by Drinker, Thomson, and Finn,[14] and by Brown.[15] Baumberger found as much as 85 per cent retention of smoke. Sayers, working with tetraethyl lead in concentrations varying from 0.35 to 10.4 mg. per cubic meter of air, found that only 15 per cent was retained and attributed this limited retention to the small particle size. Brown's studies gave rather conclusive results, showing that the percentage retention is inversely proportional to the minute volume of air breathed and to the respiratory

[12] J. P. Baumberger, *J. Pharmacol.*, *21*, 47 (1923).
[13] R. R. Sayers, *U.S. Bur. Mines Repts. Investigations* No. *2661* (1924).
[14] P. Drinker, R. M. Thomson, and J. L. Finn, *J. Ind. Hyg.*, *10*, 13 (1928).
[15] C. E. Brown, *J. Ind. Hyg.*, *13*, 293 (1931).

rate, whereas it is directly proportional to the particulate size, density of the dust suspended in air, and the extent to which the dust is wetted while passing the air-water. He found that retention was not affected by volume per respiration, vital capacity, or relative humidity of inspired air.

V. Dust Causing Extensive Pulmonary Fibrosis (Silicosis and Asbestosis)

A. HISTORY

Since ancient times it has been thought that dust caused disease. Perhaps the earliest reference to disease caused by dust and fume is that of Plinius,[16] who described the devices used by refiners to prevent the breathing of "fatal dusts."

In 1556, in *De re metallica*, Agricola[17] stated:

"On the other hand, some mines are so dry that they are entirely devoid of water, and this dryness causes the workman even greater harm for the dust which is stirred and beaten up by digging penetrates into the windpipe and lungs and produces difficulty in breathing."

The best of the early writings on silicosis is that of Ramazzini,[18] translated in 1705 in *A Treatise of the Diseases of Tradesmen*, in which he stated:

"For in hewing marble or stones out of the rock, in polishing and cutting them, they oftentimes suck in by inspiration the sharp, rough and cornered small splinters or particles that fly off: so that they are usually troubled with a cough, and some of them turn asthmatick and consumptive . . . And in dissecting the corps of such artificers, the lungs have been found stuffed with little stones. Diemerbrock gives a curious relation of several stone cutters that dy'd asthmatick, and were opened by him: in whose lungs he found such heaps of sand that in running the knife through the pulmonary vesicles he thought he was cutting some sandy body. He adds that he was informed by a master stone cutter that in cutting stones there rises such a subtile dust, as is able to penetrate through ox bladders hung in the shop . . . And this very dust he took to be the cause of the death of many unwary workmen."

In recent years the contributions to knowledge of silicosis have been sufficiently substantial, numerous, and varied to make this perhaps the most widely discussed occupational disease. Silicosis is of particular interest to students of preventive medicine not only because of its general distribution in industry but more particularly because of its tendency to increase susceptibility to tuberculosis—a characteristic inherent in the disease. The limited space of this chapter does not permit reference to many sound contributions which have led to our present understanding of dust disease. An exceedingly interesting and somewhat detailed review of the history of diseases caused by dust may be found in the work of Lanza.[19]

B. EXPOSURE TO SILICA IN INDUSTRY

Since siliceous material makes up the bulk of the earth's crust, it is not surprising to find silica exposures in industry, where raw materials from the earth are used

[16] Caius Plinius Secundus, *Naturalis historia*, Bk. II. Trans. by K. C. Bailey under the title, *The Elder Pliny's Chapter on Chemical Subjects*, Pt. 1. Longmans, Green, New York, 1929.

[17] Georgius Agricola, *De re metallica*, Bk. I. Trans. from 1st Latin ed. of 1556 by H. C. Hoover and L. H. Hoover. Mining & Sci. Press, San Francisco, 1912.

[18] B. Ramazzini, *A Treatise of the Diseases of Tradesmen*, English trans., 1705.

[19] A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

to make many of the commodities necessary to our daily lives. Rocks and minerals are often intimately associated with free silica and it is obvious that those occupations concerned with mining, rock grinding, or drilling may constitute silicosis hazards, as well as occupations concerned with the processing and industrial use of siliceous products.

According to Knopf[20] the most common forms of free silica used industrially are massive crystalline quartz, quartzite, sandstone, flint, tripoli, diatomaceous earths and silica sand. Table 1, from Ladoo,[21] illustrates the great variety of uses to which silica is put in industry and indicates the kind of silica adapted to each purpose.

TABLE 1

Industrial Uses of Silica and Types of Silica Used[21]

Uses	Types
Abrasives	
In scouring and polishing soaps and powders.	Quartz, quartzite, flint, chert, sandstone, sand, tripoli and diatomaceous earth; all in finely ground state.
In sandpaper.	Quartz, quartzite, flint, sandstone and sand; coarsely ground and closely sized.
In sand-blast work.	Quartz, quartzite, sandstone and sand, crushed into sharp angular grains uniform in size.
Metal buffing, burnishing and polishing.	Ground tripoli and other forms of ground silica.
For sawing and polishing marble, granite, etc.	Sharp, clean sand graded into various sizes.
As whetstones, grindstones, buhrstones, pulpstones, oilstones, etc.	Massive sandstone from very fine- to moderately coarse-grained.
Tube-mill lining.	Chert, flint and quartzite in dense, solid blocks.
Lithographers' graining sand.	Medium to fine sand or rather coarsely ground silica and tripoli.
Tube-mill grinding pebbles.	Rounded flint pebbles.
In tooth powders and pastes.	Various forms of pure silica finely ground.
Wood polishing and finishing.	All forms of silica ground to medium fineness.
Refractories	
In making silica fire brick and other refractories.	Fairly pure quartzite known as gannister; not less than 97 per cent SiO_2 nor more than 0.40 per cent alkalies, tightly interlocking grains desired.
Metallurgy	
In making silicon, ferrosilicon and silicon alloys of other metals, such as copper.	Moderately pure sand, massive crystalline quartz, sandstone, quartzite or chert.
As a flux in smelting basic ores.	Massive quartz and quartzite.
Foundry-mold wash.	Ground sandstone, quartz and tripoli.
Foundry parting sand.	Fine sand and ground tripoli.
Chemical industries	
As a lining for acid towers.	Massive quartz or quartzite.
As a filtering medium.	Massive diatomaceous earth and tripoli, sand, finely granular quartz or quartzite, finely ground tripoli, diatomaceous earth and other forms of silica.
In the manufacture of sodium silicate.	Pure pulverized quartz sand, pure tripoli and diatomaceous earth.
In the manufacture of carborundum.	Pure quartz sand.

Table continued

[20] A. Knopf, *U.S. Pub. Health Bull.* No. *187* (1929).

[21] R. B. Ladoo, *Silica, Nonmetallic Minerals.* McGraw-Hill, New York, 1925, p. 525.

TABLE 1 *continued*

Uses	Types
Paint	
As an inert extender.	Finely ground crystalline quartz, quartzite and flint, also finely ground sandstone, sand and tripoli.
Mineral fillers	
As a wood filler.	Finely ground crystalline quartz, quartzite, flint, tripoli and other types of ground silica.
In fertilizers. In insecticides. As a filler in rubber, hard rubber, pressed and molded goods, phonograph records, etc. In road asphalt surfacing mixtures.	Finely ground silica of all types.
Ceramics	
In the pottery industry as an ingredient of bodies and glazes.	Flint, tripoli and chert, and other amorphous silica preferred; also all other forms of very pure silica, all finely ground.
In the manufacture of ordinary glass.	Pure quartz sand.
In the manufacture of fused-quartz chemical apparatus, such as tubes, crucibles and dishes.	Very pure massive quartz preferred.
Decorative materials	
In the manufacture of gems, crystal balls, table tops, vases, statues, etc.	Rock crystal, amethyst, rose quartz, citrine quartz, smoky quartz, chrysoprase, agate, chalcedony, opal, onyx, sardonyx, jasper, etc.
Insulation	
Heat insulation for pipes, boilers, furnaces, kilns, etc.	Massive and ground diatomaceous earth.
Sound insulation in walls, between floors, etc.	Massive and ground diatomaceous earth.
Structural materials	
Sand-lime brick.	Moderately pure, sharp, angular sand, preferably finer than 20-mesh, together with a small percentage of finely pulverized silica.
Optical quartz	
For the manufacture of lenses and accessories for optical apparatus.	Clear, colorless, flawless rock crystal or massive crystallized quartz.

C. ETIOLOGY OF SILICOSIS

Silicosis has been defined by the Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association[22] as:

"A disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and by characteristic x-ray findings."

The definition adopted at the International Silicosis Conference in 1930 and reaffirmed at the 1939 conference is generally accepted and is somewhat simpler than the above. It is as follows:

[22] R. R. Sayers, *Yearbook, Am. Pub. Health Assoc.* (1932–33).

Silicosis is a pathological condition of the lungs due to inhalation of [illegible] It can be produced experimentally in animals."

Lanza[23] in "Etiology of Silicosis," as printed in the National Silicosis Conference Report on Medical Control, states:

"Simple or uncomplicated silicosis is a chronic, fibrotic disease of the lungs due to the massive invasion of the pulmonary tissue by silica (SiO_2), inhaled in the form of dust. In its early stages, silicosis may be symptom free; in its later stages shortness of breath, decreased chest expansion, lessened capacity for work, may—one or more—be present, together with an increased susceptibility to tuberculosis. The disease presents the characteristic x-ray appearance of nodulation, without which a clinical diagnosis may not be made."

The production of silicosis depends upon the following factors: the composition of inhaled dust, the number of particles of inhaled dust, the size of the particles of inhaled dust, the length of time during which particles are inhaled, and individual susceptibility.

1. Composition of Dust in Relation to Production of Fibrosis

The fact that silicosis is caused by free silica (SiO_2) is attested by a great mass of clinical experience as well as by experimental observations. Gye and Kettle[24] have shown that silica in solution, or in noncrystalline form, stimulates the proliferation of fibroblasts in tissues. Gardner[25] produced a reaction in the lungs of animals in a period as short as two years by exposure to extremely high concentrations of dust. Miller and Sayers[26] developed an intraperitoneal injection technique for testing the tissue response to dust, and their method has been used extensively by the United States Public Health Service. On the basis of this procedure it has been found that all dusts behave in one of three ways in the body tissues; they may disappear (be absorbed), cause cellular proliferation, or remain inert in the tissues.

Dust Causing an Absorptive Reaction. No cases of pneumoconiosis have been reported and confirmed among workers exposed solely to dust of the absorptive group. In experimental animals this dust disappears, leaving little or no scarring. A list of different varieties of dust investigated, which are absorbed, follows. These are of industrial origin unless otherwise stated.

Calcite—essentially pure calcium carbonate.

Precipitated calcium carbonate—containing 10.1 per cent magnesium carbonate, 0.1 per cent magnesium oxide, 0.6 per cent iron and aluminum oxides and 0.4 per cent total silica.

Gypsum—the uncalcined, natural mineral composed essentially of calcium sulfate with 1.3 per cent total silica.

Limestone—not less than 82 per cent calcium carbonate, less than 12 per cent total silica, and not more than 10 per cent free silica.

Portland cement—containing 74.4 per cent calcium oxide and 21.1 per cent total silica. Free silica not reported.

Pyrolusite—composed of 54.9 per cent manganese with no quartz reported.

[23] A. J. Lanza, Report on Medical Control, National Silicosis Conference, *U.S. Dept. Labor, Div. Labor Standards, Bull.* No. *21*, Pt. 1 (1938).

[24] W. E. Gye and E. H. Kettle, *Brit. J. Exp. Path.*, *3*, 241 (1922).

[25] L. U. Gardner, in A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

[26] J. W. Miller and R. R. Sayers, *U.S. Pub. Health Repts.*, *56*, 264 (1941). Reprint 2234.

Dust Causing a Proliferative Reaction. Each type of dust examined that falls into the proliferative group is known to produce nodular, pulmonary fibrosis and each is a form of free silica. In experimental animals such dust produces nodules, which progressively increase in size until a maximum is reached, in about 90 days. At first the nodule is similar to that produced by dust which is absorbed. Later the fibroblasts are replaced by macrophages, which become filled with dust particles. Miller and Sayers give the following examples of this group:

Bisque ware—ground semivitreous pottery bisque ware, fired at a relatively low temperature. Total silica, 72.0 per cent; quartz, about 40 to 50 per cent; the remainder semifused clay and feldspar.

Chert—total silica, 76.1 per cent; quartz about 25 per cent; other forms of free silica, 35 per cent.

Diatomite—total silica, 92.5 per cent; aluminum oxide, 3.5 per cent; ferric oxide, 1.5 per cent; calcium oxide, 0.4 per cent; magnesium oxide, 0.7 per cent; essentially pure diatomite.

Greenware—ground vitreous unfired pottery ware.

Porcelain enamel frit—total combined silica, 35 to 50 per cent; the remainder is oxides of antimony, zinc, and aluminum, and fluorides of sodium, aluminum, and calcium.

Quartz—a pure mineral dust; normal crystalline quartz of high purity (SiO_2).

Quartz—identical with the above sample but treated with 0.6 per cent crude pine fatty acids.

Quartz-sericite—the source of this dust is unknown. Chemical analysis: total silica, 81.04 per cent; calcium oxide, 0.30 per cent; magnesium oxide, 0.45 per cent; sodium oxide, 0.10 per cent; potassium oxide, 0.98 per cent; iron oxide, 0.25 per cent; aluminum oxide, 14.25 per cent; total water, 2.61 per cent. Petrographic examination: quartz, about 50 per cent; muscovite (variety, sericite), about 45 per cent; fibrous sericite, less than 5 per cent.

Bisqueware, chert, greenware—containing from 69 to 76.1 per cent total silica and 25 to 50 per cent of this as quartz.

Diatomite—essentially pure and containing 92.5 per cent total silica.

Quartz—samples of pure mineral dust and industrial dust containing over 99 per cent SiO_2.

Quartz-sericite—about 50 per cent quartz and 50 per cent varieties of sericite.

Tripoli—total silica, 98.9 per cent; chalcedonic silica (crystalline aggregates) with an occasional crystal of normal quartz.

Dust Causing an Inert Reaction. Pneumoconiosis resulting from some of the forms of dust that are inert in the peritoneum has been reported. Clinical and pathological examples of the reactions show modified nodular fibrosis if the dust contains appreciable quantities of free silica; with other types of inert dust, diffuse interstitial fibrosis may sometimes result. The nodules produced in experimental animals become gradually flattened, and the dust is dispersed into the adjacent connective tissue, often to a considerable distance. Inert dust must be considered as a possible potential cause of pulmonary fibrosis. It is much less likely to cause pneumoconiosis than is dust that produces cellular proliferations in the peritoneum. Dust samples of the following descriptions were shown by Miller and Sayers to be inert:

Aluminum—pure aluminum bronzing powder of the finest grade.

Alundum—total silica, 4.6 per cent; aluminum oxide, 88.4 per cent; ferric oxide, 6.9 per cent.

Asbestos—total silica 37.5 to 50.86 per cent.

Anthracite coal—total silica, 6.6 to 8.6 per cent.

Bentonite—clay, variety montmorillonite, about 97 per cent; feldspar, about 2 per cent; quartz, none observed.

Bisque ware—ground vitreous pottery bisque ware, fired at a relatively high temperature. Quartz, about 30 to 40 per cent; the particles were wholly or partially covered by the glass phase. (This is absent in semivitreous bisque ware.)

Bituminous coal—total silica, 0.8 to 3.5 per cent.

Calcium phosphate—petrographic examination: earthy phosphates (not apatite), about 97 per cent; normal and chalcedonic quartz, about 3 per cent.

Chromite—total silica, 7.8 per cent; quartz, less than 5 per cent.

Diamond dust—pure bortz diamond dust used as abrasive.

Feldspar—total silica, 65.9 per cent; calcium oxide, 0.81 per cent; magnesium oxide, 0.10 per cent; aluminum oxide, 19.55 per cent; iron oxide, 0.28 per cent; potassium oxide, 8.98 per cent; sodium oxide, 3.18 per cent. Petrographic examination: feldspar (plagioclase-microcline), about 95 per cent; normal quartz, about 5 per cent.

Fuller's earth—filtral clay, containing from 55.7 to 62.1 per cent total silica and 1 to 10 per cent quartz.

Glass wool—finely ground sample of commercial hard glass wool.

Hematite (jewelers' rouge)—total silica, 1.5 per cent; iron oxide, 98.3 per cent.

Kaolin—china clay and hydromica predominant; quartz and feldspar, a trace.

Lanthanum sublimate—from the burning of white flame electrodes. Lanthanum, 40.0 per cent. Petrographic examination: particles too small to identify.

Mica—silica, 46.92 per cent; magnesium oxide, 0.86 per cent; aluminum oxide, 34.95 per cent; ferric oxide, 2.65 per cent; potassium oxide, 9.54 per cent; sodium oxide, 1.02 per cent; manganese dioxide, trace. Petrographic examination: mica, both as plates and fibers, plates predominating, about 98 per cent; a very small amount of quartz and feldspar.

Precipitator ash—composed of siliceous material, magnesium, iron, aluminum and calcium oxides, or rare earth oxides of the cerium group. The total silica content ranges from 0 to 48.3 per cent and the quartz from 0 to 5 per cent.

Pyrophyllite—predominantly pyrophyllite, with a small amount of rutile and a small undetermined quantity of quartz.

Rock wool—a finely ground sample of commercial, insulating rock wool.

Selenium—selenium, 98.8 per cent; tellurium, 0.01 per cent; ash, 1.16 per cent.

Selenium—a chemically prepared sample of highest purity.

Sericite—a pure mineral dust. Total silica, 51.74 per cent; calcium oxide, 0.61 per cent; magnesium oxide, 1.74 per cent; sodium oxide, 3.40 per cent; potassium oxide, 4.48 per cent; iron oxide, 5.83 per cent; combined oxides, 31.82 per cent; total water, 6.26 per cent. Petrographic examination: sericite and feldspar residues (fibrous sericite predominates), about 95 per cent; quartz, less than 5 per cent.

Shale—silica, 61.0 per cent; aluminum oxide, 12.4 per cent; calcium oxide, 4.5 per cent; ferric oxide, 5.0 per cent; magnesium oxide, 1.3 per cent; sodium oxide, 2.3 per cent; potassium oxide, 1.5 per cent; moisture, 10.3 per cent. Petrographic examination: about 35 per cent quartz; the majority of the particles appeared to be coated with clay.

Silicon carbide—pure manufactured silicon carbide. Silicon, 67.5 per cent. Petrographic examination showed no impurities.

Soapstone—total silica, 36.8 to 49.9 per cent; calcium oxide, 1.7 to 5.0 per cent; magnesium oxide, 22.7 to 26.2 per cent. Petrographic examination: talc, about 55 to 65 per cent; dolomite, about 5 to 30 per cent; tremolite, about 15 to 30 per cent. No quartz observed.

Talc—total silica, 49 to 56.54 per cent; calcium oxide, 6.25 to 8.8 per cent; magnesium oxide, 22.6 to 30.74 per cent; calcium silicate, 0 to 11.00 per cent; calcium carbonate, 0 to 1.88 per cent; iron and aluminum oxides, 0 to 1.04 per cent; ignition loss, up to 4.60 per cent. Petrographic examination: talc, mostly fibrous, about 40 to 75 per cent; tremolite, about 25 to 60 per cent; calcite and (or) dolomite, up to 1 per cent.

Titanium oxide—a finely divided, high-purity sample.

Sodium silicate—a laboratory-prepared sample containing [illegible] sodium oxide to 3.2 parts silica. Higher ratios of sodium oxide killed the animals.

Trap rock—silica, 51.7 per cent; aluminum oxide, 16.0 per cent; ferric oxide, 2.0 per cent; ferrous oxide, 9.9 per cent; calcium oxide, 10.0 per cent; magnesium oxide, 6.2 per cent. Petrographic examination: feldspar, some slightly decomposed, about 45 per cent; pyroxene, about 45 per cent; magnetite, about 10 per cent; biotite, about 1 per cent.

Volcanic ash—silica, 54.4 per cent; aluminum oxide, 14.5 per cent; ferric oxide, 3.8 per cent; magnesium oxide, 2.6 per cent; calcium oxide, 0.7 per cent; ash, 78.2 per cent. Petrographic examination: fine volcanic ash partially altered to montmorillonite. No quartz observed.

Volcanic ash—a specially treated sample. Silica, 74.3 per cent; mixed oxides, 16.8 per cent; ferric oxide, 2.2 per cent; calcium oxide, 0.5 per cent; magnesium oxide, 2.2 per cent. Petrographic examination: glass only. No quartz or calcite observed.

The immediate response of body tissue to any dust is essentially a foreign-body reaction, but the subsequent behavior of the tissue to any given dust determines whether such a dust is harmful.

In the peritoneal injection experiments of Miller and Sayers only dust containing free silica caused fibrous proliferations.

Although it has been well established that silicon dioxide is the cause of silicosis, there has been little satisfactory explanation for its action. Earlier it was thought that the fibrous nature of the proliferation was related to the hardness or sharpness of the particles. However, Gardner[27] found that no fibrosis was produced by experimental inhalation of carborundum dust, which was even harder and sharper than silica. Thus, the action is clearly chemical, but the exact chemicophysiological reaction that takes place is still unknown.

The mixed reaction sometimes produced by dusts of the inert group in which silica is mixed with other minerals emphasizes the possible effect of mixed dusts on the development of silicosis. The probable importance of concomitant exposures has also been indicated by clinical experience. Chapman,[28] MacDonald and his associates,[29] and Kilgore[30] have reported cases of rapidly developing silicosis caused by breathing air with high concentrations of silica and alkali dust. Kettle[31] and McCord[32] failed to demonstrate such action experimentally. McCord, on the basis of extensive investigations of workers in six plants where there was exposure to silica and alkali dusts, found no evidence of an accelerator action by alkalies. In fact, the absence of silicosis in this group suggested an inhibitor action which he believes may have resulted from the marked increase in solubility of silica in the presence of alkali. Peritoneal injection in animals yielded no results to prove either an accelerator or inhibitor action of alkali in the formation of silica nodules; but alkali did cause silica to spread from the point of injection and increased its primary toxicity.

The absence of silicosis in ganister-brick manufacturing was thought at first due to an inhibiting effect of accompanying dusts; more recently it has been found

[27] L. U. Gardner, *Am. Rev. Tuberc.*, **20**, 883 (1929).
[28] A. M. Chapman, *J. Am. Med. Assoc.*, **98**, 9 (1939).
[29] G. MacDonald, A. P. Piggot, and F. W. Gilder, *Lancet*, **2**, 836 (1930).
[30] E. S. Kilgore, *J. Am. Med. Assoc.*, **99**, 1414 (1932).
[31] E. H. Kettle, *Proc. Inst. Mining Metallurgy (London) 43rd Sess.*, 1934.
[32] C. P. McCord, *Ind. Med.*, **5**, 17 (1936).

that the concentration of free silica in the work atmosphere was too low to cause fibrosis. Silicosis as found in coal miners (anthracosis) and in foundry men may be quite different in x-ray appearance and in its course from that observed in quartz grinders.

Gardner[3] investigated these effects experimentally using ferruginous chert and mixtures of quartz and calcined gypsum. He found that the percentage of free silica in air-borne dust may not be the same as in the parent substance. This fact may explain the slowness in development of silicosis in workers in certain areas and in certain types of work. When a mixture of calcined gypsum and quartz was present in air, particles of both minerals flocculated and the rate of settling of the mixture was greater than that of either dust in the pure state. Injection experiments with ferruginous chert showed that the iron in the mixture temporarily inhibited the action of silica. Gardner's summaries of these experiments follow:

1. Artificial mixture of equal parts of calcined gypsum and quartz. Materials ground separately to respirable sizes and mixed in a dusting hopper.

Quartz content of parent mixture 49.7%
Quartz content, air-floated dust in cages 29.6%
Average light-field count, air inside cages, 336 million particles per cu. ft.

No exposed animals developed silicosis within 25 months although only 15 months were required to produce nodulation with average concentration of 120 million particles pure quartz per cu. ft. of air. Of 17 animals exposed more than 25 months and up to 30 months, 7 showed nodular fibrosis of modified type. Remaining 10 developed only chronic pneumonitis.

2. Artificial mixture of 2 parts of calcined gypsum and 1 part of quartz prepared as above.

Quartz content of parent mixture 31.4%
Quartz content, air-floated dust in cages 17.1%
Quartz content, lung ash of exposed animals 15.4%
Average light-field count, air inside cages, 245 million particles per cubic foot of air.

No animal developed silicosis until exposures had been continued for 24 months. Mature nodules found in only 3 of 16 guinea pigs exposed 24–29 months. These lesions were of modified type. The other 13 showed nonnodular pneumonitis.

3. Ferruginous Chert I, a natural mixture ground at a mine to such size that a majority of the particles were less than 10 microns in diameter, but there were a considerable number of larger ones.

Free SiO_2 of parent mixture 54.6%
Free SiO_2 of settled rafter dust 38.4%
Free SiO_2 of air-floated dust in cages 10.4%
Free SiO_2 of lung ash of exposed animals 3.3%
Average light-field count, air in cages, 776 million particles per cubic foot.

No suggestion of silicotic reaction but merely pigmentation of lungs of animals exposed for maximal period, 18 months.

4. Ferruginous Chert II. A similar natural mixture from the same source as that used in exp. 3 but ground until all particles were 5 microns and less in diameter.

Free SiO_2 of parent material 67.5%
Free SiO_2 of settled rafter dust 61.0%
Free SiO_2 of air-floated dust in cages 33.5%
Free SiO_2 of lung ash of exposed animals 16.3%

[3] L. U. Gardner, "Reactions to Mixed Dusts," in *Fourth Saranac Laboratory Symposium on Silicosis*, B. E. Kuechle, ed., Employers Mutual Liability Insurance, Wausau, Wis., 1939.

Average light-field count, air in cages, 776 million particles per cubic foot.

No silicosis in any animal's lungs exposed under 3 years; in 8 of 17 animals exposed for [illegible] periods, modified nodules were present. The other 7 developed only pigmentation and chronic pneumonitis.

Thus we have both laboratory and clinical demonstration of the influence exerted by other dust on the development of silicosis.

2. Number of Particles Inhaled in Relation to Production of Fibrosis

From the findings of Gardner, quoted above, and from the discussion of flocculation and settling, in the preliminary section, it is apparent that the type of dust may play an important part in determining the number of particles in the workroom atmosphere. Not only may some components of admixtures flocculate and thus change the character of a dust, but silica itself, being a hard material with a tendency to form large particles as compared with those formed by softer minerals, may settle out more quickly than other components of a mixed dust.

However, at this point we are somewhat more concerned with the concentrations of dust that are harmful than with the factors that have produced any given concentration in the air. Determinations of free and total silica in the lungs of patients who have died after fibrosis developed and in those without fibrosis have been made by incinerating the lungs and chemically analyzing the ash for its silica content. Although there have been some variations in published data on the amount of silica in pulmonary tissue necessary to produce silicosis, Sladden,[34] McNally,[35] Badham and Taylor,[36] and Fowweather[37] found fairly comparable amounts. Drinker and Hatch[38] sum up the present knowledge somewhat as follows: as a rough guide, a total silica content as high as 0.2 per cent of dried lung can be considered normal. A content over 1 per cent is definite evidence of dust exposure, and this amount is usually accompanied by fibrosis. There is too much overlapping of data and variation in technique to be sure from published data of the significance of quantities between 0.2 and 1 per cent of ashed lung. This fact is of especial significance, as pointed out by Cummings.[39] Since there is usually no evidence of silicotic reaction in lungs containing less than 1.5 to 2 gms. of silica (1 per cent of weight of dried lung), it may be inferred that the contraction of silicosis does not necessarily follow the inhalation of silica. Silicosis occurs only after the inhalation of amounts in excess of a minimum.

In any control program we are of course concerned primarily with the amount

[34] A. F. Sladden, *Lancet*, **2**, 123 (1933).
[35] W. D. McNally, *J. Am. Med. Assoc.*, *101*, 584 (1933).
[36] C. Badham and H. B. Taylor, *Med. J. Australia*, *1*, 511 (1933).
[37] F. S. Fowweather, *Chem. Industries*, *53*, 713 (1934).
[38] P. Drinker and T. Hatch, *Industrial Dust—Hygienic Significance, Measurement and Control*. McGraw-Hill, New York, 1936.
[39] D. E. Cummings, "The Etiology of Silicosis," in *Fourth Saranac Laboratory Symposium on Silicosis*, B. E. Kuechle, ed., Employers Mutual Liability Insurance, Wausau, Wis., 1939.

of dust in the air that will cause fibrosis. The methods of determining atmospheric dust concentrations are discussed in Chapter Eight. Cummings suggested that atmospheric concentrations of silica dust should be considered in terms of two thresholds: the primary threshold, a level at which a healthy man can be employed for his lifetime without harm, about 5,000,000 particles per cubic foot (light-field count); and the secondary threshold, a level at which a healthy man will inevitably develop silicosis, about 100,000,000 particles per cubic foot (light-field count). The National Silicosis Conference summarizes the situation as follows: "There is evidence that for prolonged exposure a concentration of more than 5 million particles per cubic foot, of a highly siliceous dust, is dangerous. Therefore it is now considered good practice to hold concentrations of highly siliceous dust at 5 million particles per cubic foot, or less," as based on light-field counting methods.

Since standards of safe atmospheric dust concentration based on medical findings have been suggested tentatively for only a few industrial dusts, and since considerable study is necessary to form a basis for such standards for other industrial dusts, a tentative arbitrary measure of what is good practice may be used. This should be within the limits of good engineering practice and yet low enough to control the silicosis hazard for most industrial exposures. The following formula is frequently used to express the maximum permissible concentration of silica in air:

> Multiply the percentage of free silica by the total dust particle count per cubic foot (light-field technique). If the result is over 5 million, the concentration may be considered too high. For example: a dust containing 10 per cent free silica with an average total concentration of 30 million particles per cubic foot would give 0.10 times 30 million, which equals 3 million (good practice); a dust containing 30 per cent with an average total concentration of 50 million particles per cubic foot would equal 0.3 times 50, or 15 million (unsatisfactory). This formula is not applicable to any dust containing less than 5 per cent free silica.

The Division of Industrial Hygiene, National Institute of Health,[40] has suggested that an attempt should be made to have highly siliceous dusts kept at a concentration below 4,000,000 particles per cubic foot since considerable silicosis occurred in the pottery industry even with low dust concentrations.

Dust may also be simply a nuisance. It is considered good practice to control even relatively harmless dust sufficiently to prevent concentrations in excess of 50,000,000 particles per cubic foot (by light-field count) in the workroom air.

3. Particle Size in Relation to Development of Fibrosis

We have already mentioned the fact that large particles settle more rapidly than small ones and that particle size is important as a determining factor in the actual amount of dust in air. Particle size is even more important in determining the chemical activity of the dust and physiological responses to it. Chemical activity is, of course, increased in small particles because of the increased surface area. McCrae[41]

[40] R. H. Flinn, W. C. Dreessen, T. I. Edwards, E. C. Riley, J. J. Bloomfield, and R. R Sayers, *U.S. Pub. Health Bull.* No. 244 (1939).

[41] J. McCrae, *Pub. S. African Inst. Med. Research*, Report No. 3 (1913).

found that 70 per cent of the particles in silicotic lungs were less than [illegible] and that the largest were not greater than 10.5 μ. An upper limit of 10 μ [illegible] size that will produce silicosis has been suggested because larger particles are probably collected by mucus in the upper respiratory tract and removed out of the [illegible] ciliary action. The importance of small particles in the production of fibrosis has been well demonstrated by Tebbens, Schulz, and Drinker.[42] These investigators produced liver fibrosis in experimental animals by intravenous injections of suspended silica and found that particles less than 0.6 μ in diameter caused much more fibrosis than larger particles.

Briefly, silicosis is caused by the inhalation of silica particles less than 10 μ in diameter. Recent work suggests that particles of less than 0.6 μ may be of greatest importance in this respect.

4. Individual Predisposition

Detailed discussion of the role of individual predisposition in the etiology of silicosis is hardly necessary. Race and sex seem to play little part, although race may be a factor in the development of tuberculosis just as financial status may be a factor. Certainly it is known that of two persons working in the same dusty exposure one may contract silicosis in a few years while the other may wholly escape it. We have already mentioned the possible influence of differences in nasal filtration as a factor in causation of silicosis. It is likely that there are sound explanations for the many variations in individual susceptibility, but clarification is lacking.

D. PATHOLOGICAL ANATOMY AND X-RAY FINDINGS

Gardner,[43] whose descriptions of pathological anatomy have been used as a basis for much of this discussion, has classified the pneumoconioses as follows: *Nonspecific pneumoconiosis*, including all forms except silicosis and asbestosis, *silicosis* of the classical discrete nodular type, *modified silicosis* caused by the inhalation of dusts containing free silica mixed with certain other minerals, and *silicosis with conglomerate lesions*, in which healed or active infection probably plays a dominant role.

1. Nonspecific Pneumoconiosis

All dust other than that containing free silica and asbestos produces the same general reaction.

X-ray Examination. There is simply an accentuation of the normal, branching, treelike shadows cast chiefly by the pulmonary blood vessels. As an occasional variation in this picture there may be superimposed fine reticulations. These reticulations are thought to be caused by thickening of the sheaths of the pulmonary arteries and thickening of the interlobular septa. See Figures 4 and 5.

[42] B. D. Tebbens, R. Z. Schulz, and P. Drinker, *J. Ind. Hyg. Toxicol.*, **27**, 199 (1945).

[43] L. U. Gardner, in *Fourth Saranac Laboratory Symposium on Silicosis*, B. E. Kuechle, ed., Employers Mutual Liability Insurance, Wausau, Wis., 1939.

Gross Examination. Except with prolonged and intensive exposure, the pleural surfaces of lungs with nonspecific pneumoconiosis show only focal and lineal collections of pigment that are soft in consistency and not raised above the surrounding tissues. On cut surfaces, gross examination reveals rounded flecks of pigment 2 to 3 mm. in diameter, but a lens shows finer linear deposits in the interlobular septa and in the outer walls of the blood vessels. The entire lung may be colored by pigment as in the black lung of soft-coal miners, but no fibrosis is found unless there has been exposure to free silica. There may be small patches of emphysema in or adjacent to pigmented areas. Scars of healed infections differ from the usual scars by the presence of pigment.

Microscopic Examination. If death occurs during exposure, dust particles are found free in the peripheral air spaces or ingested by alveolar phagocytes that are found adherent to alveolar walls and in loose areolar tissue about the blood vessels, especially the arteries, and in the interlobular septa. These last two linear deposits are referred to as perilymphatic deposits because of their close relationship to the lymphatic trunks. The bronchi show relatively little dust; when any is present, however, it is found in the connective tissues just beneath the epithelium. If there has been no recent exposure (years), the intrapulmonary dust tends to be removed from the air spaces and deposited along the lymph trunks; in severe exposure large amounts of dust-filled phagocytes may remain in the alveoli. Nearly all pure substances other than silica cause little cellular reaction. Coal and some silicates, especially mica, may cause minor irritation without producing fibrosis. Unless the linear reaction is fibrous and caused by free silica, there is no altered susceptibility to tuberculosos.

2. *Discrete Nodular Silicosis*

X-Ray Examination. This form of dust disease is characterized in the x-ray by small discrete nodular shadows, uniformly distributed throughout all parts of both lungs with the possible exception of small emphysematous areas in the costophrenic region. In the absence of infection, the nodules are of uniform size, rarely exceeding 4 mm. and never more than 6 mm. in diameter. The nodules are sharp with well-defined borders. See Figures 6 and 7.

Gross Examination. The pleurae are studded with slightly elevated, grayish nodules 2 to 3 mm. in diameter, and around these there may be a flat zone of black, gray, or brown pigment. No pleural adhesions are present. The lungs are stiffer than normal but crepitus is present. There are palpable shotty nodules. The cut surfaces are seeded with black or gray nodules, 2 to 4 mm., or rarely 6 mm., in diameter. The edges of the nodules are well defined, but a lens shows pigmented strands radiating from their periphery. There is little confluence except where scars or infection are present. Usually some gross emphysema is present, but this condition may be apparent only with microscopic examination. For differentiation from perilymphatic pigmentation there must be definite pleural nodulation in an appreciable amount. Some nodules may arise along the peripheral branches of the pulmonary arteries.

FIG. 4. Normal chest.

FIG. 5. Increased linear markings. Increased linear markings may be seen in the roentgenograms of apparently healthy subjects, and they may result from infection, exposure to irritants or dust or from other causes. Increased linear markings may also be demonstrated in patients who later develop nodular silicosis. A definite diagnosis of pneumoconiosis can not be made from roentgenograms showing only increased linear or vascular markings.

FIG. 6. Nodular silicosis, uncomplicated (*courtesy L. E. Hamlin*).

FIG. 7. Nodular silicosis with infection in right upper lobe (*courtesy L. E. Hamlin*).

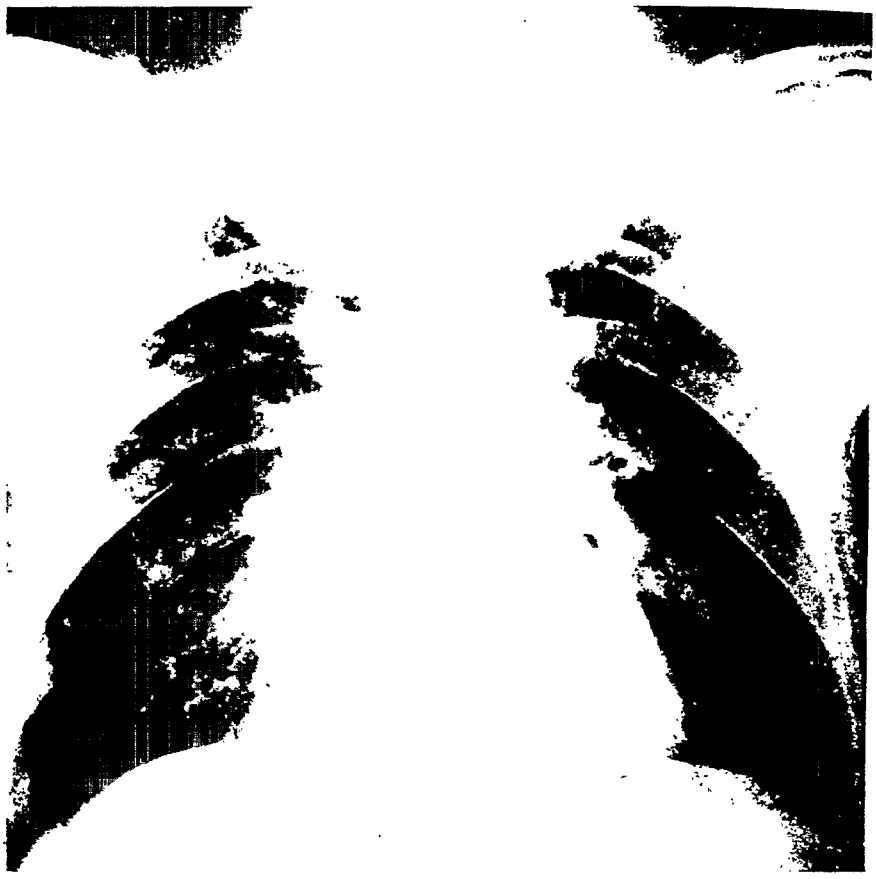

FIG. 8. Increased linear markings and indistinct nodulation. This type of abnormality is sometimes seen in roentgenograms of subjects exposed to mixed dust. This roentgenogram is about midway between that seen in nonspecific pneumoconiosis and that seen in nodular silicosis.

FIG. 9. Nodular silicosis with conglomerate lesions. It has been pointed out in the text that conglomerate lesions are ordinarily associated with infection, usually tuberculous. It is of interest to note that this patient was negative to tuberculin (P.P.D. first strength) and that he had had eleven sputums that were negative and none positive for tubercle bacilli (*courtesy L. E. Hamlin*).

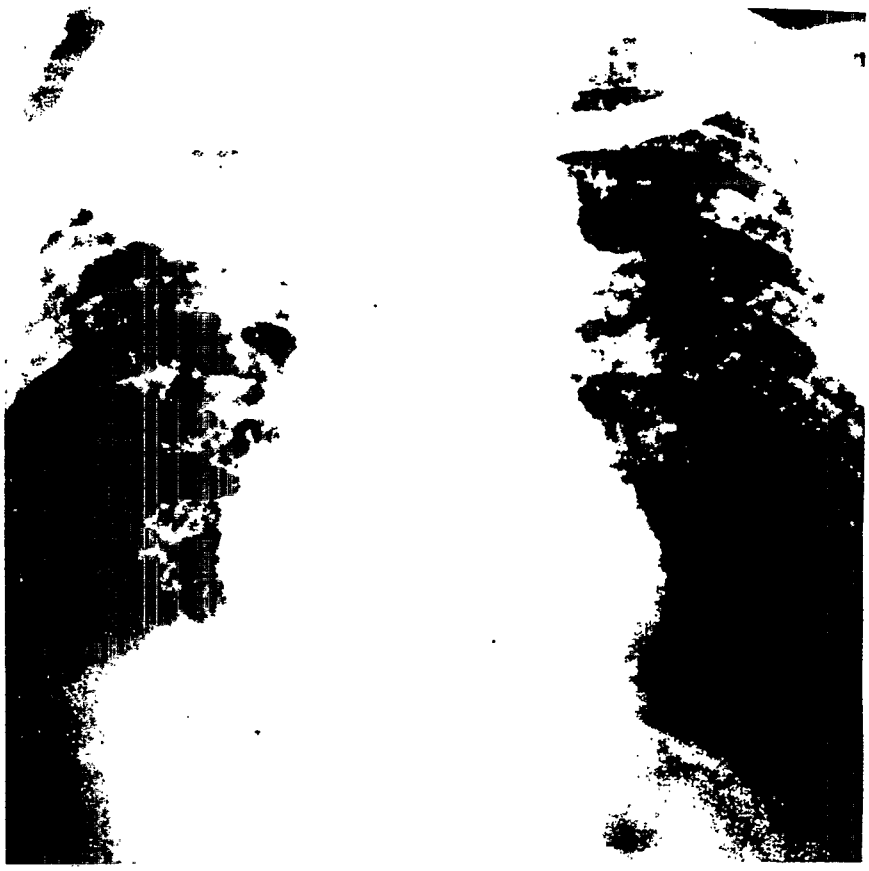

FIG. 10. Nodular silicosis with conglomerate lesions and infection (*courtesy L. E. Hamlin*).

The tracheobronchial lymph nodes are at first enlarged and firm, later small and extremely hard. Sections of these nodes reveal dense leatherlike tissue, which is silky in texture because of the decrease in collagenous connective tissue.

Microscopic Examination. Microscopic examination shows layers of dense hyaline collagenous fibers with nuclei so compressed as to be almost invisible at times. The borders are clear cut, with no exudation. There may be pigment either about the periphery or in focal points within the nodule itself. The nodule may be calcified, occasionally to the point of bone formation in its central portion. Nearly all nodules are associated with branches of the pulmonary arteries, as demonstrated by wax reconstruction of the arteries; they may form spherical or spindle-shaped masses around the arteries. Pigmentation occurs about the lymphatic trunk just as in nonspecific pneumoconiosis, but there is always some fibrosis. The air spaces immediately adjacent to the nodules are distorted and frequently dilated. There may be widespread emphysema, most marked in the costophrenic angle, where nodulation is most sparse. The emphysema is fine, not coarse or bullate. Ordinarily there is no leukocytic reaction. Early silicosis resembles nonspecific pneumoconiosis; the reaction, however, is more extensive and is fibrous rather than cellular. The closely related pathological pictures in early silicosis and in the nonspecific reaction would be expected since the x-ray findings in the two conditions are almost identical. Neither in early silicosis nor in nonspecific pneumoconiosis are the x-ray findings associated with any disability.

3. Modified Silicosis

Modified nodulation results from breathing dust containing free silica mixed with other minerals. Although a great deal is not known about mixed dust reactions, there have been sufficient examinations of the lungs of hard-coal miners to determine that lung tissue responds differently to the dust of coal mines than to pure silica dust.

X-Ray Examination. Long experience in interpretation of roentgenograms of silicotics with varied exposures may enable one to conjecture with a reasonable degree of accuracy the general type of exposure. Large amounts of nonsiliceous or silicate dust in the mixture tend to induce perilymphatic deposits of the linear type. Free silica intensifies this reaction and causes it to become fibrous. At times hyaline nodulation occurs, perhaps when there is enough free silica in the dust, but this reaction is atypical. Gross examination of the modified silicotic nodules may reveal heavy deposition of pigment with or without dense fibrosis. With a hand lens hard elevated nodules may be seen in the centers of some of the pigment deposits. See Figure 8.

Microscopic Examination. On microscopic examination it will be seen that many of the nodules consist of nothing but coarse, heavily pigmented fibers devoid of organized arrangement. Such nodules may have irregular peripheral extensions, which fuse with those of others. (This fusion of nodules may be partly responsible for the appearance produced in the conglomerate reaction.)

2. Nodular Silicosis with Large Conglomerate Lesions

X-Ray Examination. X-ray examination shows isolated or bilaterally symmetrical massive shadows usually located in the upper half of the lungs but sometimes in the lower third. These massive shadows may extend from the root to the pleural surface, or they may lie deep in the parenchyma. Serial x-rays may show that the lesions remain stationary over a period of five to six years or they may show an increase in size on successive pictures. Ultimately central rarefaction, interpreted as cavitation, may develop with subsequent evidence of infection in other parts of the lung (see Figures 9 and 10). Gardner[4] presents the following working hypotheses with regard to conglomerate lesions:

"1. In tuberculo-silicosis the infection may heal so completely that its tuberculous origin is no longer recognizable. Such an outcome has been observed in experimental animals exposed to ferruginous chert and infected with attenuated tubercle bacilli.

"2. Conglomerate lesions in the upper lung fields, particularly when they are bilateral, are in the great majority of cases due to an underlying tuberculosis in healed or latent form. In persons not exposed to dust, bilateral disease in this location proves to be tuberculous in 95 to 98 cases out of every hundred, and even unilateral disease can safely be considered tuberculous in 90 per cent of cases.

"3. Large isolated or bilaterally symmetrical conglomerations in the lower lungs should be considered tuberculous unless this origin can be excluded. In that case the possibility of organizing pneumonia due to Friedlander's bacillus or other organisms should receive consideration.

"4. The character of the fibrosis resulting from the combined activity of an infectious organism and silica dust with or without other minerals may suggest a relationship between the time of exposure to dust and the development of the infection.

"(a) When the silicosis is already established *before* tuberculosis supervenes the infection localizes in and about the nodules which retain their form but increase in size. Bronchial obstruction *caused by the infection* may produce widespread atelectasis with a consequent approximation of the nodules in the involved area. In such cases the outlines of large individual nodules in the resultant conglomeration are distinct and clearly defined.

"(b) When silicosis and chronic tuberculosis have developed simultaneously the nodular character of the conglomerate fibrosis is less obvious. Nodules are present but they are surrounded by a matrix of more diffuse fibrosis produced by the action of the silica upon an organizing pneumonic process.

"(c) When the scars of a healed infection are already present before employment in the dusty industry, the inhaled particles accumulate in particularly large quantities in their immediate vicinity. Continued accumulation in the localized area produces many nodules, which are small because they are closely packed together but typically spherical in outline because there is no activity in the underlying infectious process to produce diffuse fibrotic reaction."

Many patients with conglomerate silicotic fibrosis are dyspneic, but the toxic symptoms of the associated infection may be absent. This is reasonable, since healed tuberculosis does not cause symptoms and even active infections may be so well encapsulated by dense fibrous tissue that no tissue reaction is possible.

E. TUBERCULOSILICOSIS

The relationship between silica exposure and the development of tuberculosis has been well demonstrated in the United States as well as in other countries where

[4] L. U. Gardner, "Pathological Anatomy" in *Fourth Saranac Laboratory Symposium on Silicosis*, B. E. Kuechle, ed., Employers Mutual Liability Insurance, Wausau, Wis., 1939.

there has been such exposure. One of the most remarkable demonstrations of the relation of tuberculosis to silica dust exposure is found in the work of Mavrogordato,[45] illustrated graphically in Figure 11.

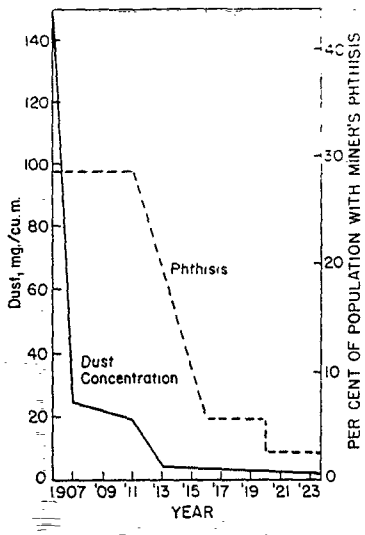

FIG. 11. Dust control and silicosis in South Africa.

The symptomatology and clinical findings in silicosis and in silicosis complicated by tuberculosis have been summarized in the National Silicosis Conference Report of the Committee on Medical Control, and these have been condensed by Lanza[46] as follows:

"SUBJECTIVE SYMPTOMS: *Dyspnea*—The complaint most frequently mentioned is shortness of breath. Depending upon the extent of the involvement, this varies from slight dyspnea, following exertion, to marked dyspnea upon the least exertion or even when at rest. The shortness of breath noted in silicotics presents one peculiarity in that it is seldom accompanied by orthopnea, the individual being no more short of breath lying down than when in an upright position. This may not be so, however, when silicosis is complicated by cardiac disease or by true asthma.

Silicosis	Silicosis with infection
Noted as a rule only after sudden or extra exertion. Seldom so marked as to interfere with routine duties. However, in cases with extensive pulmonary fibrosis, it may limit the individual's activities.	If complicating infection is not widespread, may be no more marked than in cases of simple silicosis but as infection and fibrosis increase, it becomes disabling.

"*Cough.* Many silicotics complain of a troublesome cough. This cough differs from that resulting from simple irritation due to dust which clears up upon removal from exposure. When present, it is more pronounced in the morning or upon beginning work after a rest period.

Silicosis	Silicosis with infection
The typical silicotic cough is dry and nonproductive. It usually parallels shortness of breath in degree and may contribute to disability.	The cough usually becomes more troublesome and is productive. The sputum varies from thick, tenacious, mucous material to that of a foul, purulent or purohemorrhagic consistency. Microscopic examination or animal inoculation frequently reveal tubercle bacilli or, in some cases, organisms of the fusiform spirochetal group. In advanced cases, coughing attacks are often of such severity as to leave the individual exhausted.

"*Chest Pain.* This symptom is complained of by a majority of silicotics. It varies from a feeling of tightness in the chest to the sharp pain typical of pleurisy. (Since chest pain is offered as a complaint in many conditions, it cannot be stressed as especially characteristic of silicosis.)

[45] A. Mavrogordato, *Pub. S. African Inst. Med. Research*, Report No. 19 (1926).
[46] A. J. Lanza, *Silicosis and Asbestosis*, Oxford Univ. Press, New York, 1938.

Silicosis

A late symptom in cases of simple silicosis. Then seldom more than a sense of tightness or feeling of substernal pressure

Silicosis with infection

Pleuritic pain is suggestive of a complicating infection. It is increased by exercise and by coughing and may be distressing in advanced cases with extensive infection.

"*Hemoptysis.* True hemoptysis seldom occurs. Frequently, however, the sputum may be blood-streaked following a severe coughing attack. Hemoptysis must always be considered as suggestive of tuberculosis.

Silicosis

Occasional blood-streaked sputum. May result from alveolar rupture following sudden exertion in advanced cases.

Silicosis with infection

May be the first indication of tuberculous infection. May be consequent upon the development of pneumothorax. May occasionally be excessive if cavities are present.

"*General Complaints.* Weakness, loss of weight, digestive disturbances, night sweats, insomnia, dizziness, and edema of the extremities are not characteristic of uncomplicated silicosis but are apt to be present if infection supervenes, especially when the infection becomes extensive.

"*OBJECTIVE SYMPTOMS*: Changes in the general appearance are infrequent in simple silicosis unless far advanced. Such changes as are manifested are usually due to complicating conditions.

Silicosis

Early cases appear unchanged; in fact, it is common to find these individuals showing a slight increase in weight, possibly because they are less active. As the disease progresses, respiratory embarrassment is noticeable and there is a general loss of muscle tone.

Silicosis with infection

The appearance sooner or later becomes that of chronic phthisis. The bony landmarks of the thorax become prominent and there is an increase in the anterior-posterior diameter of the chest, possible hypertrophy of the accessory respiratory muscles of the chest, and in the final stages, retraction of the supra and infra clavicular spaces. Cyanosis and clubbing of the fingers are not prominent except in those cases of long standing, with cardiac disturbances.

"*Chest Expansion.* Decrease in the expansion of the chest may be demonstrated in cases with extensive pulmonary fibrosis.

Silicosis

In early cases it is usually not possible to demonstrate decreased expansion. In advanced cases, expansion may be lessened by 20 to 30 per cent but remains equal on both sides.

Silicosis with infection

Decrease in expansion may not be noted in early silicosis with slight infection but as the condition progresses, a definite decrease is readily observed. When infection is more pronounced in one area of the lung, expansion may be more markedly decreased on the affected side, particularly if there is pleural involvement.

"*Prolonged Expiration.* In most cases, decreased chest expansion is preceded and later accompanied by a definite change in respiratory rhythm. Close observation reveals that even at rest there is a distinct tendency to prolongation of the expiratory phase, which, as silicosis advances, becomes more marked. Following exercise, the silicotic may breathe less rapidly than the normal person under similar conditions as the lungs cannot be emptied rapidly enough to permit more rapid respiration; but although the respiratory rate is not so rapid as in the normal person, it will persist for a longer period.

Silicosis

Early in the development of a simple silicosis, prolonged expiration may be evident only after exertion. Later the degree of prolongation usually parallels the increase in pulmonary fibrosis.

Silicosis with infection

In cases of early silicosis with slight infection, prolongation of the expiratory phase may be no more marked than in simple silicosis. As the condition progresses and fibrosis increases, it may simulate characteristic asthmatic respiratory rhythm.

Superficial palpation of the chest. Except in the late stages of silicosis with infection, rales are revealed by palpation of the chest. However, where there is a measurable decrease in expansion, one may note by palpation that, upon forced inspiration, the anterior chest wall is lifted forward by the accessory muscles of respiration.

Silicosis

No change noted until extensive fibrosis has taken place, when there may be an increase in tactile fremitus generally.

Silicosis with infection

When infection is extensive, tactile fremitus is increased and occasionally friction rubs may be elicited. Extensive thickening of the pleura or pneumothorax may result in a decrease or absence of tactile fremitus over affected areas.

"*Percussion.* There is usually an impairment of the percussion noted over the whole chest but unless one is particularly observant, this may not be detected.

Silicosis

Since impairment in resonance is general over all lung areas, it is difficult to demonstrate until advanced fibrotic changes have developed. Decrease in diaphragmatic excursion may sometimes be revealed by percussion.

Silicosis with infection

Increase in loss of normal resonance. When massive areas of fibrosis have developed, this may amount to absolute dullness over areas involved. Amphoric resonance may be elicted where there is pneumothorax. Decreased diaphragmatic excursion may readily be shown in advanced infection.

"*Auscultation.* Breath sounds are usually decreased in intensity and the characteristic prolongation of expiration is readily noted.

Silicosis

Decrease in breath sounds general and more marked as the condition progresses. Subcrepitant râles, which clear up after coughing, are occasionally heard.

Silicosis with infection

Usually heard, some of the following: Persistent post-tussic crepitant and subcrepitant râles; coarse rhonchi associated with productive coughing, wheezing or musical râles increased by exertion and coughing, amphoric breath sounds over cavities and areas of pneumothorax; pleural friction rubs occasionally."

Part of the symptomatology associated with silicosis may be due to pathological processes other than fibrosis. Filley, Hawley, and Wright,[47] using isolated perfused lungs of guinea pigs found that colloidal silica produced bronchial constriction, although soluble silica did not. This at least suggests that part of the disability in silicosis may result from bronchial constriction. Such findings also offer the first rational approach to a basis for explaining the improvement of symptoms in patients with silicosis after aluminum therapy. The inability of soluble silica to cause bronchial constriction, however, raises a question as to the validity of the assumption that this same reaction occurs in workers exposed to silica.

That exposure to silica dust may increase the tendency to contract respiratory diseases other than tuberculosis has been suggested by Sayers.[48] He concludes from a survey of the mining industry that:

"1. The principal pulmonary diseases to which the miner is subject are bronchitis, influenza and pneumonia, pulmonary tuberculosis, anthracosilicosis, and silicosis.

"2. Statistics indicate that much higher morbidity and death rates from pulmonary diseases are experienced in dusty than in nondusty industries, especially where silica dust is used or produced.

"3. Investigations by the Public Health Service reveal that hard-coal miners suffer a high

[47] G. F. Filley, J. G. Hawley, and G. W. Wright, *J. Ind. Hyg. Toxicol.*, **27**, 37 (1945).
[48] R. R. Sayers, *U.S. Bur. Mines Circ.* No. **7146** (1941).

mortality from influenza and pneumonia not only during periods of epidemics but at other times as well. In 1915–23 and 1906–25 influenza and pneumonia were responsible for 30% of the total deaths among hard-coal miners, compared with 25 per cent among metal miners and 7% of those who were engaged in pursuits other than coal mining. Six per cent of hard-coal miners showed silicosis, compared with 2 per cent in the bituminous coal fields. [illegible] The rate for metal miners is still higher."

F. DIAGNOSIS OF SILICOSIS

To make a definite diagnosis of silicosis one must have (*a*) a roentgenogram showing the appearance characteristic of fibrous nodulation and (*b*) proof of the subject's exposure to significant amounts of dust containing free silica. Gardner[49] points out the importance of an occupational history since a nodular or mottled x-ray pattern may occur in other conditions, including so-called "arcwelder's siderosis" and baritosis, in which no fibrous nodulation can be demonstrated by pathological examination. Organic iron from the blood may cast shadows that simulate silicosis. A striking example of the effect of blood is seen in roentgenograms of men trapped underground in mine explosions; serial films demonstrate the sudden appearance of fine nodulations, which disappear in a few weeks. Pulmonary sarcoidosis, a tuberclelike infection of unknown cause, occurring throughout the population at large, may produce similar shadows. Another condition to be considered is miliary calcification of the lungs, commonly called "wheatena"; the dense, not uniformly scattered nodules are thought to represent a stage in the healing of some infection of unknown nature. Likewise, cardiac decomposition may, by the accompanying pulmonary congestion, produce an x-ray picture that somewhat resembles silicosis.

All of this suggests the difficulty in diagnosing silicosis unless full details of all findings are available to the examining physician.

G. EVALUATION OF DISABILITY IN SILICOSIS

1. Statement of the Problem

To both management and labor the term "silicosis" has become nearly synonymous with the term "total disability." Accordingly, some time should be devoted to the details involved in evaluating pulmonary "ability" or "disability" in this disease. Actually, uncomplicated silicosis is almost notorious for its lack of symptoms even in relatively advanced stages. In fairness to both employer and employee it is necessary to go beyond simple diagnosis. In examining a patient with silicosis an attempt should be made to answer certain questions: Does his chest x-ray suggest that he has silicosis? Does he have a history of sufficient exposures to free silica dust to account for any roentgenographic changes present? Does he have complicating tuberculosis or other infection? Is he incapacitated or disabled by the condition? The first three of these questions are usually answered by the examining physician. Too frequently, however, not enough attention is paid to the fourth question, concerning disability. As a result of this an employee may be dismissed from work

[49] L. U. Gardner, *Am. Ind. Hyg. Foundation*, Annual Meeting, Pittsburgh, November 14, 1945.

unjustly or he may collect compensation for disability unjustly. For medical-legal purposes as well as for the purpose of fitting each afflicted employee into work within his capacity it is helpful to have records of progress on individual patients over a prolonged period, preferably many years. Industrial medicine may make some contribution to an understanding of pulmonary physiology by accumulating specific data on large groups of people, and in the final analysis this can best be done by men closely associated with employees, with management, and with the problems of industrial medicine.

We have already summarized the diagnostic criteria for silicosis and have discussed the findings indicative of complicating tuberculosis. It is necessary to bear in mind the fact that cardiac insufficiency, anemia, and other diseases may produce symptoms not readily differentiated from those of pneumoconioses. In establishing a diagnosis of decreased functional capacity from a pneumoconiosis, these diseases must first be ruled out. Dyspnea as a symptom of disease has long been recognized by all physicians, and diagnostic aids such as ordinary laboratory procedures, electrocardiographic studies, and other tests are available for diagnosis of complicating diseases with pneumoconiosis. After a definite diagnosis of silicosis has been established, physiological measurements of pulmonary function may be used as an aid to the usual clinical examination in determining the extent of disability.

2. Pathological Physiology and General Principles

A great deal of work on the physiology of respiration has been done in this country and in Europe. Contributions of especial significance have been made by Cournand and his associates and by McCann and the group working with him. These sources have been drawn upon heavily for the material in this section. Cournand and Richards[50] have suggested a simple, yet useful, classification of the types of pulmonary insufficiency and discussed the mechanisms involved in each type. Their grouping is as follows: (*1*) ventilatory pulmonary insufficiency, which is concerned with defective air movement into and out of the lungs; (*2*) respiratory pulmonary insufficiency, which is concerned with defective gaseous exchange between the blood and alveolar air; (*3*) combined ventilorespiratory insufficiency; (*4*) combined cardiopulmonary insufficiency.

Ventilatory Insufficiency. The ventilatory aspect of pulmonary function is largely mechanical. Adequate ventilation is dependent upon the movement of sufficient air into and out of the lungs, that is, upon a breathing capacity great enough to supply the body with the oxygen necessary for its needs. The flow of air to and from the lung is dependent on the chest bellows, regulated by a well-coordinated neuromuscular mechanism, the tracheobronchial pulmonary air passageway, and the state of pulmonary tissue, particularly as related to the amount of elasticity and fibrosis.

The breathing requirement, or the amount of oxygen required at any given

[50] A. Cournand and W. Richards, Jr., *Am. Rev. Tuberc.*, *44*, 26 (1941).

[illegible] with the metabolism, posture, oxygen and carbon dioxide [illegible] emotional or nervous states, and exercise. It is primarily governed by reflex stimulation of the respiratory centers.

The cardinal sign in ventilatory insufficiency is hyperpnea and its concomitant symptom, dyspnea.

Respiratory Insufficiency. The respiratory function is concerned with gaseous interchange between the alveolar air and the blood. This is dependent upon the degree of ventilation of the individual alveoli and the relative number of alveoli that are well ventilated, the number, size, and distribution of capillaries in contact with the alveoli, and the rate of blood flow through these capillaries, as well as the oxygen-carrying capacity of the blood, the gradient of pressure of respiratory gases across the alveolar-capillary partition and the physical properties of this partition.

The cardinal sign in respiratory insufficiency is cyanosis. However, if the insufficiency is slight, cyanosis may not be present and hypercapnia may predominate. In such instances measurements of oxygen removed from inspired air or carbon dioxide added to expired air or of the oxygen saturation of arterial blood may be necessary to determine the actual extent of the respiratory failure.

Cardiocirculatory Insufficiency. The cardiocirculatory function may be affected along with the respiratory and ventilatory functions. The causes of disturbance in the heart and circulation that may be encountered in connection with chronic pulmonary disease are: hypertension in the pulmonary circulation and subsequent right-heart hypertrophy; obstruction to the flow of blood by displacement of the mediastinum, increased pressure in the thorax, or disturbance in the mechanics of breathing; decreased cardiac function due to the effects of anoxia on the heart muscle and cardiac regulatory centers; and independent heart disease of any type.

It must always be borne in mind that patients with heart disease respond to pulmonary function tests similarly to those with pulmonary fibrosis and emphysema.[51] The fact, demonstrated by Enzer, Simonson, and Evans,[52] that recovery of pulse rate during and after exercise is of little value in the segregation of normal persons from those with silicosis may be significant in differential diagnosis of purely pulmonary disease as compared with cardiac disease. However, Enzer himself points out that his tests were performed by subjects who worked until fatigue developed. Enzer's normal subjects actually did more work than his silicotic patients before fatigue occurred, which may account for the lack of difference in the pulse rates. He found, nevertheless, a definite trend toward prolonged recovery time in his patients when certain types of work were done. At any rate there was a greater correlation between lung disease and pulmonary functions than between lung disease and pulse rates after exercise. We believe that information based on some standard form of exercise such as the Master Two-Step Test,[53] which has been

[51] N. L. Kaltreider and Wm. S. McCann, *J. Clin. Investigation*, *16*, 23 (1937).
[52] N. Enzer, E. Simonson, and A. M. Evans, *J. Ind. Hyg. Toxicol.*, *23*, 147 (1945).
[53] A. M. Master and E. T. Oppenheimer, *Am. J. Med. Sci.*, *177*, 223 (1929).

carefully standardized for age and sex, would be helpful in making such an analysis.

A fundamental concept necessary to an understanding of symptoms in any disease is that symptoms develop only when the adaptive mechanism of the body is required to function in a degree beyond that normally required for adaptation. In other words, symptoms appear when the adaptive mechanism is put under strain. This is particularly true in pulmonary disease. As an example, Harrison[54] found that "a person becomes short of breath when his actual volume of breathing becomes more than a certain fraction of his maximum possible volume and the closer the actual volume approaches the maximum possible ventilation, the more severe the dyspnea becomes." Accordingly, measurements that indicate the relative degree of ventilation or aeration required under given circumstances in comparison with the total ability to ventilate or with other known lung capacities are of more value than measurements that indicate merely a particular capacity.

Likewise, as pointed out by Hurtado and Boller,[55] there may be wide normal variations from the median when any one function such as vital capacity or residual air is measured, whereas if this function is taken as a percentage of total capacity the normal variations are much closer to the median. Thus again is demonstrated the value of expressing a capacity in terms of percentage of another capacity.

3. *Terminology*

A serious drawback to the proper comparison of different series of observations on respiratory functions has been the use of different terms and even of different meanings for the same term. It is therefore essential to adopt, if possible, a single nomenclature in order to have a clear understanding of the subject. Christie and Meakins[56] have proposed a fairly satisfactory usage, which we shall adopt here, except for the suggestion of Hurtado and Boller that the term "functional residual air" be replaced by the term "mid-capacity," and with the addition from later authors of terms describing ventilation and its components. The classification as suggested may be summarized as follows:

1. *Residual air* is the amount of air remaining in the lungs after fullest possible expiration.

2. *Reserve air* is the amount of air expired from the mid-capacity position to the maximum possible deflation.

3. *Mid-capacity* is the amount of air remaining in the lungs after a normal expiration. Christie and Meakins speak of this as the resting respiratory level. Hurtado and Boller comment on this as follows: "The term 'functional residual air' is synonymous with this term. It appears to be more convenient, however, to use the term mid-capacity as being more descriptive and more commonly used. The term 'functional residual air' may be easily confused with residual air, or it may suggest that it is a subdivision of the latter. Mid-capacity represents the sum of the residual and reserve air."

4. *Complementary air* is the volume of air inspired from the position of mid-capacity to that of the maximum possible inflation. It includes the tidal air.

[54] K. R. Harrison, *Failure of the Circulation.* Williams & Wilkins, Baltimore, 1935.
[55] A. Hurtado and C. J. Boller, *J. Clin. Investigation*, **12**, 793 (1933).
[56] R. V. Christie and J. C. Meakins, *J. Clin. Investigation*, **11**, 1099 (1932).

5. [illegible] deepest possible expiration. Vital capacity is the sum of the complementary and [illegible] volume.

6. [illegible] is the sum of the residual air and the vital capacity.

7. *Tidal air* is the amount of air moving in or out during quiet breathing. These relationships are shown more clearly in the diagram in Christie and Meaking's "Lung Volume and Its Subdivision."[55]

In recent publications, pulmonary physiologists have stressed the importance of somewhat more dynamic measurements. Cournand and Richards[50] and Kaltreider and McCann[56] have described ventilatory measurements. These, we believe, should be added to the above terminology:

8. *Minute volume* is the volume of air ventilated per minute under any given conditions. This is the same as the breathing requirement.

9. *Maximum ventilatory volume* is the maximum volume of air that can be ventilated per unit of time (usually expressed in liters per minute). This is synonomous with the *maximum breathing capacity* of Cournand and Richards.[50]

10. *Breathing reserve* is the excess of breathing capacity beyond the actual ventilation in any given state, that is, maximum minute ventilation minus minute ventilation. This may be expressed as percentage of maximum ventilation.

11. *Ventilation equivalent for oxygen* is the amount of air ventilated in order to yield 100 ml of oxygen to the body.

4. *Tests for Ventilatory Efficiency*

The ventilatory function is tested by determinations of chest capacity and breathing volume. Probably the most revealing and important measurement is that of maximum ventilatory volume, at least when it is used as a basis for comparison with other measurements. Actually, Peabody[57] has shown that dyspnea is more closely related to vital capacity than to total ventilation.

There are three common methods for the determination of maximum ventilatory volume: (*a*) maximum ventilation in exhausting exercise[51]; (*b*) maximum ventilation with carbon dioxide rebreathing[58]; (*c*) maximum ventilation by performing maximum ventilation effort, using voluntary rate and volume.[50]

The method employed by Kaltreider and McCann appears to give rather uniform results. They measured the air breathed from a spirometer during the last one and one half minutes of exhausting exercise. A test requiring this much co-operation on the part of the patient, no matter how valid its results, is somewhat beyond the scope of what may be employed in general in the examination of industrial patients, whose subjective reactions are extremely variable. Furthermore, the results obtained by this method (average 71 liters per minute for normal male subjects) are somewhat lower than those obtained by the voluntary forced breathing test of Hermannsen[59] as adapted by Cournand and Richards[50] (average 154 liters per minute for males, 100 for females).

The method of carbon dioxide rebreathing is rather difficult to control in the ordinary outpatient clinic and has the added disadvantage of the inaccuracies that develop during rapid breathing in a closed system.

[57] F. W. Peabody, *Harvey Lectures*, **12**, 248 (1916–17).
[58] R. Goiffon, R. Parent, and J. Waltz, *Ann. méd.*, **35**, 362 (1934); **36**, 57 (1934).
[59] J. Hermannsen, *Z. ges. exptl. Med.*, **90**, 130 (1933).

The voluntary rapid- and deep-breathing method described first by Hermannsen[59] and later adapted by Cournand, Richards and Darling[60] and by Wright[60a] also depends on effort by the patient. There is, however, greater likelihood of obtaining reliable results by this method than by those in which maximum exhausting effort is required. The technique has the advantage of being rather simple to perform, and according to the author its results are readily reproducible. In this method maximum deep breathing is measured by having the patient breathe from a spirometer as deeply and as rapidly as possible but with emphasis on depth so that the individual breaths are somewhat short of vital capacity. The patient is tested again for rapid breathing. This time he breathes as rapidly as possible and as deeply but the emphasis is on rapidity. In these tests the author states that a surprisingly uniform pattern is produced. The breathing is carried on for 12 to 15 seconds.

As a substitute for spirometry the exhaled air may be collected in a Douglas bag.[60a] The equipment essential for this technique includes only a close-fitting face mask, a two-way valve of low resistance, a two-way cock, some large rubber tubing, a large Douglas bag, and a flowmeter such as may generally be found in a gas laboratory. The subject inhales room air as deeply and rapidly as possible, but with emphasis on depth, and exhales directly into the Douglas bag for a period of thirty seconds. The volume of air in the bag is then measured. It is desirable to have several men perform this test in a group so that maximum effort can be fostered by competition. Results obtained with this type of maximum effort are ordinarily more consistent than are results obtained by measurement of tidal air, in which psychic factors play a dominant role.

When maximum ventilation is used as a basis for comparison, notation should be made of the method used in determining it so that the results may be compared with those found in the literature.

The functions usually compared with total breathing capacity are vital capacity, minute volume and pulmonary reserve, at a given amount of work or at dyspnea.

Vital Capacity. Peabody[57] in a study of dyspnea in patients with heart disease, and Hurtado and his associates[61, 62] studying patients with pulmonary fibrosis and emphysema, showed that the degree of dyspnea was closely correlated with the vital capacity. Harrison and his co-workers[63] found that the degree of dyspnea in heart disease was more closely related to the expression $\frac{\text{total ventilation}}{\text{vital capacity}}$ than to either of these factors alone, and this has been corroborated by Kaltreider and McCann.[51] Vital capacity can be determined simply by spirometry. Various formulas

[60] A. Cournand, D. W. Richards, Jr., and R. C. Darling, *Am. Rev. Tuberc.*, *40*, 487 (1939).

[60a] G. W. Wright, *personal communication*, 1947.

[61] A. Hurtado, W. W. Fray, N. L. Kaltreider, W. D. W. Brooks, and W. S. McCann, *J. Clin. Investigation*, *13*, 102 (1934).

[62] A. Hurtado, N. L. Kaltreider, and W. W. Fray, W. D. W. Brooks, and Wm. S. McCann, *J. Clin. Investigation*, *14*, 81 (1935).

[63] T. R. Harrison, F. Turley, E. Jones, and J. A. Calhoun, *Arch. Internal Med.*, *12*, 833 (1931).

have also been devised, based on height, weight, body surface area, and x-ray measurements in correlation with certain chest measurements.[49] The closest correlation of predicted values with actual findings was obtained by Hurtado and Fray with the chest measurements and x-ray findings. However, it would appear somewhat simpler and more accurate to use spirometric methods for routine work inasmuch as the equipment is needed for determinations of total ventilation. Kaltreider and McCann found that the vital capacity, the arterial saturation of the blood, and the ability to expand the chest decreased as dyspnea increased. Dyspnea is experienced when the expression $\frac{\text{total ventilation}}{\text{vital capacity}}$ is greater than 51 (with total ventilation determined by exercise method), and when this value is exceeded at low levels of work it is an indication of pathological dyspnea. The maximum ventilation is only roughly proportional to vital capacity in normal subjects, but in patients with pulmonary disease the relation is closer.

Minute Volume. The significance of minute volume is rather striking when it is considered as the breathing requirement of a person at a given time and under given circumstances relative to rest, work, emotional state, and metabolism. This concept of minute volume is ably developed by Cournand and Richards.[50] Minute volume may be measured with a spirometer or by collecting air in a Douglas bag. This measurement is important because from it and the maximum ventilation we obtain the breathing reserve (Maximum Ventilation minus Breathing Requirement). Minute volume may be compared directly with maximum ventilation as a percentage, i.e., (minute ventilation)/(maximum minute ventilation) x 100. In studies by Kaltreider and McCann[51] it was found that dyspnea was first noted by normal individuals at values between 46 and 70 per cent, with an average of 59 per cent. In patients with pulmonary disease the percentage often may exceed this value and at rest may even be well above it.

Pulmonary Reserve. The excess breathing capacity beyond the actual ventilation (minute volume) in any given physical state is the breathing reserve. Cournand and Richards have discussed this concept as follows:

The maximum breathing capacity in each subject is a fixed value and the breathing reserve varies inversely with the breathing requirement. For comparative purposes this may be expressed in per cent of maximum breathing capacity. Thus, in a subject whose maximum breathing capacity is 150 liters per minute, and ventilation at rest is 5 liters per minute, the breathing reserve is 150 − 5 = 145 liters; and the ratio

$$\frac{\text{breathing reserve}}{\text{maximum breathing capacity}} \times 100 \text{ at rest} = \frac{145}{150} \times 100 = 96.6 \text{ per cent.}$$

Similarly, in the same subject, if the ventilation during exercise is 25 liters per minute, the reserve is 150 − 25 = 125 liters per minute and the ratio is 83.3 per cent. In 37 patients out of 105 who were given a standard exercise test without

[49] A. Hurtado and W. W. Fray, *J. Clin. Investigation*, 12, 807 (1933).

experiencing dyspnea, the reserve (even in the first minute following exercise) was 73 per cent of the maximum breathing capacity. Although dyspnea was complained of by a few patients when the reserve was 75 per cent of maximum breathing capacity, the threshold of dyspnea was almost always between 60 and 70 per cent of the maximum breathing capacity. It is of interest if possible to predict, from findings in a patient at rest, whether dyspnea will occur easily. These authors found that if

$$\frac{\text{breathing reserve}}{\text{maximum breathing capacity}} \times 100 \text{ at rest is above } 93$$

no dyspnea will develop with the standard exercise used.

5. *Tests for Respiratory Insufficiency*

The respiratory function is best evaluated by determining the oxygen used from inspired air, the carbon dioxide given off in expired air, or the oxygen content of arterial blood.

Ventilatory Equivalent for Oxygen. The oxygen used is usually determined as the ventilatory equivalent, that is, the number of liters of air breathed in order to supply 100 ml. of oxygen. Such measurements can be made by analyzing samples of expired air for their carbon dioxide and oxygen content by a method such as that described by Van Slyke and Sendroy,[65] or a fairly accurate determination may be made by rebreathing an air–oxygen mixture from a closed system spirometer, removing the carbon dioxide chemically and measuring the oxygen used by the decrease in the spirometer content. There is considerable variation among normals in oxygen consumption, and it is probable that oxygen saturation of arterial blood is the better indication of aeration of blood as it passes through the lungs.

Oxygen Saturation of Arterial Blood. When lung capillary perfusion by oxygen is satisfactory there is usually a high percentage of oxyhemoglobin in the blood and this usually does not fall below 95 per cent even with severe exercise. (See Oxygen, Volume II.) A decrease in oxygen saturation of arterial blood may be due to various causes. In chronic pulmonary disease anoxemia may be caused by the flow of blood through capillaries that are unventilated or poorly ventilated. In heart disease pulmonary congestion may produce inadequate capillary ventilation. Also in heart disease the general circulation may be so retarded that the blood is seriously depleted of oxygen when it reaches the lungs. Determinations of the oxygen and carbon dioxide contents may be performed on the Van Slyke apparatus. This is, of course, somewhat too tedious and time-consuming for routine work in industry. Recently there has been developed a spectrophotometric method[66] for determining the oxyhemoglobin count of blood in the ear lobe. The procedure is simple and is apparently within the same degree of accuracy as chemical methods. It might well be adapted for investigations into blood oxygenation as there is a time lag of only about five seconds and continuous readings may be obtained.

[65] D. D. Van Slyke and J. Sendroy, Jr., *J. Biol. Chem.*, **95**, 509 (1932).
[66] G. A. Millikan, *Rev. Sci. Instruments*, **13**, 434 (1942).

6. Exercise Tests in General

Tests depending upon either maximum effort or moderate effort are employed in measuring pulmonary or cardiac efficiency. Maximum effort or effort to fatigue and dyspnea are sometimes used and have their place in the study of pulmonary physiology. For a study of the effects of maximum effort, the most satisfactory equipment for measuring the amount of work performed is the bicycle ergometer. However, for most investigations in industrial clinics moderate exercise may give valuable information as to the state of the patient. In moderate exercise the patient may be asked to squat, hop fifty times, lift a weight repeatedly, etc. None of these tests, however, is weighted to take into consideration normal variations in age and sex. In this respect the Master Two-Step Test[67] has definite advantages as it has been well standardized at least for cardiac function as to age and sex, is simple to perform, and is almost quantitative in terms of foot-pounds of work per given time.

7. Summary

An attempt should be made by plant physicians and those charged with the management of workers in industries in which silicosis is a hazard to determine actual disability in patients. By the use of simple respiratory equipment, such as a slightly modified basal metabolism apparatus,[60] the vital capacity and maximum pulmonary ventilation can be determined. The breathing requirement at rest and in standard exercise can be measured with a Douglas Bag or a Tisot Spirometer. Such examination should be possible within a reasonable time even for a busy industrial physician, especially if clerical help is utilized for occupational history taking and nurses are available for the routine portions of the examination. If ventilation is measured by modern methods commonly employed industrially by physicists, as deflection of a wire or alterations in heat conductivity, pneumotachograms[65,65a] can be included in the data without requiring any additional time. In this way considerable information on a new diagnostic procedure for lung and chest diseases (measurement of changes in rate of air flow during the different phases of the respiratory cycle) might be obtained. True, the appraisal of disability by pulmonary function tests is no substitute for appraisal by sound experienced clinical judgment; but it furnishes records for year by year comparison, and it gives definite facts on which to base medical-legal opinion as to disability. In any consideration of disability in pneumoconiosis one fundamental concept must be emphasized. From an economic and sociological point of view it may be wrong to consider a patient as having disability if experience demonstrates that he is capable of doing his daily work. This is especially true if conditions are such that aggravation of an existing condition is unlikely.

[67] A. M. Master and E. T. Oppenheimer, *Am. J. Med. Sci.*, **177**, 223 (1929).

[65] A. Fleisch, "Neuere Ergebnisse uber Mechanik und propriozeptive Steuerung der Atmungsbewegung." *Ergebn. d. Physiol.* **36**, 249 (1934).

[65a] L. Silverman, *J. Ind. Hyg. Toxicol.*, **28**, 183 (1946).

II. CONTROL OF SILICOSIS IN INDUSTRY

1. Engineering Control

The fundamental basis for silicosis prevention is engineering control. Enclosed processes, exhaust ventilation, wet methods, and other engineering techniques can be used to insure safe concentrations of dust in the air breathed by workmen. Engineering control of dusts is discussed in Chapter Ten.

Personal protective equipment such as filter respirators and air-line or supplied-air respirators (See Chapter Fourteen) will under some circumstances afford protection, but such devices are poor substitutes for air sufficiently free of dust to prevent the development of pneumoconioses.

2. Medical Control

All employees with potential exposure to dangersous concentrations of free silica should be given annual medical examinations. These should include x-ray films of good quality competently interpreted, clinical examinations, laboratory examinations in cases where it is necessary to determine infection or concomitant disease such as cardiocirculatory dysfunction, and pulmonary function tests. When there is a question of tuberculous infection, the patient should be examined with sufficient frequency and detail to determine the exact status of the disease.

The type of routine x-ray examination used will be a major factor in determining the cost of the program to the medical department. Most roentgenologists feel that the standard 14-in. × 17-in. film is necessary for definite diagnosis. However, as a screening procedure, several less expensive films are available. Paper film is sometimes substituted for ordinary transparent film. Roentgenograms of this type usually show too little detail to be satisfactory even as a screening process. Roentgenograms of the 35-mm. or 4-in. × 5-in. size, on the other hand, may be used rather successfully for differentiating normal from pathological chests. The 35-mm. film is cheaper and requires less time for developing than the 4-in. × 5-in. It must, however, be used purely as a screening device, as there is not sufficient detail for diagnosis of abnormal findings. Films of the 4-in × 5-in. size frequently may be interpreted directly, necessitating the re-x-raying of fewer employees.

The approximate relative cost of the different sizes of film in the amounts used by large industries is as follows:

Size of film	Cost per patient (approx.)
35-mm. film	4½¢
4-in. × 5-in. film	7 ¢
14-in. × 17-in. film	80 ¢

The number of employees to be examined would alter this but the relative costs should remain fairly constant.

The value of the routine chest survey in the control of silicosis cannot be overstressed, and if photoroentgenograms are used for screening purposes costs may be reduced to a level that can be absorbed by most industries.

The question as to whether or not a workman should continue at his [illegible] is a serious responsibility and requires great care on the part of the examining physician as well as a thorough understanding of working conditions in the particular occupation and an appreciation of public health and moral responsibility both to the individual and to his associates. As a guide for management of the patient with silicosis or tuberculosis, Lanza[69] has formulated the following rules for the various general groups seen:

"I. Partial disability due to silicosis without infection:

"(a) May continue at usual occupation, if environment is satisfactorily controlled as regards dust concentrations and tuberculosis contact;

"(b) Degree of fibrosis and rate of development may require that the individual seek less arduous employment and avoid even minimal exposures to silica-containing atmosphere.

"II. Partial disability due to silicosis with complicating pulmonary infection:

"(a) Primary silicosis. Infection mild and nontuberculous. In most instances, after successful treatment of nontuberculous complicating infection, may be allowed to return to usual work, provided environment under which they are working is safely controlled.

"(b) Primary silicosis with pulmonary tuberculosis as complicating infection. Those individuals with silicosis who develop pulmonary tuberculosis are considered totally unfit for further employment in an industry affording even minimal exposures to free silica. This disability is obviously total so long as active tuberculosis is present. Following successful treatment, these cases regain their health to the point where they can be safely employed at other work."

3. Aluminum Prophylaxis and Therapy

The use of aluminum for the prevention of silicosis was first reported by Denny, Robson, and Irwin[70] in 1937. At about the same time similar investigations were undertaken by Gardner and his associates. Since that time considerable attention has been focused on aluminum and its compounds both in laboratories and in clinics.

Tabershaw and Tebbens[71] summarized the principles that have evolved from laboratory work on both finely divided metallic aluminum and amorphous hydrated alumina as follows: If alumina and silica localize in the same phagocytic cell the effect of the silica is neutralized. Further progression of the tissue reaction characteristically produced by silica is then arrested. Mature silicotic nodules become static, and immature lesions (inflammatory and early fibrous changes) are resolved. Aluminum may remain in the tissue and protect the individual for a long period—over a year. Aluminum does not ordinarily produce toxic effects in the tissues, although it appears to increase the susceptibility of experimental animals to tuberculosis when large doses are given.

Occasionally, allergy to aluminum has been reported,[72] but this is apparently a rare occurrence. Denny, Robson, and Irwin have preferred to use metallic aluminum, but Gardner has found that certain hydrated aluminas are more satisfactory. McIntyre Research Limited, the United States agency created for licensing the use

[69] A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938, p. 56.

[70] J. J. Denny, W. D. Robson, and D. A. Irwin, *Can. Med. Assoc. J.*, *37*, 1 (1937); *40*, 213 (1939).

[71] I. R. Tabershaw and B. D. Tebbens, *J. Ind. Med.*, *14*, 709 (1945).

[72] L. H. Cotter, *J. Ind. Hyg. Toxicol.*, *26*, 7 (1944).

of aluminum by physicians for therapeutic or prophylactic purposes, makes only metallic aluminum available. Some investigators, notably Hannon,[73] have reported marked success in the treatment of patients with severe disability. However, when purely objective criteria have been used to measure disability the results have been somewhat less striking. Gardner, Dworski, and Delahant[74] have suggested that variations in response to aluminum therapy may be due in part to the nature of the dust that has caused the silicosis. They suggest that dust which is primarily free silica may react with aluminum more readily than dust which contains high percentages of iron. The use of aluminum generally in industry for the therapy or prophylaxis of silicosis should be contemplated only with the utmost caution.

I. ASBESTOSIS

Asbestos is a hydrated magnesium silicate. More than 90 per cent of the raw mineral used in this country and Great Britian is produced in the Canadian chrysotile mines. Asbestos is used in two general types of manufacturing processes. It may be used either by itself or mixed with other insulating materials such as diatomaceous earth for fireproofing, packing, or insulating; or it may be combined with cotton and woven as a textile for fireproof and heat-resistant clothing and other substances. Lanza[75] estimates that there are about 10,000 persons exposed to asbestos in the United States. Most observers feel that the incidence of asbestosis in American asbestos workers is quite low. Asbestosis has, however, been reported more frequently in England and Canada.

Lanza, McConnell, and Fehnel[76] concluded from their study of asbestosis that: Prolonged exposure to asbestos dust causes pulmonary fibrosis different from that produced in silicosis and demonstrable by roentgenogram. Clinically it appears to be milder than silicosis.

Definite cardiac enlargement frequently was found to be associated with asbestosis.

A predisposition to tuberculosis, due to asbestos dust, was not indicated although it was not known to what extent asbestosis may add to the mortality from pneumonia and acute nontuberculous pulmonary infections.

Symptoms. The onset of asbestosis, as of silicosis, is slow although symptoms are apt to be somewhat more marked than in silicosis. In silicosis there may be marked x-ray findings with little complaint of symptoms, whereas the opposite is likely to be the case in asbestosis. As in silicosis, dyspnea is the cardinal symptom. Anorexia occurs frequently in advanced stages, and cyanosis and clubbing of the fingers are apt to appear with greater constancy in asbestosis.

Pathology. The fibrosis in asbestosis is diffuse and tends to predominate in the basal portions of the lungs in contrast to the generalized nodular fibrosis of silicosis

[73] J. W. G. Hannon, *Trans. Can. Inst. Mining Met.*, **48**, 180 (1944).
[74] L. U. Gardner, M. Dworski, and A. B. Delahant, *J. Ind. Hyg. Toxicol.*, **26**, 211 (1944).
[75] A. J. Lanza, *J. Am. Med. Assoc.*, **106**, 368 (1936).
[76] A. J. Lanza, W. J. McConnell, and J. W. Fehnel, *U.S. Pub. Health Repts.*, **50**, 1 (1935)

with a predominance in the upper portions of the lung. Bronchiectasis and bronchiolectasis are frequent, especially in the more fibrous portions. Whereas the proliferation of fibrous tissue is caused by chemical action in silica exposure, it is induced by mechanical action in asbestos exposures. Gardner[77] found that the fibrosis-producing character of asbestos could be almost eliminated by grinding the fibers so that no particles more than 2 μ in length were present. As previously mentioned, asbestos is classified among the inert dusts.

Microscopic Anatomy. Johnstone[78] described the microscopic appearance of the lungs somewhat as follows: in the early phases of the disease there is thickening of the alveolar septa which results from fibroblastic proliferation. The alveolar spaces contain numerous phagocytes. With progression of the disease fibrosis becomes more marked; the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few alveoli that remain in the area of fibrosis are lined with low cuboidal epithelium giving them an almost glandular appearance.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures described first by McDonald.[79] These bodies are 20 to 100 μ in length and are bulbous on one or both ends so that they appear club- or dumbbell-shaped. They are brownish in color, do not stain, and give a Prussian-blue reaction for iron. Simson[80] has produced these bodies in guinea pigs by experimental exposure to atmosphere containing asbestos. Lynch[81] concludes that these "curious bodies" signify exposure to asbestos dust but do not necessarily indicate asbestosis.

X-Ray Examination. For an excellent review of roentgenographic findings in both silicosis and asbestosis the reader might well refer to Pendergrass.[82] Characteristic differences in the roentgenographic findings in silicosis and asbestosis are tabulated below:

Asbestosis	Silicosis
Fibrosis diffuse (film may have ground glass appearance from pleural involvement).	Fibrosis nodular.
Findings may be either bilateral or unilateral.	Findings characteristically bilateral.
Lesions largely in the lower one half or two thirds of the lung fields.	Lesions predominately in the upper two-thirds of the lung fields.
Emphysema in upper portion of lung fields.	Emphysema in lower portion of lung fields.

Control. Prevention of asbestosis depends entirely upon preventing exposure to concentrations of dust sufficiently high to produce the characteristic reaction.

[77] L. U. Gardner, *Ind. Med.*, **9**, 45 (1940).
[78] R. T. Johnstone, *Occupational Diseases*. Saunders, Philadelphia, 1942.
[79] S. McDonald, *Brit. Med. J.*, **2**, 1025 (1927).
[80] F. W. Simson, *Brit. Med. J.*, **1**, 885 (1928).
[81] K. M. Lynch, *J. Am. Med. Assoc.*, **109**, 1947 (1936).
[82] E. P. Pendergrass, "Roentgen-Ray Diagnosis in Silicosis and Asbestosis," in A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

Dreessen, DallaValle, Edwards, Miller, and Sayers[83] have found evidence to indicate that 5,000,000 particles per cubic foot of air is a satisfactory figure for the maximum permissible atmospheric concentration of asbestos to which workers may be exposed. Definite permissible standards, however, have not been established.

VI. Dust Causing Minimal Fibrosis or No Fibrosis

Gardner[77] has pointed out that any known inorganic dust other than free silica and the asbestos silicates may produce nonspecific dust reactions, as described on page 486. X-ray markings may be found in other conditions such as chronic infection and heart disease. No interference with pulmonary function or disability is produced by dust of this type and there is no influence on susceptibility to tuberculosis.

A. SILICATES

McCord,[84] from a number of sources, has compiled a list of the commonly used silicates, which is presented here:

Olivine—a magnesium silicate widely present in all basic rocks. Many varieties of this stone are known.

Calcium silicate—nonexistent in nature, but a common product of industry; may be found at blast furnaces and in production of cement and hydrated limes. Other silicates of calcium are known.

Willemite—a zinc silicate (rare).

Sodium silicate (meta)—the well-known water-soluble silicate, commonly called water glass. This is widely used in industry, notably as a filler in soaps. This is the only crystalline silicate of sodium, the others being amorphous glass.

Tremolite—a magnesium-calcium silicate.

Asbestos—tremolite, actinolite, chrysotile, or amianthus, all of which are essentially magnesium silicates.

Jade—another form of magnesium-calcium silicate.

Crocidolite—"blue asbestos." It is a sodium iron silicate.

Talc—a hydrated magnesium silicate with extensive industrial application.

Soapstone—a form of talc.

Agalite—a variety of talc resembling asbestos. It is used in the coating of paper, and as a paper filler.

Meerschaum—also called sepiolite. It is closely akin to talc.

Serpentine—hydrated silicate of magnesium. It is a common building stone.

Sillimanite—aluminum-containing silicate. The source of many stone tools of the stone age

Topaz—a semiprecious stone of silicate origin. Chemically it is an aluminum fluosilicate.

Fuller's earth—a hydrated silica-aluminum compound, associated with ferric oxide.

Kaolin (kaolinite)—many related silicates of mixed constituency.

Clays—hydrated aluminum silicates containing iron. Titanium, quartz and mica, are likely to be present in clays.

[83] W. C. Dreessen, J. M. DallaValle, T. I. Edwards, J. W. Miller, and R. R. Sayers, *U.S. Pub. Health Bull.* No. *241* (1938).

[84] C. P. McCord, *Ind. Med.*, *2*, 4 (1933).

Corundum—a silicate of aluminum. It is widely used as a [illegible] and [illegible] varieties, including blues, reds, greens, yellows, violets and white.

Mica—a large group of silicates of [illegible]. Mica is characterized by their well-known tendency for cleavage in [illegible]. Used in industry, mainly in electrical work for insulation.

Garnet—complex silicates of aluminum, iron, calcium, and magnesium. Besides being a semi-precious gem stone, garnets are used as watch bearings, in gem cutting, polishing, etc.

Feldspar—a large group of aluminum silicates entering into many minerals, for example granite.

Permutite—a sodium-aluminum silicate of complex structure, much used in water softening.

Lava—mixed silicates of volcanic origin.

Pumice—volcanic, glassy, spongy lava. It is primarily used as a polishing agent.

Shale—a loose term applied to clays and other silicates that have been subjected to high pressures in the earth.

Slate—a substance of clay or shale origin, that has been subjected to high pressure and has metamorphosed. It contains or may contain much free silica.

Slags—Products of metal blast furnaces that contain native impurities as well as minerals such as dolomite introduced as fluxes, etc. Almost all forms of silicates may be included. Free silica may be present.

Silicon carbide—carborundum, a synthetic mineral.

Silundum—another form of synthetic silicate, akin to carborundum in chemical structure.

Fibrox—silicon oxycarbide. It closely resembles carborundum and is somewhat similarly made.

It may be stated with some certainty that none of these dusts, unless in combination with free silica, will produce nodular fibrosis; and proof of disability from breathing such dusts is lacking.

B. NONSILICEOUS DUST

Nonsiliceous dusts such as calcite, calcium carbonate, gypsum, limestone, Portland cement, and pyrolusite dusts have been listed in the classification of Miller and Sayers[85] as among those causing an absorptive tissue response and, accordingly, are not considered as producers of pneumoconiosis.

Apparent X-ray Nodulation without Fibrosis. Only brief mention will be made of baritosis and siderosis. Pendergrass[82] reports having seen several patients exposed to barium dust with widespread dense nodulation typical of that seen in simple silicosis. The individuals examined were symptom-free and not incapacitated. Sander[86] has described a similar condition caused by the inhalation of iron oxide fumes at welding operations. Exposure to iron dust or fume may produce an x-ray picture characterized by generalized nodulation. Autopsy specimens have revealed that the nodules are collections of iron with no tissue reaction or fibrosis.

[85] J. W. Miller and R. R. Sayers, *U.S. Pub. Health Repts.*, **56**, 264 (1941).
[86] O. A. Sander, *J. Ind. Hyg. Toxicol.*, **26**, 79 (1944).

VII. Dust Causing Chemical Irritation

It is hardly necessary to enumerate in detail the chemical irritants. These are usually either alkaline or acid in reaction. Examples of the alkaline group are hydroxides, cement, and soaps; of the acid group, acids, fluorides, and chromates.

Such chemicals may irritate the skin causing erythema or burns, even to the point of necrosis. Injury to the conjunctivae may occur with lacrimation and conjunctivitis; or this type of dust may produce inflammation of the nasal mucosa, sometimes to the extent of ulceration of the nasal septum. These substances also may irritate the remainder of the respiratory tract sufficiently to induce coughing and, in more severe exposures, to cause bronchitis or pneumonitis.

There is little evidence to suggest that prolonged exposure to irritant dust, in concentrations that are too low to produce symptoms at the time of exposure, will cause chronic disease or increase susceptibility to infection. Local irritation by dust may result from purely mechanical as well as chemical characteristics. Mechanical injury may result from breathing long sharp fibers as of lint or other vegetable materials.

A severe and even fatal pneumonitis may follow the inhalation of cadmium dust or fume. This is particularly important since the dust does not produce any immediate irritation or warning symptoms. A few hours following exposure symptoms of gastritis may develop and subsequently pulmonary edema and pneumonitis may occur.

Beryllium has recently been considered as a cause of occupational disease because of the occurrence of acute and chronic manifestations in beryllium workers. The acute phase has been officially named[86a] "Acute pneumonitis of beryllium workers" and the chronic phase has been named[86a] "Pulmonary granulomatosis of beryllium workers."

The acute reactions are characterized by dermatitis, conjunctivitis, irritation of the upper respiratory tract and, in some instances, pulmonary edema. Such lesions may be associated with exposure to the acid compounds of beryllium and may arise in beryllium extraction plants.

The chronic lesions in the lungs are granulomatous. There is associated thickening of the walls of the air spaces and subsequent interference with gaseous exchange, producing dyspnea. Cor Pulmonale may develop. Chronic cases have appeared in persons engaged in the manufacture of fluorescent lamps and in the casting of beryllium alloys in which the percentage of beryllium was relatively high. The subject has been well reviewed in the Saranac Symposium on the Beryllium Problem, Sept. 29, 1947.

VIII. Dust Causing Systemic Poisoning

It is not possible to list here the thousands of minerals, drugs, and chemicals that may exert a harmful effect on the body. One need only recall the great surface

[86a] O. A. Sander, *Summary of Saranac Symposium on the Beryllium Problem*, Industrial Hygiene Foundation, 1948.

area of the lungs to realize that they may act as a gateway to the body for entrance of any material that is suspended in the air. If this suspended material is toxic, pathological reactions may result, providing sufficient dust is inhaled to cause injury.

Dust counts ordinarily are not used to measure the degree of air contamination by poisonous substances. With a toxic dust, air contamination is usually defined in terms of weight per unit volume, such as milligrams per 10 cu. meters or milligrams per cubic meters (10 cu. meters is frequently used as the unit of air because this volume roughly approximates the average lung ventilation during an eight-hour working day). Such measurements correspond more closely to measurements used in pharmacological practice. Large particles of a toxic dust may be of greater importance than are large silica particles since the former may be absorbed, in some instances, from the mucous membranes of the upper respiratory tract.

Perhaps the classical example of a dust or fume causing systemic poisoning is lead (see Chapter Twenty-One, Volume II).

IX. Dust Causing Allergic Manifestations such as Dermatitis, Hay Fever, and Asthma

The layman who has consulted his doctor for allergy tests is well aware of the almost innumerable kinds of dust that may cause hay fever, ranging from pollens to house dust or chicken feathers. Since this is not the place for a discussion of immunology, it is sufficient to say that lacrimation, watery nasal discharge, asthma with its difficult expirations, or dermatitis may develop when a person inhales or contacts a material to which he is sensitive. This allergy is usually the result of a previous sensitizing dose or doses and symptoms do not develop, as a rule, until some time has elapsed following the sensitizing exposure (days or weeks). It is probable that any organic dust may be allergenic. Habeeb[87] has attributed bronchial constriction in silicotic patients to silica allergy. This conclusion is based largely on his own findings of eosinophilia in patients with silicosis, and the *in vitro* evidence of bronchial constriction by colloidal silica.[88] A high incidence of eosinophilia has not, however, been found associated with silicosis by other authors. Gardner[89] has reported attempts to use silica on skin tests but was forced to conclude that any reaction was the result of the primary toxicity of silica rather than of allergy to silica. In the light of these findings, further proof would be necessary to show that symptoms associated with silicosis are allergic manifestations.

X. Dust Causing a Febrile Reaction (Acting in an Unknown Manner, Possibly as an Allergen)

Examples of febrile reactions caused by inhalation of dust or fume are metal-fume fever, which is discussed under metallic poisons (Vol. II), and "cotton fever."

[87] W. J. Habeeb, *Ohio State Med. J.*, *41*, 1101 (Dec. 1945).
[88] G. F. Filley, J. G. Hawley, and G. W. Wright, *J. Ind. Hyg. Toxicol.*, *27*, 37 (1945).
[89] L. U. Gardner, *personal communication.*

Workers exposed to zinc oxide fumes, as in the welding or burning of galvanized iron, are subject to a peculiar reaction, consisting primarily of chills and fever occurring some hours after excessive exposure. Details of this reaction are discussed elsewhere, but there is some evidence to suggest that it may be similar to the foreign protein type of reaction.

A similar reaction is found in cotton mill workers, in whom a condition known as "cotton fever" or "Monday fever" occurs. Employees experience a sudden attack of coughing with breathlessness following exposure to cotton lint, after a week end away from the work environment. This reaction is of fairly short duration, and affected persons usually show no further trouble during the week but may have a recurrence of the same condition on the following Monday. Eventually, though, they suffer continually and may have to give up work. Prausnitz[90] has shown that this condition results from allergy to the protein fraction of cotton dust. The febrile response described may be a different entity from the similar condition reported first by Neal, Schneiter, and Caminita[91] and by Ritter and Nussbaum.[92] They found symptoms similar to a cold, with fever and complaints of sinus trouble, occurring in the first few weeks of work rather than consistently on Mondays. The cause of this malady is described as an endotoxin liberated by the bacterium Aerobacter cloacae which grows in the cotton. A similar condition may occur in hemp handlers and those exposed to bagasse.

XI. Summary

The fundamental principles of anatomy and physiology necessary to an understanding of the pneumoconioses have been discussed. Dust has been considered as a cause of extensive pulmonary fibrosis, minimal or no pulmonary fibrosis, chemical irritation, systemic poisoning, allergic reactions, and febrile responses. An attempt has been made to present pertinent data on these subjects in a way that will help engineers and industrial hygienists to understand the physiological problems involved in dust diseases, and in addition to give sufficient medical data to assist physicians in diagnosis and examination of employees exposed to dust.

[90] C. Prausnitz, *Med. Research Council (Brit.), Special Rep. Series* No. *212*, (1936).
[91] P. A. Neal, R. Schneiter, and B. H. Caminita, *J. Am. Med. Assoc., 119*, 1074 (1942).
[92] W. L. Ritter and M. A. Nussbaum, *Ind. Med., 13*, 966 (1944).