A Resource Guide for

Medical Overview Lectures

Spring 2009

Table of Contents

1 Introduction

This guide is intended to provide teaching materials that will be used in conjunction with lectures provided by Eugene Schwartz, MD. The purpose of the Dr. Schwartz's lectures and training is to provide an overview of medical information that may assist the claims examiners and hearing representatives in adjudicating cases. Dr. Schwartz will provide guidance on reading physicians' reports and narratives regarding Energy Employees Occupational Illness Compensation Program Act (EEOICPA) claimants.

Medical Resource materials, acronym lists and medical definitions are included in this teaching guide. Web references are also provided in the form of web links. This material is intended for training purposes only and should not be construed for use in adjudicatory matters or for application to the assessment of any individual case. The evaluation of medical evidence should only be performed by a district medical consultant (DMC).

2 Web links

2.1 Web links in the DMC Handbook

The following web links are taken from the DMC Handbook found on the shared directory.

1. National Library of Medicine

   http://www.pubmed.gov

2. ATSDR Toxicological Profiles

   http://www.atsdr.cdc.gov/toxpro2.html

3. National Toxicology Program (NTP) – Report on Carcinogens (RoC)

   http://ntp.niehs.nih.gov/?objectid=EF215565-F1F6-975E-7BEF7505A220D573

4. International Agency for Research on Cancer (IARC) – List of all agents evaluated

   http://monographs.iarc.fr/ENG/Classification/crthallalph.php

5. NIOSH

   http://www.cdc.gov/niosh/pubs/type.html

   Criteria Documents

   http://www.cdc.gov/niosh/pubs/criteria_date_desc_nopubnumbers.html

   Current Intelligence Bulletins

   http://www.cdc.gov/niosh/pubs/cib_date_desc_nopubnumbers.html

6. OSHA

   http://www.osha.gov/html/a-z-index.html#B

7. Toxnet – Toxicology Data Network

   http://toxnet.nlm.nih.gov/

8. Hazmap

http://hazmap.nlm.nih.gov/

http://www.haz-map.com/

Brown, JA. "An internet database for the classification and dissemination of information about hazardous chemicals and occupational disease." Am J Ind Med 51:428-435, 2008.

2.2 Other helpful links

The following links are for training purposes only.

The International Labour Organization Classifications:
http://www.ilo.org/public/english/bureau/stat/class/index.htm

The National Cancer Institute:
http://www.cancer.gov/

3 DMC Handbook Section II – Medical Issues

A. ROLE OF THE DMC

District Medical Consultants (DMCs) **assist** the DEEOIC by reviewing and evaluating the medical evidence of record and providing medical opinions regarding various aspects of selected compensation cases. DMCs do not review every case, rather medical input from DMCs is sought for selected cases identified by CEs. Such input may include:

- Causality issues involving the work relatedness of a given disease, the role of the covered illness in the death of a claimant; the appearance of secondary or consequential diseases or injuries, etc.
- The explanation of treatment modalities, the interpretation of clinical test results and the clarification of other physicians' reports.
- Determining the level of impairment in a given case in accordance with the AMA's Guides to the Evaluation of Permanent Impairment and DEEOIC's guidance.
- Assessing an individual's ability to work.

Ideally the medical opinion should be provided by a qualified physician with expertise in treating, diagnosing or researching the illness claimed to be caused or aggravated by the alleged exposure.

The DMCs' medical reports are evidence that enable the program's claims examiners (CEs) to reach adjudication decisions regarding causality, and/or impairment in compensation cases. Because of its programmatic and legal constraints, DEEOIC expects these medical opinions to be solidly based on the facts as accepted by the CE and expressed in the Statement of Accepted Facts (SOAF) and on state-of-the-art medical knowledge. Above all, these opinions should be as objective as possible. There will be instances where the DMC will have to make a determination based upon historically incomplete, vague, or contradictory evidence. In these instances, the DMC will have to accept the facts as provided by the CE and formulate a medical opinion based upon what is provided. It should be noted that the DMC's opinion, while critically important, is one of many pieces of evidence that is considered along with the totality of evidence in the case file.

B. CAUSATION

1. One of the major roles of the DMC is to provide the program with reports and opinions regarding causation. According to the program's legal requirements, a case can be accepted if the evidence in a particular case shows that there was a plausible relationship between the exposure at the workplace and the employee's illness or, in some cases, death.

DMCs may be asked to render their opinions regarding the causality of the specific occupational illnesses noted in Part B, including chronic beryllium disease, beryllium sensitivity and chronic silicosis. The statute provides specific diagnostic criteria for these conditions as well as the minimum duration of employment and latency. Causality for covered radiogenic cancers is determined by a process of dose reconstruction as performed by NIOSH.

DMCs may also be asked their opinions regarding causality and impairment for covered illnesses noted in Part E. Clinical guidance regarding case definitions for some key conditions is found in E-500 (see, for example, Exhibit 2). Impairment determinations are made by specially qualified DMCs who will utilize the evaluation process set forth in the AMA's Guides to the Evaluation of Permanent Impairment.

2. Legal Standards of Certainty and Concepts

There is a wide range of legal standards and concepts for judging certainty depending on the specific venue (e.g., criminal convictions, arrests, searches, police stops, a range of administrative or civil actions, etc.). These range from:

a. Highest - beyond a reasonable doubt (e.g., used to determine guilt in criminal cases);

b. Clear and convincing evidence (e.g., used in special civil cases such as commitment determinations);

c. Mid – preponderance of evidence (usual standard in civil cases and usually means more likely than not)[1];

d. Low - reasonable suspicion[2];

e. Lowest - mere suspicion (hunch).

In the EEOICP the causation standard for Part E seems to fall between level c and d (above).

Specifically, under Part E of the EEOICPA, the criteria for a covered illness requires, in part, that "it is at least as likely as not that exposure to a toxic substance at a [covered] facility was a significant factor in aggravating, contributing to, or causing the [illness]."[3]

3. Cause, Contribute, Aggravate

---

[1] For reasonable doubt, clear and convincing, and preponderance cite to *McCormick on Evidence*, sections 339-341 (K. Broun. ed. 6th ed West 2006).
[2] For probable cause and reasonable suspicion cite La Fave, et al, Criminal Procedure, sec 3.3 and 3.9(4th ed West 2004).
[3] 42 USC 7385s-3(a)(1)(B)

In establishing this relationship and developing the report, the DMC should take into consideration the following:

The program recognizes three types of causation: direct causation, contribution and aggravation.

a. Direct causation refers to a clear, linear, one-on-one relationship between the exposure and the illness or death in the absence of other diseases or conditions. A classic example of this type of causation is:

> A 67 year old male who never smoked worked at a covered DOE facility for 40 yrs where he was exposed to asbestos as a pipe fitter for a period no less than 15 years, beginning at age 30. He retired at age 65 and was in good health until age 67 when he developed a mass in the right upper lobe of the lung which was diagnosed as a poorly differentiated squamous cell carcinoma. A lobectomy was performed but the patient died 24 hours post surgery.

In this example, the only condition identified was the squamous cell carcinoma and the duration, intensity and latency of the asbestos exposure was likely sufficient to produce the carcinoma. Clinical literature amply supports such a relationship.

b. Contribute. The statute doesn't limit or restrict workplace exposure(s) as the "sole cause", "exclusive cause", "only cause", "primary cause" or the "sufficient cause". Workplace exposure(s) can contribute to an increased risk of illness, progression or acceleration (that "hasten") of the adverse outcome. A contributing cause may 1) increase the likelihood of suffering or harm, or 2) result in the earlier onset of a condition (hastening).

Two examples follow:

> Mr. B., a 56 year old male, worked at DOE facilities for 20 yrs as a heavy equipment operator and had extensive exposure to diesel fumes. He also smoked 1 pack/day for 20 years and now files a claim for COPD (chronic obstructive pulmonary disease).
>
> Mr. C., a 69 year old male, worked at a covered facility for 3 years. Beginning at age 30, he was a sheet metal worker and was exposed to asbestos. He was a heavy smoker with a 50 pack-year history of cigarettes. He claims his lung cancer, diagnosed at age 67, was related to his work as a sheet metal worker.

These cases exemplify how workplace exposure(s) may contribute to an illness, even though workplace exposure(s) might not be the sole or exclusive cause of

the condition. In these cases the challenge is to determine if workplace exposures: 1) may have increased the risk of the claimed condition, or 2) may have hastened the onset of the condition.

c. Aggravation can be defined as the worsening of a previously existing disease, condition or physical impairment by a workplace exposure or event. Consider whether workplace exposure(s) worsen, intensify or exacerbate symptoms, increase the clinical severity or clinical complications or lead to adverse outcomes of a pre-existing condition. Also consider whether workplace exposures "light up" or activate a condition that may have remained latent or inactive (e.g., TB).

Examples of aggravation include: a) increased frequency or severity of asthmatic attacks resulting from exposure to workplace chemicals, b) greater liver damage resulting from workplace exposure to solvents in a worker with mild liver damage who is a recovered alcoholic.[4]

In these cases, the DMC should consider and explain in his/her report whether it was at least as likely as not that the claimant's workplace exposure(s) was a significant factor in aggravating the employee's illness or death.

An example of aggravation is as follows:

> Mr. D's claimed illness of asbestosis was accepted by the district office to have been caused by his employment at a DOE site. Mr. D, a 65 y/o man, died in 2004 and his death certificate mentions cardiopulmonary arrest, coronary artery disease, ischemic cardiomyopathy and congestive heart failure as the reasons for his death. Multiple notes from treating physicians indicate that from 2000-2004 Mr. D was diagnosed with asbestosis, developed several episodes of pneumonia and suffered from severe dyspnea at rest. He received supplemental O_2 and several medications including bronchodilators. PFTs (pulmonary function studies) were increasingly abnormal in 1996, 1998 and 2002.

In this case, the claims examiner may be asking the physician whether Mr. D's congestive heart failure and ischemic cardiomyopathy were aggravated by his accepted condition of asbestosis. It is apparent from the medical evidence that Mr. D suffered from cardiac disease and that this led to his demise. However, it is also apparent that asbestosis played a significant role in his clinical course. Asbestosis is a chronic, progressive lung disease that impedes or restricts the intake of O_2 and its passage through the lungs to the blood stream. In Mr. D, this process was made obvious by the severe changes in the 1996 and 1998 PFTs. In turn, these abnormalities worsened the hypoxia (low oxygen) at the level of the

[4] For more examples of aggravation see, for example, "A guide to the work-relatedness of disease" pp 15-20 – NIOSH 1979

cardiac muscle fibers which gave rise to the ischemic cardiomyopathy and contributed to its inexorable progression.

4. Consequential Conditions

In addition, the program accepts as work-related a condition, disease or injury that arises as a consequence of a condition previously accepted by the program. In some cases, the DMC is asked to provide a causal link between the two conditions. Neither the fact that the illness manifests itself after the accepted covered illness was diagnosed, nor the belief of the claimant that the illness was caused by the accepted covered illness, is sufficient in itself to prove a causal relationship.

Generally, consequential illnesses and injuries fall into the following categories: recognized complications of the disease accepted by the program, complications of the treatment for the accepted condition, and injuries or diseases arising from unforeseen occurrences when the claimant is seeking or undergoing medical treatment for the accepted condition. Classic examples of consequential illnesses and injuries include: the development of pulmonary hypertension in a case of COPD; the development of osteoporosis and hypertension because of the long term use of steroids to treat chronic beryllium disease; and a traumatic fracture of the distal tibia and fibula which occurred upon falling on the sidewalk while walking to the doctor's office.

5. Framework: Basic Elements to Determine Causality - 5 steps

Address the specific questions posed by the CE and consider the following 5 steps:

a. Exposure. The CE will make sure that the claimant is a "covered worker" by documenting employment. For Part E, for example, eligible claimants are limited to DOE contractors, subcontractors, certain workers covered by sections of RECA, and certain survivors. The CE will check for "possible", "potential" or documented exposures and may utilize available records, including but not limited to:

- Records regarding specific work-sites;
- Known exposures for specific job titles or work areas;
- Industrial hygiene or other monitoring data (e.g., medical monitoring records);
- Plant records (e.g., incident or accident reports);
- The site exposure matrices (SEM); and/or
- Consultations with the EEOICP's specialists in industrial hygiene and/or toxicology.

In addition, at the time a claim is filed, the worker or survivor is asked to complete an Occupational History Questionnaire.

Consider the "nature, frequency and duration of exposure" as well as the intensity and route of exposure if this information is available. Given the need to

rely on historical data, the complete and specific information regarding all aspects of exposure may not be available. The question may require you to rely on "accepted" facts as found in the statement of accepted facts (SOAF).

The regulations note, "Proof of exposure to a toxic substance may be established by the submission of any appropriate document or information that is evidence that such substance was present at the facility in which the employee was employed and that the employee came into contact with such substance." "The OWCP site exposure matrices may be used to provide probative factual evidence that a particular substance was present at either a DOE facility or RECA section 5 facility."

b. Health Effect (Outcome). Consider the claimed health condition. Medical evidence as found in medical records, including hospital and clinic records, lab tests and imaging reports may be provided. Complete specific medical information may not be available and you may have to rely on less than perfect information to infer a diagnosis or clinical condition (e.g., from death certificates).

Guidance regarding covered diseases and illnesses and disease criteria for case definitions are specified in program regulations, bulletins and manuals for various conditions including, but not limited to:

1. Specified cancers
2. Chronic Beryllium Disease (CBD) – based on year of diagnosis (the statutory criteria for CBD under Part B do not apply to Part E)
3. Beryllium sensitivity
4. Chronic Silicosis
5. Asbestosis
6. Pneumoconiosis
7. COPD
8. Other conditions (see Exhibit 2, E-500)

c. Plausible Linkage. Consider the "plausible" connection between workplace exposure(s) and the claimed health outcome, based on the facts of the case. The program does not require 100% certainty, rather the conclusion of work-relatedness turns on the plausibility of the exposure/disease association.

Evaluating work-relatedness should be "evidence-based", grounded in scientific evidence, when available. Identifying and evaluating scientific evidence most often requires a review of the current epidemiologic literature regarding:

- The health effects of relevant occupational groups;
- The health effects of the claimed or established toxic exposures; and
- The known epidemiologic characteristics of the claimed illness.

Due diligence required to draw a conclusion regarding the plausible existence of an association between workplace exposures and the claimed illness will generally require a review of the current peer-reviewed literature on the specific

topic. Due diligence (taking due care) will often require a search using the National Library of Medicine data-base (www.pubmed.gov) as well as:

- A review of the key journal articles identified by the pubmed search (a review of abstracts is not sufficient).
- A review of the relevant authoritative textbooks.
- A review of professional society and government agency opinions, reports and guidelines.[5]

d. Judge Each Causal Element. Make individual determinations based on the totality of the evidence. Weigh the available evidence for each causal element including:

- "Cause" – "direct cause", "the cause", "sufficient cause";
- "Contribute" – consider increased risk or harm, or hastening;
- "Aggravate" – consider impact on the clinical severity (i.e., worsening) of a pre-existing condition.

Except in unusual situations it is almost always impossible to determine which person's disease was caused by a workplace exposure with 100% certainty.[6] Most diseases have multiple causes and each person may also be exposed to multiple exposures or may have other risk factors for the condition. While epidemiologic data may provide guidance for evaluating the risk of groups, individual determinations often rely on an expert opinion because of the methodological complexity, compounded by imperfect knowledge and incomplete evidence.

Consider any unusual features of the clinical condition including, but not limited to:

- The clinical course (e.g., rapid progression, aggressive disease, unusual pathology);
- Age at onset (e.g., early age of onset);
- Rarity of the condition in the general population;
- The known clustering or likelihood of occurrence among workers similarly exposed;
- Latency – note specific program criteria and any unusual patterns;
- The possibility of interaction arising from multiple exposures.[7]

e. Consider Alternative Explanations. Weigh the likelihood that other factors may have caused, contributed or aggravated the clinical condition including genetic susceptibility, life-style factors or non-occupational exposures.

---

[5] See Section IV below for additional references and resources for DMCs.
[6] Samet JM: Improving Presumptive Disability Decision-making for Veterans. NAS, Washington, DC 2008
[7] Samet 2008.

Suggestion: Consider the plausibility of the purported association between the exposure and outcome and then consider if the plausibility meets the "at least as likely as not" threshold.

6. Selected Knotty Issues in Causation

- People differ substantially in their response to noxious exposures.
- Many diseases of occupational origin are multifactoral (multicausal), with non-occupational and occupational factors playing contributory roles.
- The clinical and pathologic expression of most occupational diseases are indistinguishable from those of non-occupational origin.

a. Duration of exposure intensity and latency. It is acknowledged that brief and intense exposures can be associated with adverse health effects, for example, accidental inhalations.

In general, there is no known threshold for many carcinogens.

Latency may be shortened by more intense or higher cumulative exposures.

The program provides some guidance for the minimum duration of exposure and latency for specific medical conditions.[8]

b. Smoking and Workplace Exposures. A history of smoking does not negate the role of workplace exposures in making a supportive determination.

The American Thoracic Society (ATS) statement on the Occupational Contribution to the Burden of Airway Disease (2003) notes: "Despite the difficulty of disentangling the effects of cigarette smoke from those of other exposures, an increasingly impressive body of scientific literature is available demonstrating that specific occupational exposures contribute to the development of COPD." p 788.[9]

"Overall, the magnitude of effect of occupational exposures appears consistent with that of cigarette smoking." P 788.[10]

Henrick notes (Thorax, 1996) that only 15-20% of smokers actually develop COPD. "There is evidence, however, that when smokers additionally work with noxious respirable agents, COPD occurs with unusual frequency and/or severity."

---

[8] See, PM E-500 Exhibit 2.
[9] ATS: American Thoracic Society Statement: Occupational Contribution to the Burden of Airway Disease. Am J Resp Care Med 167:787-797 2003. Found at http://www.thoracic.org/sections/publications/statements/pages/eoh/burden1-11.html.
[10] Id.

This "…indicates interaction between smoking and working environment."[11]

7. DMC Check-list

The most common problems with DMC reports fall into the following 5 categories:

1. The reports don't answer the specific question that was posed and/or the exact language used in the program was not used.
2. The correct causal criteria were not used.
3. Rationale is incomplete, vague, inconsistent, inaccurate or not well developed.
4. No references were provided to support the rationale.
5. Reports were not timely.

DMC reports must:

- Address the specific question(s) posed in the request.
- Use the specific language used by the DEEOIC
- Provide a fully developed ("fully rationalized") report.
- Perform a search of the relevant peer-reviewed literature.

In an effort to improve the quality of DMC reports consider the following check-list before submitting your report:

1. Have I answered the specific questions posed?
2. Have I used the specific program criteria?
3. Have I considered each aspect of causation: "cause", "contribute" and "aggravated"?
4. Is the rationale fully developed?
5. Have I performed a literature search to assure due diligence?
6. Have I included supportive references?
7. Have I submitted my report within the 21 day time frame?
8. Have I signed the conflict of interest statement?
9. Have I clearly stamped the report "medical confidential"?

[11] Hendrick DJ. Occupation and chronic obstructive pulmonary disease (COPD). Thorax 51:947-955 1996.

4 PM E-500 Exhibit 2

Matrix for Confirming Sufficient Evidence of Covered Illness

SILICOSIS, CHRONIC

Criteria	Sufficient evidence of covered illness	Sufficient evidence of possible covered illness, requires physician review
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	10 years or more	5-10 years
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of silicosis made by a medical doctor And 2. Any <u>one</u> of the following three criteria a. A chest radiograph, interpreted by NIOSH certified B reader classifying the existence of pneumoconioses of category 1/0 or higher; <u>or</u> b. Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are consistent with silicosis Such as nodules, or fibrosis usually with upper lung zone predominance c. Lung biopsy findings consistent with silicosis Such as silicotic nodules	Some, but not all criteria to establish the illness are met** Or Medical record (includes any provider report, results of imaging studies, surgical or pathology reports, or other acceptable record) mention of silicosis, possible silicosis, restrictive lung disease, fibrosis, or pneumoconiosis <u>Or</u> Death certificate mention of silicosis, possible silicosis, restrictive lung disease, fibrosis or pneumoconiosis Or A chest radiograph, interpreted by NIOSH certified B reader classifying the existence of pneumoconioses of category 0/1 Or Lung biopsy findings suggestive of silicosis
Additional considerations for causation	*None needed*	*None needed*

* The actual latency period for disease development is a function of the duration and intensity of exposure.
** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.
*** References utilized include American Thoracic Society consensus statement.

SILICOSIS, ACUTE

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Weeks to months	Weeks to months
Medical Evidence for illness and diagnostic testing criteria	1. Any one of the following two criteria; and a. A written diagnosis of acute silicosis made by a medical doctor; or b. Death certificate or other acceptable documentation of death due to acute silicosis And 2. The medical record contains no other diagnoses, such that would otherwise account for the acute sudden severe lung illness, such as other infection or ARDS	Some, but not all criteria to establish the illness are met** Or Written evidence of sudden lung illness causing death or severe, overwhelming lung illness, even if attributed to tuberculosis or other illness or infection Or Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are suggestive of acute silicosis *Such as: air space obliteration, alveolar filling pattern, pulmonary edema, pulmonary hemorrhage, infiltrate, alveolar proteinosis* Or Results of lung function testing (PFT or spirometry) showing sudden worsening Or Lung biopsy findings suggestive of acute silicosis Such as alveoli filled with proteinaceous material
Additional considerations for causation	*None needed*	*None needed*

* The actual latency period for the development is a function of the exposure's duration and intensity.
** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.
*** References utilized include American Thoracic Society consensus statement.

SILICOSIS, ACCELERATED

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	2-5 years	< 2years or > 5 years
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of accelerated silicosis made by a medical doctor And 2. Any one of the following three criteria a. A chest radiograph, interpreted by NIOSH certified B reader classifying the existence of pneumoconioses of category 1/0 or higher; or b. Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are consistent with silicosis Such as nodules or fibrosis usually with upper lung zone predominance c. Lung biopsy findings consistent with silicosis Such as silicotic nodules	Some, but not all criteria to establish the illness are met** Or Medical record (includes any provider report, results of imaging studies, surgical or pathology reports, or other acceptable record) mention of accelerated silicosis, silicosis, possible silicosis, restrictive lung disease, fibrosis, or pneumoconiosis Or Death certificate mention of silicosis, possible silicosis, restrictive lung disease, fibrosis or pneumoconiosis Or A chest radiograph, interpreted by NIOSH certified B reader classifying the existence of pneumoconioses of category 0/1 Or Lung biopsy findings suggestive of silicosis
Additional considerations for causation	*None needed*	*None needed*

* The actual latency period for the development of this disease is a function of the duration and intensity of exposure.
** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.
*** References utilized include American Thoracic Society consensus statement.

SILICOSIS, COMPLICATED

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Years to decades	Years to decades
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of progressive massive fibrosis (PMF) or complicated silicosis made by a medical doctor And 2. Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are consistent with PMF Progression and coalescence of the upper lung zone nodules to form masses (conglomerate lesions) When they cause contraction of the lobes, an "angel wing pattern" can be seen	Some, but not all criteria to establish the illness are met**
Additional considerations for causation	*None needed*	*None needed*

* The actual latency period for the development of this disease is a function of the duration and intensity of exposure.
** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.
*** References utilized include American Thoracic Society consensus statement.

BERYLLIUM SENSITIZATION

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review
DOE exposure criteria	Verification that an employee worked in a facility where beryllium was present	Verification that an employee worked in a facility where beryllium was present
Latency	First DOE exposure must have preceded first abnormal test for beryllium sensitization	First DOE exposure must have preceded first abnormal test for beryllium sensitization
Medical Evidence for illness and diagnostic testing criteria	1. Medical documentation one of following two criteria* a. Beryllium sensitivity or sensitization established by an abnormal BeLPT performed on either blood or lung lavage cells; or b. Positive reaction to beryllium patch testing And 2. No signs, or symptoms, or any medical evaluation evidence of abnormalities suggestive of possible chronic beryllium disease	If BeLPT was borderline or uninterpretable, it is recommended that the test be repeated. After two borderline LPTs, it is recommended that the employee be counseled to pursue appropriate medical follow-up for additional beryllium testing options and/or disease evaluation After third uninterpretable BeLPT, it is recommended the employee undergo patch testing for beryllium sensitization, if not still working with beryllium
Additional considerations for causation	*None needed*	*None needed*

ASBESTOS RELATED DISORDERS

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Pleural plaques: 20 or more years Pleural effusions: 5-30 years	Pleural plaques: < 20 years Pleural effusions: <5 or > 30 years
Medical Evidence for illness and diagnostic testing criteria	Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are consistent with these disorders Pleural plaques Pleural thickening, not associated with an area of prior surgery or trauma Rounded atelectasis Bilateral pleural effusions, also called benign asbestos related pleural effusion	Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are consistent with these disorders Pleural thickening in an area of prior surgery or trauma Pleural effusion, if the record does not indicate that there is another disease process that would otherwise account for the effusion, such as congestive heart failure (CHF), cancer, or other lung disease
Additional considerations for causation	*None needed*	*None needed*

* The actual latency period for the development of this disease is a function of the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

*** References utilized include American Thoracic Society consensus statement.

LUNG FIBROSIS

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Years	Years
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of lung fibrosis made by a medical doctor And 2. Any one of the following three criteria a. Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are consistent with fibrosis Such as small lung fields or volumes, minimal ground glass opacities, and/or bibasilar reticular abnormalities b. Results of breathing tests (PFTs or spirometry) showing a restrictive or mixed pattern Such as FVC <80% predicted c. Lung biopsy findings consistent with fibrosis And 3. There is no evidence in the medical record that the lung fibrosis is present due to another disease process.	Some, but not all criteria to establish the illness are met** Or Medical record (includes any provider report, results of imaging studies, surgical or pathology reports, or other acceptable record) of lung fibrosis Or Death certificate mention of fibrosis Or Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are suggestive of fibrosis
Additional considerations for causation	*None needed*	*None needed*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

PM E-500 Exhibit 2 – Matrix for Confirming Sufficient Evidence of Covered Illness

PNEUMOCONIOSIS

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Years	Years
Medical Evidence for illness and diagnostic testing criteria	1. Written evidence of one of the following two criteria a. A written diagnosis of pneumoconiosis made by a medical doctor; or b. Results of breathing tests (PFTs or spirometry) showing a restrictive lung pattern FVC < 80% predicted And 2. Any one of the following three criteria a. A chest radiograph, interpreted by NIOSH certified B reader classifying the existence of pneumoconiosis of category 1/0 or higher; or b. Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are consistent with asbestosis and/or findings of pleural plaques or rounded atelectasis; or c. Lung biopsy findings consistent with pneumoconiosis	Some, but not all criteria to establish the illness are met** Or Medical record (includes any provider report, results of imaging studies, surgical or pathology reports, or other acceptable record) of silicosis, possible asbestosis, restrictive lung disease, or pneumoconiosis Or Death certificate mention of silicosis, possible asbestosis, restrictive lung disease, or pneumoconiosis Or A chest radiograph, interpreted by NIOSH certified B reader classifying the existence of pneumoconiosis of category 0/1 Or Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are suggestive of pneumoconiosis.
Additional considerations for causation	*None needed*	*None needed*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Years	Months or years
Medical Evidence for illness and diagnostic testing criteria	1. Any one of the following three criteria a. A written diagnosis of COPD or chronic bronchitis made by a medical doctor Chronic bronchitis is defined as the presence of chronic productive cough for 3 months in each of two successive years and other causes of cough have been excluded b. Results of PFTs or spirometry showing an obstructive or mixed pattern $FEV_1/FVC < 70\%$ and $FEV_1 < 80\%$ predicted. c. Results from a chest x-ray or other imaging technique that are consistent with COPD Such as air trapping, flattening of diaphragms, enlarged lung fields. And 2. The employee has a history of being a never smoker*** And 3. There is no other lung disease present that would account for the findings	Some, but not all criteria to establish the illness are met** Emphysema is caused by only a small subset of the toxic substances associated with chronic bronchitis, however it may be aggravated by the others on this list.
Additional considerations for causation	*There is currently no medical testing or means to distinguish COPD due to any of the above toxic substance exposures and COPD due to other causes. Physician review is required.*	*Physician review is required. Also, if all criteria are otherwise met, individuals with Alpha-1 Antitrypsin Deficiency (AAT Deficiency) may be considered to have a covered illness.*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

***ATS criterion for a never smoker, or non-smoker, is < 20 packs of cigarettes in a lifetime, but this piece of information may not be found in most medical records.

PM E-500 Exhibit 2 – Matrix for Confirming Sufficient Evidence of Covered Illness

DIABETES

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria	The are no generally accepted toxic substance known to cause or accelerate diabetes.	However, diabetes can be a consequence of the treatment of some covered illnesses.
Latency	N/A	N/A
Medical Evidence for illness and diagnostic testing criteria	N/A	N/A
Additional considerations for causation	N/A	N/A

MESOTHELIOMA

Criteria	Sufficient evidence to establish a covered illness. If some but not all criteria are met, physician review recommended	Evidence that suggests a covered illness exists and that physician review is recommended
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	30-50 years	20-29 or > 50 years
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of mesothelioma made by a medical doctor And 2. Pathology report consistent with mesothelioma from surgical or biopsy specimen	Some, but not all criteria to establish the illness are met** Or Medical record (includes any provider report, results of imaging studies, surgical or pathology reports, or other acceptable record) or death certificate mention of mesothelioma or pleural malignancy Or Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are suggestive of mesothelioma Such as large, unilateral pleural effusion, pleural mass, pleural rind, or diffuse pleural thickening
Additional considerations for causation	*None needed*	None needed

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

*** References utilized include American Thoracic Society consensus statement.

PM E-500 Exhibit 2 – Matrix for Confirming Sufficient Evidence of Covered Illness

LUNG CANCER

Criteria	Sufficient evidence to establish a covered illness. If some but not all criteria are met, physician review recommended	Evidence that suggests a covered illness exists and that physician review is recommended
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	10-20 years	>20 years
Medical Evidence for illness and diagnostic testing criteria	1. Any one of the following two criteria a. A written diagnosis of lung cancer (malignancy) made by a medical doctor; or b. Pathology report consistent with lung cancer (small cell, oat cell, large cell, squamous cell, adenocarcinoma) from surgical or biopsy specimen And 2. The employee has a history of being a never smoker***	Some, but not all criteria to establish the illness are met** Or Medical record (includes any provider report, results of imaging studies, surgical or pathology reports, or other acceptable record) or death certificate mention of lung cancer (malignancy) *Or* Results from a chest x-ray or computer assisted tomography (CT) or other imaging technique that are suggestive of lung cancer Such as lung mass
Additional considerations for causation	*There is currently no medical testing or means to distinguish cancer due to any of the above toxic substance exposures and cancer due to other causes. Physician review is required.*	Physician review is required.

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

***ATS criterion for a never smoker, or non-smoker, is < 20 packs of cigarettes in a lifetime, but this piece of information may not be found in most medical records.

KIDNEY DISEASE

Criteria	Sufficient evidence to establish a covered illness. If some but not all criteria are met, physician review recommended	Evidence that suggests a covered illness exists and that physician review is recommended
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Months or years	Days, months, or years
Medical Evidence for illness and diagnostic testing criteria	1. Any one of the following two criteria a. A written diagnosis of kidney disease made by a medical doctor Other terms are chronic renal disease, chronic renal failure, renal insufficiency b. The worker required dialysis And 2. The worker does not have high blood pressure or diabetes And 3. The type of kidney disease diagnosed is consistent with one known to be caused by the identified toxic substance.	Some, but not all criteria to establish the illness are met**
Additional considerations for causation	*Additional testing may be required to help establish a causal link between a toxic substance and a specific kidney disease. This may include additional urine testing, such as β_2-microglobulin or retinol binding protein and/or biological tests to detect residual evidence of the toxic substance in the body. The need for this additional testing should be determined by the reviewing physician.* *Physician review is required.*	*Physician review is required.*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

ASTHMA, OCCUPATIONAL

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Weeks, months, or years	Weeks, months, or years
Medical Evidence for illness and diagnostic testing criteria	1. The following three criteria: i. Onset of asthma occurring after first DOE exposure (except resolved asthma childhood) And ii. A written diagnosis of occupational asthma or asthma caused by toxic substance made by a medical doctor And iii. The diagnosis of asthma was made based on any one of the following criteria a. Methacholine challenge test results showing a $PC_{20} \leq 8$ mg/ml; or b. Post-bronchocodialator reversibility of $FEV_1 \geq 12\%$ and 200 ml; or c. Post-bronchocodialator reversibility of $FEV_1 \geq 12\%$, but <20 ml, with subsequent improvement in $FEV_1 \geq 20\%$ after steroid trial And	Some, but not all criteria to establish the illness are met** Occupational asthma via sensitization to a new agent in the workplace can occur in workers with pre-existing asthma. Additional testing that can be consistent with the diagnosis, but does not establish the diagnosis. 1. Positive skin prick testing or serologic IgE (RAST) testing to the toxic substance
Additional considerations for causation	1. An association between symptoms of asthma and work, including wheeze and/or shortness of breath that are better on days away from work, especially on holiday or vacation. And 2. One or more of the following criteria: a. work-related change in FEV_1 or PEF rate; or b. work-related change in bronchial hyperresponsiveness; or c. positive response to specific inhalation challenge test (note this is not recommended if not already performed)	*None needed*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers request for additional information from the worker for exposure and/or diagnostic testing criteria. This request should also be made if there is insufficient information to establish exposure or illness.

ASTHMA, IRRITANT INDUCED

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Days, months, or years	Days, months, or years
Medical Evidence for illness and diagnostic testing criteria	1. The three following criteria: a. Onset of asthma occurring after first DOE exposure (except resolved asthma childhood) And b. A written diagnosis of occupational asthma, irritant induced asthma, or asthma caused by toxic substance made by a medical doctor And	Some, but not all criteria to establish the illness are met**
Additional considerations for causation	1. An association between symptoms of asthma and work, including wheeze and/or shortness of breath are better on days away from work, especially on holiday or vacation. And 2. One or more of the following criteria: a. work-related change in FEV_1 or PEF rate; or b. positive response to specific inhalation challenge test (note this is not recommended if not already performed); or c. Onset of asthma in clear association with a symptomatic exposure to an irritant agent in the workplace. This includes RADS, occurring after a single exposure to a substance with irritant properties present in a very high concentration, if other disease processes have been ruled out.	*None needed*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

ASTHMA, IRRITANT AGGRAVATED

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Days or months	Days or months
Medical Evidence for illness and diagnostic testing criteria	1. History of asthma as an adult prior to DOE exposure And	Some, but not all criteria to establish the illness are met**
Additional considerations for causation	1. The two following criteria a. An association between symptoms of asthma and work, including wheeze and/or shortness of breath are better on days away from work, especially on holiday or vacation. And 2. The worker was symptomatic or required medication before and had increase in symptoms or medication requirement after beginning to work with the above substance.	*None needed*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

HEART ATTACK

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Weeks, months, or years	Weeks, months, or years
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of heart attack or sudden death due to heart disease by a medical doctor And 2. The heart attack or sudden death occurred after being away from nitrate exposure for a couple of days following a number of days of regular nitrate exposure (classically on a Monday morning).	Some, but not all criteria to establish the illness are met** This is strongly supported by a history of recurrent headaches following a similar pattern
Additional considerations for causation	Due to high prevalence of heart disease and heart attacks, physician review is recommended for determination of causation.	*Physician review recommended*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

For nitrates only.

NEUROPATHY, TOXIC

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review.
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Days, months, or years	Days, months, or years
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of peripheral neuropathy, toxic neuropathy, or neuropathy due to a toxic substance. And 2. The physician's diagnosis was made by all three of the following criteria. Note: the definition of the classic syndrome will vary among the different toxic substances. a. Symptoms consistent with the classic syndrome caused by the specific toxic substance Sensory; or Motor; or Sensorimotor b. Signs consistent with the classic syndrome caused by the specific toxic substance Decreased or abnormal distal sensation Such as stocking-glove numbness, allodynia, and/or hyperalgesia Decreased or absent distal reflexes Distal muscle weakness and/or atrophy c. Results of electrodiagnostic studies consistent with a neuropathy caused by the specific toxic substance. Should include both needle EMG and nerve conduction studies (NCS)	Some, but not all criteria to establish the illness are met**
Additional considerations for causation	Electrodiagnostic testing can distinguish some but not all toxic neuropathies from those due to other causes. There are many medical causes of peripheral neuropathy, especially sensorimotor neuropathies. Physician review required.	*Physician review is required.*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers request for additional information from the worker for exposure and/or diagnostic testing criteria. This request should also be made if there is insufficient information establish a possible exposure or illness.

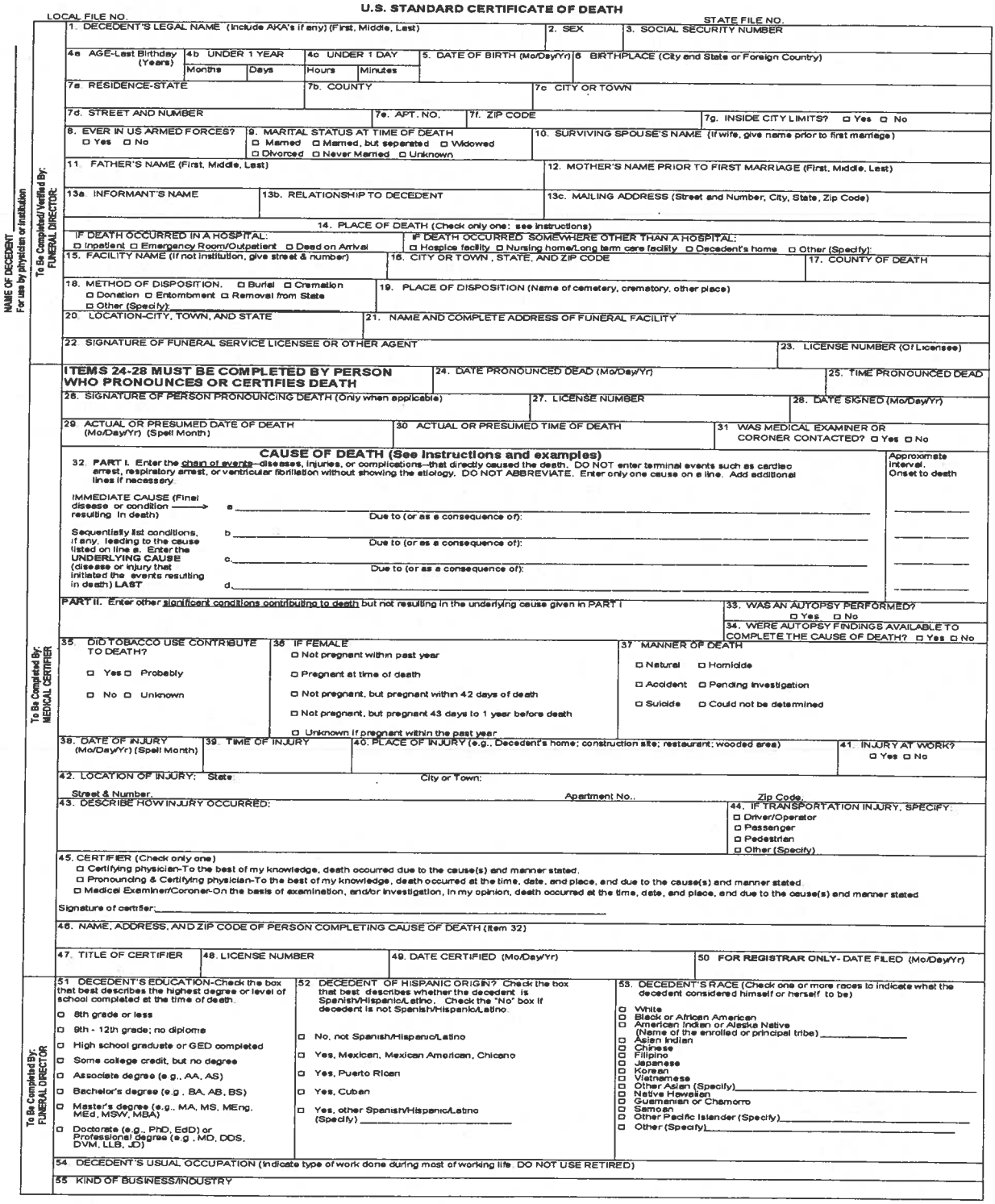
ENCEPHALOPATHY, CHRONIC TOXIC

Criteria	Sufficient evidence to establish a covered illness	Sufficient evidence to establish a possible illness requiring physician review
DOE exposure criteria*	DOE Facilities Specific job titles/ processes Applicable dates	DOE Facilities Specific job titles/ processes Applicable dates And Additional information is needed**
Latency*	Years	Days, months, or years
Medical Evidence for illness and diagnostic testing criteria	1. A written diagnosis of chronic toxic encephalopathy (ICD9 code 349.82 or analogous conditions) made by a medical doctor And 2. A formal neuropsychological assessment that included a battery of neurobehavioral tests is consistent with the diagnosis. 3. Appropriate neuroimaging studies (e.g. brain MRI, head CT) have been performed to investigate findings consistent with the diagnosis, or suggestive of unrelated causes.	
Additional considerations for causation	*Some patterns on the history and neurobehavioral test profile may be more consistent with chronic toxic encephalopathy than with unrelated causes (e.g. greater decrements in performance vs. verbal IQ). Physician review is required.*	*Physician review is required.*

* The actual latency period for the development of this disease is a function of the specific causative toxic substance as well as the duration and intensity of exposure.

** Triggers DOL request for additional information from the worker for exposure and/or diagnostic testing criteria elements. A request for additional information should also be made if there is insufficient information present to establish a possible exposure or illness.

5 Death Certificate



U.S. STANDARD CERTIFICATE OF DEATH

LOCAL FILE NO. STATE FILE NO.

NAME OF DECEDENT — For use by physician or institution — To Be Completed/Verified By: FUNERAL DIRECTOR:

1. DECEDENT'S LEGAL NAME (Include AKA's if any) (First, Middle, Last) | 2. SEX | 3. SOCIAL SECURITY NUMBER

4a. AGE-Last Birthday (Years) | 4b. UNDER 1 YEAR (Months / Days) | 4c. UNDER 1 DAY (Hours / Minutes) | 5. DATE OF BIRTH (Mo/Day/Yr) | 6. BIRTHPLACE (City and State or Foreign Country)

7a. RESIDENCE-STATE | 7b. COUNTY | 7c. CITY OR TOWN

7d. STREET AND NUMBER | 7e. APT. NO. | 7f. ZIP CODE | 7g. INSIDE CITY LIMITS? ☐ Yes ☐ No

8. EVER IN US ARMED FORCES? ☐ Yes ☐ No | 9. MARITAL STATUS AT TIME OF DEATH ☐ Married ☐ Married, but separated ☐ Widowed ☐ Divorced ☐ Never Married ☐ Unknown | 10. SURVIVING SPOUSE'S NAME (If wife, give name prior to first marriage)

11. FATHER'S NAME (First, Middle, Last) | 12. MOTHER'S NAME PRIOR TO FIRST MARRIAGE (First, Middle, Last)

13a. INFORMANT'S NAME | 13b. RELATIONSHIP TO DECEDENT | 13c. MAILING ADDRESS (Street and Number, City, State, Zip Code)

14. PLACE OF DEATH (Check only one: see instructions)

IF DEATH OCCURRED IN A HOSPITAL: ☐ Inpatient ☐ Emergency Room/Outpatient ☐ Dead on Arrival

IF DEATH OCCURRED SOMEWHERE OTHER THAN A HOSPITAL: ☐ Hospice facility ☐ Nursing home/Long term care facility ☐ Decedent's home ☐ Other (Specify):

15. FACILITY NAME (If not institution, give street & number) | 16. CITY OR TOWN, STATE, AND ZIP CODE | 17. COUNTY OF DEATH

18. METHOD OF DISPOSITION: ☐ Burial ☐ Cremation ☐ Donation ☐ Entombment ☐ Removal from State ☐ Other (Specify): | 19. PLACE OF DISPOSITION (Name of cemetery, crematory, other place)

20. LOCATION-CITY, TOWN, AND STATE | 21. NAME AND COMPLETE ADDRESS OF FUNERAL FACILITY

22. SIGNATURE OF FUNERAL SERVICE LICENSEE OR OTHER AGENT | 23. LICENSE NUMBER (Of Licensee)

To Be Completed By: MEDICAL CERTIFIER

ITEMS 24-28 MUST BE COMPLETED BY PERSON WHO PRONOUNCES OR CERTIFIES DEATH

24. DATE PRONOUNCED DEAD (Mo/Day/Yr) | 25. TIME PRONOUNCED DEAD

26. SIGNATURE OF PERSON PRONOUNCING DEATH (Only when applicable) | 27. LICENSE NUMBER | 28. DATE SIGNED (Mo/Day/Yr)

29. ACTUAL OR PRESUMED DATE OF DEATH (Mo/Day/Yr) (Spell Month) | 30. ACTUAL OR PRESUMED TIME OF DEATH | 31. WAS MEDICAL EXAMINER OR CORONER CONTACTED? ☐ Yes ☐ No

CAUSE OF DEATH (See instructions and examples)

32. PART I. Enter the chain of events—diseases, injuries, or complications—that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary.

Approximate Interval: Onset to death

IMMEDIATE CAUSE (Final disease or condition resulting in death) → a. _____ Due to (or as a consequence of):

Sequentially list conditions, if any, leading to the cause listed on line a. Enter the UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST

b. _____ Due to (or as a consequence of):

c. _____ Due to (or as a consequence of):

d. _____

PART II. Enter other significant conditions contributing to death but not resulting in the underlying cause given in PART I

33. WAS AN AUTOPSY PERFORMED? ☐ Yes ☐ No

34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? ☐ Yes ☐ No

35. DID TOBACCO USE CONTRIBUTE TO DEATH? ☐ Yes ☐ Probably ☐ No ☐ Unknown

36. IF FEMALE ☐ Not pregnant within past year ☐ Pregnant at time of death ☐ Not pregnant, but pregnant within 42 days of death ☐ Not pregnant, but pregnant 43 days to 1 year before death ☐ Unknown if pregnant within the past year

37. MANNER OF DEATH ☐ Natural ☐ Homicide ☐ Accident ☐ Pending Investigation ☐ Suicide ☐ Could not be determined

38. DATE OF INJURY (Mo/Day/Yr) (Spell Month) | 39. TIME OF INJURY | 40. PLACE OF INJURY (e.g., Decedent's home; construction site; restaurant; wooded area) | 41. INJURY AT WORK? ☐ Yes ☐ No

42. LOCATION OF INJURY: State: City or Town: Street & Number: Apartment No.: Zip Code:

43. DESCRIBE HOW INJURY OCCURRED:

44. IF TRANSPORTATION INJURY, SPECIFY: ☐ Driver/Operator ☐ Passenger ☐ Pedestrian ☐ Other (Specify)

45. CERTIFIER (Check only one)
☐ Certifying physician-To the best of my knowledge, death occurred due to the cause(s) and manner stated.
☐ Pronouncing & Certifying physician-To the best of my knowledge, death occurred at the time, date, and place, and due to the cause(s) and manner stated.
☐ Medical Examiner/Coroner-On the basis of examination, and/or investigation, in my opinion, death occurred at the time, date, and place, and due to the cause(s) and manner stated.

Signature of certifier: _____

46. NAME, ADDRESS, AND ZIP CODE OF PERSON COMPLETING CAUSE OF DEATH (Item 32)

47. TITLE OF CERTIFIER | 48. LICENSE NUMBER | 49. DATE CERTIFIED (Mo/Day/Yr) | 50. FOR REGISTRAR ONLY- DATE FILED (Mo/Day/Yr)

To Be Completed By: FUNERAL DIRECTOR

51. DECEDENT'S EDUCATION-Check the box that best describes the highest degree or level of school completed at the time of death.
☐ 8th grade or less
☐ 9th - 12th grade; no diploma
☐ High school graduate or GED completed
☐ Some college credit, but no degree
☐ Associate degree (e.g., AA, AS)
☐ Bachelor's degree (e.g., BA, AB, BS)
☐ Master's degree (e.g., MA, MS, MEng, MEd, MSW, MBA)
☐ Doctorate (e.g., PhD, EdD) or Professional degree (e.g., MD, DDS, DVM, LLB, JD)

52. DECEDENT OF HISPANIC ORIGIN? Check the box that best describes whether the decedent is Spanish/Hispanic/Latino. Check the "No" box if decedent is not Spanish/Hispanic/Latino.
☐ No, not Spanish/Hispanic/Latino
☐ Yes, Mexican, Mexican American, Chicano
☐ Yes, Puerto Rican
☐ Yes, Cuban
☐ Yes, other Spanish/Hispanic/Latino (Specify) _____

53. DECEDENT'S RACE (Check one or more races to indicate what the decedent considered himself or herself to be)
☐ White
☐ Black or African American
☐ American Indian or Alaska Native (Name of the enrolled or principal tribe) _____
☐ Asian Indian
☐ Chinese
☐ Filipino
☐ Japanese
☐ Korean
☐ Vietnamese
☐ Other Asian (Specify) _____
☐ Native Hawaiian
☐ Guamanian or Chamorro
☐ Samoan
☐ Other Pacific Islander (Specify) _____
☐ Other (Specify) _____

54. DECEDENT'S USUAL OCCUPATION (Indicate type of work done during most of working life. DO NOT USE RETIRED)

55. KIND OF BUSINESS/INDUSTRY

REV. 11/2003

Death Certificate

MEDICAL CERTIFIER INSTRUCTIONS for selected items on U.S. Standard Certificate of Death
(See Physicians' Handbook or Medical Examiner/Coroner Handbook on Death Registration for instructions on all items)

ITEMS ON WHEN DEATH OCCURRED

Items 24-25 and 29-31 should always be completed. If the facility uses a separate pronouncer or other person to indicate that death has taken place with another person more familiar with the case completing the remainder of the medical portion of the death certificate, the pronouncer completes items 24-28. If a certifier completes items 24-25 as well as items 29-49, items 26-28 may be left blank.

ITEMS 24-25, 29-30 – DATE AND TIME OF DEATH

Spell out the name of the month. If the exact date of death is unknown, enter the **approximate** date. If the date cannot be approximated, enter the date the body is found and identify as **date found**. Date pronounced and actual date may be the same. Enter the exact hour and minutes according to a 24-hour clock; estimates may be provided with "Approx." placed before the time.

ITEM 32 – CAUSE OF DEATH (See attached examples)

Take care to make the entry legible. Use a computer printer with high resolution, typewriter with good black ribbon and clean keys, or print legibly using permanent **black** ink in completing the CAUSE OF DEATH Section. **Do not abbreviate** conditions entered in section.

Part I (Chain of events leading directly to death)
•Only **one** cause should be entered on each line. Line (a) **MUST ALWAYS** have an entry. **DO NOT** leave blank. Additional lines may be added if necessary.
•If the condition on Line (a) resulted from an underlying condition, put the underlying condition on Line (b), and so on, until the full sequence is reported. **ALWAYS** enter the **underlying cause of death** on the lowest used line in Part I.
•For each cause indicate the best estimate of the interval between the presumed onset and the date of death. The terms "unknown" or "approximately" may be used. General terms, such as minutes, hours, or days, are acceptable, if necessary. **DO NOT** leave blank.
•The terminal event (for example, cardiac arrest or respiratory arrest) should not be used. If a mechanism of death seems most appropriate to you for line (a), then you must always list its cause(s) on the line(s) below it (for example, cardiac arrest **due to** coronary artery atherosclerosis *or* cardiac arrest **due to** blunt impact to chest).
• If an organ system failure such as congestive heart failure, hepatic failure, renal failure, or respiratory failure is listed as a cause of death, always report its etiology on the line(s) beneath it (for example, renal failure **due to** Type I diabetes mellitus).
•When indicating neoplasms as a cause of death, include the following: 1) primary site *or* that the primary site is unknown, 2) benign or malignant, 3) cell type *or* that the cell type is unknown, 4) grade of neoplasm, and 5) part or lobe of organ affected. (For example, a primary well-differentiated squamous cell carcinoma, lung, left upper lobe.)
•Always report the fatal injury (for example, stab wound of chest), the trauma (for example, transection of subclavian vein), and impairment of function (for example, air embolism).

PART II (Other significant conditions)
•Enter all diseases or conditions contributing to death that were not reported in the chain of events in Part I and that did not result in the **underlying cause of death**. See attached examples.
•If two or more possible sequences resulted in death, or if two conditions seem to have added together, report in Part I the one that, in your opinion, most directly caused death. Report in Part II the other conditions or diseases.

CHANGES TO CAUSE OF DEATH

Should additional medical information or autopsy findings become available that would change the cause of death originally reported, the original death certificate should be amended by the certifying physician by **immediately** reporting the revised cause of death to the State Vital Records Office.

ITEMS 33-34 - AUTOPSY

•33 - Enter "Yes" if either a partial or full autopsy was performed. Otherwise enter "No."
•34 - Enter "Yes" if autopsy findings were available to complete the cause of death; otherwise enter "No". Leave item blank if no autopsy was performed.

ITEM 35 - DID TOBACCO USE CONTRIBUTE TO DEATH?

Check "yes" if, in your opinion, the use of tobacco contributed to death. Tobacco use may contribute to deaths due to a wide variety of diseases; for example, tobacco use contributes to many deaths due to emphysema or lung cancer and some heart disease and cancers of the head and neck. Check "no" if, in your clinical judgment, tobacco use did not contribute to this particular death.

ITEM 36 - IF FEMALE, WAS DECEDENT PREGNANT AT TIME OF DEATH OR WITHIN PAST YEAR?

This information is important in determining pregnancy-related mortality.

ITEM 37 - MANNER OF DEATH

•Always check Manner of Death, which is important: 1) in determining accurate causes of death; 2) in processing insurance claims; and 3) in statistical studies of injuries and death.
•Indicate "Pending investigation" if the manner of death cannot be determined whether due to an accident, suicide, or homicide within the statutory time limit for filing the death certificate. This should be changed later to one of the other terms.
•Indicate "Could not be Determined" **ONLY** when it is impossible to determine the manner of death.

ITEMS 38-44 - ACCIDENT OR INJURY – to be filled out in all cases of deaths due to injury or poisoning.

•38 - Enter the exact month, day, and year of injury. Spell out the name of the month. **DO NOT** use a number for the month. (Remember, the date of injury may differ from the date of death.) Estimates may be provided with "Approx." placed before the date.
•39 - Enter the exact hour and minutes of injury or use your best estimate. Use a 24-hour clock.
•40 - Enter the general place (such as restaurant, vacant lot, or home) where the injury occurred. **DO NOT** enter firm or organization names. (For example, enter "factory", **not** "Standard Manufacturing, Inc.")
•41 - Complete if anything other than natural disease is mentioned in Part I or Part II of the medical certification, including homicides, suicides, and accidents. This includes all motor vehicle deaths. The item **must** be completed for decedents ages 14 years or over and may be completed for those less than 14 years of age if warranted. Enter "Yes" if the injury occurred at work. Otherwise enter "No". An injury may occur at work regardless of whether the injury occurred in the course of the decedent's "usual" occupation. Examples of injury at work and injury not at work follow:

Injury at work	**Injury not at work**
Injury while working or in vocational training on job premises	Injury while engaged in personal recreational activity on job premises
Injury while on break or at lunch or in parking lot on job premises	Injury while a visitor (not on official work business) to job premises
Injury while working for pay or compensation, including at home	Homemaker working at homemaking activities
Injury while working as a volunteer law enforcement official etc.	Student in school
Injury while traveling on business, including to/from business contacts	Working for self for no profit (mowing yard, repairing own roof, hobby)
	Commuting to or from work

•42 - Enter the complete address where the injury occurred including zip code.
•43 - Enter a brief but specific and clear description of how the injury occurred. Explain the circumstances or cause of the injury. Specify **type of gun** or **type of vehicle** (e.g., car, bulldozer, train, etc.) when relevant to circumstances. Indicate if more than one vehicle involved; specify type of vehicle decedent was in.
•44 -Specify role of decedent (e.g. driver, passenger). Driver/operator and passenger should be designated for modes other than motor vehicles such as bicycles. Other applies to watercraft, aircraft, animal, or people attached to outside of vehicles (e.g. surfers).

Rationale: Motor vehicle accidents are a major cause of unintentional deaths; details will help determine effectiveness of current safety features and laws.

REFERENCES
For more information on how to complete the medical certification section of the death certificate, refer to tutorial at http://www.TheNAME.org and resources including instructions and handbooks available by request from NCHS, Room 7318, 3311 Toledo Road, Hyattsville, Maryland 20782-2003 or at www.cdc.gov/nchs/about/major/dvs/handbk.htm

REV. 11/2003

Death Certificate

Cause-of-death – Background, Examples, and Common Problems

Accurate cause of death information is important
•to the public health community in evaluating and improving the health of all citizens, and
•often to the family, now and in the future, and to the person settling the decedent's estate.

The cause-of-death section consists of two parts. Part I is for reporting a chain of events leading directly to death, with the immediate cause of death (the final disease, injury, or complication directly causing death) on line a and the underlying cause of death (the disease or injury that initiated the chain of events that led directly and inevitably to death) on the lowest used line. Part II is for reporting all other significant diseases, conditions, or injuries that contributed to death but which did not result in the underlying cause of death given in Part I. The cause-of-death information should be YOUR best medical OPINION. A condition can be listed as "probable" even if it has not been definitively diagnosed.

Examples of properly completed medical certifications

CAUSE OF DEATH (See instructions and examples)

32. PART I. Enter the chain of events--diseases, injuries, or complications--that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary.

		Approximate interval: Onset to death
IMMEDIATE CAUSE (Final disease or condition ——> resulting in death)	a. Rupture of myocardium	Minutes
	Due to (or as a consequence of):	
Sequentially list conditions, if any, leading to the cause listed on line a. Enter the UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST	b. Acute myocardial infarction	6 days
	Due to (or as a consequence of):	
	c. Coronary artery thrombosis	5 years
	Due to (or as a consequence of):	
	d. Atherosclerotic coronary artery disease	7 years

PART II. Enter other significant conditions contributing to death but not resulting in the underlying cause given in PART I

Diabetes, Chronic obstructive pulmonary disease, smoking

33. WAS AN AUTOPSY PERFORMED? ■ Yes ☐ No

34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? ■ Yes ☐ No

35. DID TOBACCO USE CONTRIBUTE TO DEATH? ■ Yes ☐ Probably ☐ No ☐ Unknown

36. IF FEMALE: ■ Not pregnant within past year ☐ Pregnant at time of death ☐ Not pregnant, but pregnant within 42 days of death ☐ Not pregnant, but pregnant 43 days to 1 year before death ☐ Unknown if pregnant within the past year

37. MANNER OF DEATH ■ Natural ☐ Homicide ☐ Accident ☐ Pending investigation ☐ Suicide ☐ Could not be determined

CAUSE OF DEATH (See instructions and examples)

32. PART I. Enter the chain of events--diseases, injuries, or complications--that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary.

		Approximate interval: Onset to death
IMMEDIATE CAUSE (Final disease or condition ——> resulting in death)	a Aspiration pneumonia	2 Days
	Due to (or as a consequence of):	
Sequentially list conditions, if any, leading to the cause listed on line a. Enter the UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST	b Complications of coma	7 weeks
	Due to (or as a consequence of):	
	c. Blunt force injuries	7 weeks
	Due to (or as a consequence of):	
	d. Motor vehicle accident	7 weeks

PART II. Enter other significant conditions contributing to death but not resulting in the underlying cause given in PART I

33. WAS AN AUTOPSY PERFORMED? ■ Yes ☐ No

34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? ■ Yes ☐ No

35. DID TOBACCO USE CONTRIBUTE TO DEATH? ☐ Yes ☐ Probably ■ No ☐ Unknown

36. IF FEMALE: ☐ Not pregnant within past year ☐ Pregnant at time of death ☐ Not pregnant, but pregnant within 42 days of death ☐ Not pregnant, but pregnant 43 days to 1 year before death ☐ Unknown if pregnant within the past year

37. MANNER OF DEATH ☐ Natural ☐ Homicide ■ Accident ☐ Pending investigation ☐ Suicide ☐ Could not be determined

38. DATE OF INJURY (Mo/Day/Yr) (Spell Month)	39. TIME OF INJURY	40. PLACE OF INJURY (e.g., Decedent's home; construction site; restaurant; wooded area)	41. INJURY AT WORK?
August 15, 2003	Approx. 2320	road side near state highway	☐ Yes ■ No

42. LOCATION OF INJURY State: Missouri City or Town: near Alexandria

Street & Number: mile marker 17 on state route 46s Apartment No.: Zip Code:

43. DESCRIBE HOW INJURY OCCURRED:

Decedent driver of van, ran off road into tree

44. IF TRANSPORTATION INJURY, SPECIFY ■ Driver/Operator ☐ Passenger ☐ Pedestrian ☐ Other (Specify)

Common problems in death certification

The elderly decedent should have a clear and distinct etiological sequence for cause of death, if possible. Terms such as senescence, infirmity, old age, and advanced age have little value for public health or medical research. Age is recorded elsewhere on the certificate. When a number of conditions resulted in death, the physician should choose the single sequence that, in his or her opinion, best describes the process leading to death, and place any other pertinent conditions in Part II. If after careful consideration the physician cannot determine a sequence that ends in death, then the medical examiner or coroner should be consulted about conducting an investigation or providing assistance in completing the cause of death.

The infant decedent should have a clear and distinct etiological sequence for cause of death, if possible. "Prematurity" should not be entered without explaining the etiology of prematurity. Maternal conditions may have initiated or affected the sequence that resulted in infant death, and such maternal causes should be reported in addition to the infant causes on the infant's death certificate (e.g., Hyaline membrane disease due to prematurity, 28 weeks due to placental abruption due to blunt trauma to mother's abdomen).

When SIDS is suspected, a complete investigation should be conducted, typically by a medical examiner or coroner. If the infant is under 1 year of age, no cause of death is determined after scene investigation, clinical history is reviewed, and a complete autopsy is performed, then the death can be reported as Sudden Infant Death Syndrome.

When processes such as the following are reported, additional information about the etiology should be reported:

Abscess
Abdominal hemorrhage
Adhesions
Adult respiratory distress syndrome
Acute myocardial infarction
Altered mental status
Anemia
Anoxia
Anoxic encephalopathy
Arrhythmia
Ascites
Aspiration
Atrial fibrillation
Bacteremia
Bedridden
Biliary obstruction
Bowel obstruction
Brain injury
Brain stem herniation
Carcinogenesis
Carcinomatosis
Cardiac arrest
Cardiac dysrhythmia
Cardiomyopathy
Cardiopulmonary arrest
Cellulitis
Cerebral edema
Cerebrovascular accident
Cerebellar tonsillar herniation
Chronic bedridden state
Cirrhosis
Coagulopathy
Compression fracture
Congestive heart failure
Convulsions
Decubiti
Dehydration
Dementia (when not otherwise specified)
Diarrhea
Disseminated intravascular coagulopathy
Dysrhythmia
End-stage liver disease
End-stage renal disease
Epidural hematoma
Exsanguination
Failure to thrive
Fracture
Gangrene
Gastrointestinal hemorrhage
Heart failure
Hemothorax
Hepatic failure
Hepatitis
Hepatorenal syndrome
Hyperglycemia
Hyperkalemia
Hypovolemic shock
Hyponatremia
Hypotension
Immunosuppression
Increased intracranial pressure
Intracranial hemorrhage
Malnutrition
Metabolic encephalopathy
Multi-organ failure
Multi-system organ failure
Myocardial infarction
Necrotizing soft-tissue infection
Old age
Open (or closed) head injury
Paralysis
Pancytopenia
Perforated gallbladder
Peritonitis
Pleural effusions
Pneumonia
Pulmonary arrest
Pulmonary edema
Pulmonary embolism
Pulmonary insufficiency
Renal failure
Respiratory arrest
Seizures
Sepsis
Septic shock
Shock
Starvation
Subdural hematoma
Subarachnoid hemorrhage
Sudden death
Thrombocytopenia
Uncal herniation
Urinary tract infection
Ventricular fibrillation
Ventricular tachycardia
Volume depletion

If the certifier is unable to determine the etiology of a process such as those shown above, the process must be qualified as being of an unknown, undetermined, probable, presumed, or unspecified etiology so it is clear that a distinct etiology was not inadvertently or carelessly omitted.

The following conditions and types of death might seem to be specific or natural but when the medical history is examined further may be found to be complications of an injury or poisoning (possibly occurring long ago). Such cases should be reported to the medical examiner/coroner.

Asphyxia
Bolus
Choking
Drug or alcohol overdose/drug or alcohol abuse
Epidural hematoma
Exsanguination
Fall
Fracture
Hip fracture
Hyperthermia
Hypothermia
Open reduction of fracture
Pulmonary emboli
Seizure disorder
Sepsis
Subarachnoid hemorrhage
Subdural hematoma
Surgery
Thermal burns/chemical burns

REV. 11/2003

FUNERAL DIRECTOR INSTRUCTIONS for selected items on U.S.

Standard Certificate of Death (For additional information concerning all items on certificate see Funeral Directors' Handbook on Death Registration)

ITEM 1. DECEDENT'S LEGAL NAME
Include any other names used by decedent, if substantially different from the legal name, after the abbreviation AKA (also known as) e.g. Samuel Langhorne Clemens AKA Mark Twain, **but not** Jonathon Doe AKA John Doe

ITEM 5. DATE OF BIRTH
Enter the full name of the month (January, February, March etc.) Do not use a number or abbreviation to designate the month.

ITEM 7A-G. RESIDENCE OF DECEDENT (information divided into seven categories)
Residence of decedent is the place where the decedent actually resided. The place of residence is not necessarily the same as "home state" or "legal residence". Never enter a temporary residence such as one used during a visit, business trip, or vacation. Place of residence during a tour of military duty or during attendance at college is considered permanent and should be entered as the place of residence. If the decedent had been living in a facility where an individual usually resides for a long period of time, such as a group home, mental institution, nursing home, penitentiary, or hospital for the chronically ill, report the location of that facility in item 7. If the decedent was an infant who never resided at home, the place of residence is that of the parent(s) or legal guardian. **Never** use an acute care hospital's location as the place of residence for any infant. If Canadian residence, please specify Province instead of State.

ITEM 10. SURVIVING SPOUSE'S NAME
If the decedent was married at the time of death, enter the full name of the surviving spouse. If the surviving spouse is the wife, enter her name prior to first marriage. This item is used in establishing proper insurance settlements and other survivor benefits.

ITEM 12. MOTHER'S NAME PRIOR TO FIRST MARRIAGE
Enter the name used prior to first marriage, commonly known as the maiden name. This name is useful because it remains constant throughout life.

ITEM 14. PLACE OF DEATH
The place where death is pronounced should be considered the place where death occurred. If the place of death is unknown but the body is found in your State, the certificate of death should be completed and filed in accordance with the laws of your State. Enter the place where the body is found as the place of death.

ITEM 51. DECEDENT'S EDUCATION *(Check appropriate box on death certificate)*
Check the box that corresponds to the highest level of education that the decedent completed. **Information in this section will not appear on the certified copy of the death certificate. This information is used to study the relationship between mortality and education (which roughly corresponds with socioeconomic status). This information is valuable in medical studies of causes of death and in programs to prevent illness and death.**

ITEM 52. WAS DECEDENT OF HISPANIC ORIGIN? *(Check "No" or appropriate "Yes" box)*
Check "No" or check the "Yes" box that best corresponds with the decedent's ethnic Spanish identity as given by the informant. Note that "Hispanic" is not a race and item 53 must also be completed. Do not leave this item blank. With respect to this item, "Hispanic" refers to people whose origins are from Spain, Mexico, or the Spanish-speaking Caribbean Islands or countries of Central or South America. Origin includes ancestry, nationality, and lineage. There is no set rule about how many generations are to be taken into account in determining Hispanic origin; it may be based on the country of origin of a parent, grandparent, or some far-removed ancestor. Although the prompts include the major Hispanic groups, other groups may be specified under "other". "Other" may also be used for decedents of multiple Hispanic origin (e.g. Mexican-Puerto Rican). **Information in this section will not appear on the certified copy of the death certificate. This information is needed to identify health problems in a large minority population in the United States. Identifying health problems will make it possible to target public health resources to this important segment of our population.**

ITEM 53. RACE *(Check appropriate box or boxes on death certificate)*
Enter the race of the decedent as stated by the informant. Hispanic is not a race; information on Hispanic ethnicity is collected separately in item 52. American Indian and Alaska Native refer only to those native to North and South America (including Central America) and does not include Asian Indian. Please specify the name of enrolled or principal tribe (e.g., Navajo, Cheyenne, etc.) for the American Indian or Alaska Native. For Asians check Asian Indian, Chinese, Filipino, Japanese, Korean, Vietnamese, or specify other Asian group; for Pacific Islanders check Guamanian or Chamorro, Samoan, or specify other Pacific Island group. If the decedent was of mixed race, enter each race (e.g., Samoan-Chinese-Filipino or White, American Indian). **Information in this section will not appear on the certified copy of the death certificate. Race is essential for identifying specific mortality patterns and leading causes of death among different racial groups. It is also used to determine if specific health programs are needed in particular areas and to make population estimates.**

ITEMS 54 AND 55. OCCUPATION AND INDUSTRY
Questions concerning occupation and industry must be completed for all decedents 14 years of age or older. This information is useful in studying deaths related to jobs and in identifying any new risks. For example, the link between lung disease and lung cancer and asbestos exposure in jobs such as shipbuilding or construction was made possible by this sort of information on death certificates. **Information in this section will not appear on the certified copy of the death certificate.**

ITEM 54. DECEDENT'S USUAL OCCUPATION
Enter the usual occupation of the decedent. This is not necessarily the last occupation of the decedent. Never enter "retired". Give kind of work decedent did during most of his or her working life, such as claim adjuster, farmhand, coal miner, janitor, store manager, college professor, or civil engineer. If the decedent was a homemaker at the time of death but had worked outside the household during his or her working life, enter that occupation. If the decedent was a homemaker during most of his or her working life, and never worked outside the household, enter "homemaker". Enter "student" if the decedent was a student at the time of death and was never regularly employed or employed full time during his or her working life. **Information in this section will not appear on the certified copy of the death certificate.**

ITEM 55. KIND OF BUSINESS/INDUSTRY
Kind of business to which occupation in item 54 is related, such as insurance, farming, coal mining, hardware store, retail clothing, university, or government. DO NOT enter firm or organization names. If decedent was a homemaker as indicated in item 54, then enter either "own home" or "someone else's home" as appropriate. If decedent was a student as indicated in item 54, then enter type of school, such as high school or college, in item 55. **Information in this section will not appear on the certified copy of the death certificate.**

NOTE: This recommended standard death certificate is the result of an extensive evaluation process. Information on the process and resulting recommendations as well as plans for future activities is available on the Internet at: http://www.cdc.gov/nchs/vital_certs_rev.htm.

REV. 11/2003

6 Medical Glossary

Glossary/Terms	Explanation/Definition
Actinic keratosis	A scaly or crusty lesion that forms on the skin surface related to sun exposure. Known to be "Pre-skin cancers"
Adenomas (e.g. fibroadenomas)	A benign tumor of a glandular structure or of glandular origin
Alveolar ducts	The somewhat enlarged terminal sections of the bronchioles that branch into the terminal alveoli
Alveoli	Final branching of the respiratory tree and act as the primary gas exchange units of the lung.
Amyloidosis	A disorder characterized by the deposition of insoluble fibrous proteins in organs or tissues of the animal body
Atypia	The condition of being uncharacteristic or lacking uniformity
Basal Cell	One of the innermost cells of the deeper epidermis of the skin
Basophils	A white blood cell with basophilic granules that is similar in function to a mast cell
Bronchodilator	relating to or causing expansion of the bronchial air passages
Cor Pulmonale	Failure of the right side of the heart caused by long-term high blood pressure in the pulmonary arteries and right ventricle of the heart
Cyanosis	Bluish or purplish tinge to the skin and mucous membranes due to deficient oxygenation of the blood
Cytogenetics	A branch of biology that deals with the study of heredity and variation by the methods of both cytology and genetics
Cytology	A branch of biology dealing with the structure, function, multiplication, pathology, and life history of cells

Glossary/Terms	Explanation/Definition
Dyscrasia	Dyscrasias is a nonspecific term that refers to any disease or disorder. However, it usually refers to blood diseases.
Dysplasis	Abnormal growth or development
Dyspnea	Difficult or labored respiration
Effusions	The escape of a fluid from anatomical vessels by rupture or exudation
Eosinophils	White blood cells that are one of the immune system components responsible for combating infection and parasites in vertebrates
Fibrosed	To form fibrous or SCAR tissue
Granulomas	A mass or nodule of chronically inflamed tissue with granulations that is usually associated with an infective process
Hematologic	Of or relating to blood or to hematology
Heme	The deep red, nonprotein, ferrous component of hemoglobin
Hilar nodes	Any of the lymph nodes in the hilum or the triangular depression or indented region at the junction of each lung and its bronchi.
Histopathology	A branch of pathology concerned with the tissue changes characteristic of disease
Hyperplasia	An abnormal or unusual increase in the elements composing a part
Immunophenotypes	The immunochemical and immunohistological characteristics of a cell or group of cells
In-situ	In the natural or original position or place
Interpolation	A method of constructing new data points within the range of a discrete set of known data points.
Interstitial	Affecting the interstitial tissues of an organ or part
Leukocytosis	An increase in the number of white blood cells in the circulating blood that occurs normally or abnormally

Glossary/Terms	Explanation/Definition
Leukopenia	A decrease in the number of circulating white blood cells
Lipomas	A tumor of fatty tissue
Lymphedema	Edema due to faulty lymphatic drainage
Lymphocytes	Any of the colorless weakly motile cells that originate from stem cells and differentiate in lymphoid tissue that are the typical cellular elements of lymph, that include the cellular mediators of immunity, and that constitute 20 to 30 percent of the white blood cells of normal human blood
Megakaryocytes	is a bone marrow cell responsible for the production of blood platelets
Metaplasia	The change in the type of adult cells in a tissue to a form abnormal for that tissue
Metastasis	Refers to the spread of cancer to other organs.
Methacholine	Drug used to diagnose hypersensitivity of the bronchial air passages
Monocytes	Large white blood cell with finely granulated chromatin dispersed throughout the nucleus that is formed in the bone marrow, enters the blood, and migrates into the connective tissue where it differentiates into macrophage white blood cells
Monocytic	A white blood cell that increases during a variety of conditions including severe infections
Myeloblastic	A large mononuclear non-granular bone marrow cell; *especially*: one that is a precursor of a myelocyte
Myelodysplastic Syndromes	A group of diseases in which the production of blood cells by the bone marrow is disrupted
Neutrophils	The most common type of white blood cell
Opacities	Shadows referred to by Radiologists - On chest x-rays may be benign or malignant.

Glossary/Terms	Explanation/Definition
Parenchyma	The essential and distinctive tissue of an organ or an abnormal growth as distinguished from its supportive framework
Parietal	Refers to the wall e.g. chest wall.
Peritoneum	The serous membrane that forms the lining of the abdominal cavity
Pleural disease	Abnormality in the tissue that lines the lung.
Pleural scarring	Asbestosis is characterized by pleural scarring that is brought on by asbestos dust exposure
Pluripotential	Capable of differentiating into one of many cell types
Pruritus	Localized or generalized itching due to irritation of sensory nerve endings
Rhinitis	Inflammation of the mucous membrane of the nose marked especially by rhinorrhea, nasal congestion and itching, and sneezing
S.O.A.P	Subjective, Objective, Assessment, Plan
Sarcoidosis	A chronic disease of unknown cause that is characterized by the formation of nodules resembling true tubercles especially in the lymph nodes, lungs, bones, and skin
Silicoproteinosis	Associated with silica dust exposure that is in acute silicosis
Squamous Cell	A form of cancer of the carcinoma type that may occur in many different organs, including the skin, lips, mouth, esophagus, urinary bladder, prostate, and lungs
Uremia	Accumulation in the blood of constituents normally eliminated in the urine that produces a severe toxic condition and usually occurs in severe kidney disease
Visceral	refers to the "organ" e.g. lung

7 Common Abbreviations

A	
AAA	abdominal aortic aneurysm
A-a gradient	alveolar to arterial gradient
AAD	antibiotic-associated diarrhea
AAO	alert, awake, and oriented
A&O	alert & oriented
AAS	acute abdominal series
ABD	abdomen
ABG	arterial blood gas
AC	before eating
ACLS	advanced cardiac life support
ACTH	adrenocorticotropic hormone
ADH	anti-diuretic hormone
ADR	adverse drug reaction. | acute dystonic reaction
ad lib	as much as needed
AED	antiepileptic drug
AF	atrial fibrillation or afebrile
AFB	acid-fast bacilli
AFP	alpha-fetoprotein
A /G	albumin/globulin ratio
AI	aortic insufficiency
AKA	above the knee amputation
ALD	alcoholic liver disease
ALL	acute lymphocytic leukemia
amb	ambulate
AML	acute myelogenous leukemia
ANA	antinuclear antibody
ANS	autonomic nervous system

AOB	alcohol on breath
AODM	adult onset diabetes mellitus
AP	anteroposterior or abdominal - perineal
ARDS	acute respiratory distress syndrome
ARF	acute renal failure
AS	aortic stenosis
ASAP	as soon as possible
ASCVD	atherosclerotic cardiovascular disease
ASD	atrial septal defect
ASHD	atherosclerotic heart disease
AV	atrioventricular
A-V	arteriovenous
A-VO2	arteriovenous oxygen

B	
BBB	bundle branch block
BCAA	branched chain amino acids
BE	barium enema
BEE	basal energy expenditure
bid	twice a day
BKA	below the knee amputation
BM	bone marrow or bowel movement
BMR	basal metabolic rate
BOM	bilateral otitis media
BP	blood pressure
BPH	benign prostatic hypertrophy
BPM	beats per minute
BRBPR	bright red blood per rectum
BRP	bathroom priviledges
BS	bowel or breath sounds

BUN	blood urea nitrogen
BW	body weight
BX	biopsy

C	
c	with
C&S	culture and sensitivity
CA	cancer
Ca	calcium
CAA	crystalline amino acids
CABG	coronary artery bypass graft
CAD	coronary artery disease
CAT	computerized axial tomography
CBC	complete blood count
CBG	capillary blood gas
CC	chief complaint
CCU	clean catch urine or cardiac care unit
CCV	critical closing volume
CF	cystic fibrosis
CGL	chronic granulocytic leukemia
CHF	congestive heart failure
CHO	carbohydrate
CI	cardiac index
CML	chronic myelogenous leukemia
CMV	cytomegalovirus
CN	cranial nerves
CNS	central nervous system
CO	cardiac output
C/O	complaining of

COLD	chronic obstructive lung disease
COPD	chronic obstructive pulmonary disease
CP	chest pain or cerebral palsy
CPAP	continuous positive airway pressure
CPK	creatine phosphokinase
CPR	cardiopulmonary resuscitation
CRCL	creatinine clearance
CRF	chronic renal failure
CRP	C-reactive protein
CSF	cerebrospinal fluid
CT	computerized tomography
CVA	cerebrovascular accident or costovertebral angle
CVAT	CVA tenderness
CVP	central venous pressure
CXR	chest X-ray

D	
DAT	diet as tolerated
DAW	dispense as written
DC	discontinue or discharge
D&C	dilation and curettage
DDx	differential diagnosis
D5W	5% dextrose in water
DI	diabetes insipidus
DIC	disseminated intravascular coagulopathy
DIP	distal interphalangeal joint
DJD	degenerative joint disease
DKA	diabetic ketoacidosis
dL	deciliter
DM	diabetes mellitus

DNR	do not resuscitate
DOA	dead on arrival
DOE	dyspnea on exertion
DPL	diagnostic peritoneal lavage
DPT	diphtheria, pertussis, tetanus
DTR	deep tendon reflexes
DVT	deep venous thrombosis
DX	diagnosis

E	
EAA	essential amino acids
EBL	estimated blood loss
ECG	electrocardiogram
ECT	electroconvulsive therapy
EFAD	essential fatty acid deficiency
EMG	Electromyogram
EMV	eyes, motor, verbal response (Glasgow coma scale)
ENT	ears, nose, and throat
EOM	extraocular muscles
ESR	erythrocyte sedimentation rate
ET	endotracheal
ETT	endotracheal tube
ERCP	endoscopic retrograde cholangio -pancreatography
ETOH	ethanol
EUA	examination under anesthesia

F	
FBS	fasting blood sugar
FEV	forced expiratory volume
FFP	fresh frozen plasma

FRC	functional residual capacity
FTT	failure to thrive
FU	follow-up
FUO	fever of unknown origin
FVC	forced vital capacity
Fx	fracture

G	
GC	gonorrhea
GETT	general by endotracheal tube
GFR	glomerular filtration rate
GI	gastrointestinal
gr	grain; 1 grain = 65mg. Therefore Vgr = 325mg
GSW	gun shot wound
gt or gtt	drops
GTT	glucose tolerance test
GU	genitourinary
GXT	graded exercise tolerance (Stress test)

H	
HA	headache
HAA	hepatitis B surface antigen
HAV	hepatitis A virus
HBP	high blood pressure
HCG	human chorionic gonadotropin
HCT	hematocrit
HDL	high density lipoprotein
HEENT	head, eyes, ears, nose, throat
Hgb	hemoglobin

H/H	henderson- hasselbach equation or hemoglobin/ hematocrit
HIV	human immunodeficiency virus
HLA	histocompatibility locus antigen
HJR	hepatojugular reflex
HO	history of
HOB	head of bed
HPF	high power field
HPI	history of present illness
HR	heart rate
HS	at bedtime
HSM	hepatosplenomegaly
HTLV-III	human lymphotropic virus, type III (AIDS agent, HIV)
HSV	herpes simplex virus
HTN	hypertension
Hx	history

I	
I&D	incision and drainage
I&O	intake and output
ICS	intercostal space
ICU	intensive care unit
ID	infectious disease or identification
IDDM	insulin dependent diabetes mellitus
IG	immunoglobulin
IHSS	idiopathic hypertropic subaortic stenosis
IM	intramuscular
IMV	intermittent mandatory ventilation
INF	intravenous nutritional fluid
IPPB	intermittent positive pressure breathing

IRBBB	incomplete right bundle branch block
IRDM	insulin resistant diabetes mellitus
IT	interthecal
ITP	idiopathic thrombocytopenic purpura
IV	intravenous
IVC	intravenous cholangiogram | inferior vena cava
IVP	intravenous pyelogram

J	
JODM	juvenile onset diabetes mellitus
JVD	jugular venous distention

K	
KOR	keep open rate
KUB	kidneys, ureters, bladder
KVO	keep vein open

L	
L	left
LAD	left axis deviation or left anterior descending
LAE	left atrial enlargement
LAHB	left anterior hemiblock
LAP	left atrial pressure or leukocyte alkaline phosphatase
LBBB	left bundle branch block
LDH	lactate dehydrogenase
LE	lupus erythematosus
LIH	left inguinal hernia
LLL	left lower lobe
LMP	last menstrual period

LNMP	last normal menstrual period
LOC	loss of consciousness or level of consciousness
LP	lumbar puncture
LPN	licensed practical nurse
LUL	left upper lobe
LUQ	Left Upper Quadrant
LV	left ventricle
LVEDP	left ventricular end diastolic pressure
LVH	left ventricular hypertrophy

M	
MAO	monoamine oxidase
MAP	mean arterial pressure
MAST	medical antishock trousers
MBT	maternal blood type
MCH	mean cell hemoglobin
MCHC	mean cell hemoglobin concentration
MCV	mean cell volume
MI	myocardial infarction or mitral insufficiency
mL	milliliter
MLE	midline episiotomy
MMEF	maximal mid expiratory flow
mmol	millimole
MMR	measles, mumps, rubella
MRI	magnetic resonance imaging
MRSA	methicillin resistant staph aureus
MS	multiple sclerosis or mitral stenosis, or morphine sulfate
MSSA	methicillin-sensitive staph aureus
MVA	motor vehicle accident
MVI	multivitamin injection

MVV	maximum voluntary ventilation

N	
NAD	no active disease
NAS	no added salt
NCV	nerve conduction velocity
NED	no evidence of recurrent disease
ng	nanogram
NG	nasogastric
NIDDM	non-insulin dependent diabetes mellitus
NKA	no known allergies
NKDA	no known drug allergies
NMR	nuclear magnetic resonance
NPO	nothing by mouth
NRM	no regular medications
NSAID	non-steroidal anti- inflammatory drugs
NSR	normal sinus rhythm
NT	nasotracheal

O	
OB	obstetrics
OCG	oral cholecystogram
OD	overdose or right eye
OM	otitis media
OOB	out of bed
OOP	out of plaster
OPV	oral polio vaccine
OR	operating room
OS	left eye
OU	both eyes

P	
P	para
PA	posteroanterior
PAC	Premature atrial contraction
PAO2	alveolar oxygen
PaO2	peripheral arterial oxygen content
PAP	pulmonary artery pressure
PAT	paroxysymal atrial tachycardia
P&PD	percussion and postural drainage
PC	after eating
PCWP	pulmonary capillary wedge pressure
PDA	patent ductus arteriosus
PDR	physicians desk reference
PE	pulmonary embolus, or physical exam or pleural effusion
PEEP	positive end expiratory pressure
PFT	pulmonary function tests
pg	picogram
PI	pulmonic insufficiency disease
PKU	phenylketonuria
PMH	previous medical history
PMI	point of maximal impulse
PMN	polymorphonuclear leukocyte (neutrophil)
PND	paroxysmal nocturnal dyspnea
PO	by mouth
POD	post-op day
PP	postprandial or pulsus paradoxus
PPD	purified protein derivative
PR	by rectum
PRBC	packed red blood cells
PRN	as needed

PS	pulmonic stenosis
PT	prothrombin time, or physical therapy
Pt	patient
PTCA	percutaneous transluminal coronary angioplasty
PTH	parathyroid hormone
PTHC	percutanous transhepatic cholangiogram
PTT	partial thromboplastin time
PUD	peptic ulcer disease
PVC	premature ventricular contraction
PVD	peripheral vascular disease

Q	
q	every (e.g. q6h = every 6 hours)
qd	every day
qh	every hour
q4h, q6h....	every 4 hours, every 6 hours etc.
qid	four times a day
QNS	quantity not sufficient
qod	every other day
Qs/Qt	shunt fraction
Qt	total cardiac output

R	
R	right
RA	rheumatoid arthritis or right atrium
RAD	right atrial axis deviation
RAE	right atrial enlargement
RAP	right atrial pressure
RBBB	right bundle branch block
RBC	red blood cell

RBP	retinol-binding protein
RDA	recommended daily allowance
RDW	red cell distribution width
RIA	radioimmunoassay
RIH	right inguinal hernia
RLL	right lower lobe
RLQ	right lower quadrant
RML	right middle lobe
RNA	ribonucleic acid
R/O	rule out
ROM	range of motion
ROS	review of systems
RPG	retrograde pyelogram
RRR	regular rate and rhythm
RT	respiratory or radiation therapy
RTA	renal tubular acidosis
RTC	return to clinic
RU	resin uptake
RUG	retrograde urethogram
RUL	right upper lobe
RUQ	right upper quadrant
RV	residual volume
RVH	right ventricular hyperthrophy
Rx	treatment

S	
s	without | ss = one-half
SA	sinoatrial
SAA	synthetic amino acid
S&E	sugar and acetone

SBE	subacute bacterial endocarditis
SBFT	small bowel follow through
SBS	short bowel syndrome
SCr	serum creatinine
SEM	systolic ejection murmur
SG	Swan-Ganz
SGA	small for gestational age
SGGT	serum gamma- glutamyl transpeptidase
SGOT	serum glutamic- oxaloacetic transaminase
SGPT	serum glutamic- pyruvic transaminase
SIADH	syndrome of inappropriate antidiuretic hormone
sig	write on label
SIMV	synchronous intermittent mandatory ventilation
sl	sublingual
SLE	systemic lupus erythematous
SMO	slips made out
SOAP	subjective, Objective, Assessment, Plan
SOB	shortness of breath
SQ	subcutaneous
STAT	immediately
SVD	spontaneous vaginal delivery
Sx	symptoms

T	
T&C	type and cross
TAH	total abdominal hysterectomy
T&H	type and hold
TB	tuberculosis
TBG	total binding globulin
Td	tetanus-diphtheria toxoid

TIA	transient ischemic attack
TIBC	total iron binding capacity
tid	three times a day
TIG	tetanus immune globulin
TKO	to keep open
TLC	total lung capacity
TMJ	temporo mandibular joint
TNTC	too numerous to count
TO	telephone order
TOPV	trivalent oral polio vaccine
TPN	total parenteral nutrition
TSH	thyroid stimulating hormone
TT	thrombin time
TTP	thrombotic thrombocytopenic purpura
TU	tuberculin units
TUR	transurethral resection
TURBT	TUR bladder tumors
TURP	transurethral resection of prostate
TV	tidal volume
TVH	total vaginal hysterectomy
tw	twice a week
Tx	treatment, transplant

U	
UA	urinalysis
UAC	uric acid | umbilical artery catheter
UAO	upper airway obstruction
UBD	universal blood donor
UC	ulcerative colitis | umbilical cord
ud	as directed

UFH	unfractionated heparin
UGI	upper gastrointestinal
URI	upper respiratory infection
URQ	upper right quadrant
US	ultrasound
UTI	urinary tract infection
UUN	urinary urea nitrogen
UVA	ultraviolet A light

V	
VAD	venous access device
VC	vital capacity
VCT	venous clotting time
VCUG	voiding cysourethrogram
VDRL	Venereal Disease Research Laboratory (test for syphilis)
VMA	vanillymadelic acid
VO	verbal or voice order
V/Q	ventilation - perfusion
VRE	vancomycin-resistant enterococcus
VSS	vital signs stable
VT	ventricular tachycardia
VV	varicose veins
VW	vessel wall
VWD	von Willebrand's disease
VZV	varicella zoster virus

W	
WB	whole blood
WBC	white blood cell or count
WBR	whole body radiation

WD	well developed
WF	white female
WIA	wounded in action
WID	widow, widower
WM	white male
WN	well nourished
WNL	within normal limits
WO	written order | weeks old | wide open.
WOP	without pain
W.P.	whirlpool
WPW	Wolff-Parkinson-White
W-T-D	wet to dry
W/U	workup

X	
X2d	times 2 days.
XI	eleven
XII	twelve
XL	extended release. | extra large.
XM	crossmatch
XMM	xeromammography
XOM	extraocular movements
XRT	X-ray therapy (radiation therapy)
XS	excessive
XULN	times upper limit of normal

Y	
YF	yellow fever
YLC	youngest living child

yo	years old
YOB	year of birth
yr	year
ytd	year to date

Z	
ZDV	zidovudine
ZE	Zollinger-Ellison
Z-ESR	zeta erythrocyte sedimentation rate
Zn	zinc
ZnO	zinc oxide
ZSB	zero stools since birth

8 Medical Terminology Guide

The following is an excerpt from a training guide used by the American Academy of Professional Coders. The goal is to provide some word derivations to assist the CE in understanding medical terminology.

8.1 Word Endings

Word Endings – Conventions for Changing from Singular to Plural

Medical term ends in	Form Plural by	Example of Singular	Example of Plural
a	e	concha	conchae
ex or ix	ices	calix	calices
is	es	hemarthrosis	hemarthroses
nx	Drop x and add ges	larynx	larynges
on	Drop on and add a	ganglion	ganglia
um	Change um to a	bacterium	bacteria
us	Change us to i	sulcus	sulci

Erlich, Medical Terminology for Health Professionals, page 4

8.2 Building Words from Word Elements

Combining Forms	Meaning
acr/o	extremities, top
cyan/o	blue
cyt/o	cell
derm/o	skin
dermat/o	skin
erythr/o	red
leuk/o	white
melan/o	black, dark
poli/o	gray
xanth/o	yellow

The following examples of prefixes often cause confusion:

Combining Forms	Meaning
a-	without, away from
hypo-	low, decreased
hyper-	high, increased
intra-	within
inter-	between

The following examples of suffixes often cause confusion:

Combining Forms	Meaning
-itis	inflammation
-lysis	reduction or relief of
-megaly	enlarged
-otomy	incision
-ectomy	remove surgically
-ostomy	artificial opening surgically created
-rrhaphy	suture
-rhexis	rupture
-rrahgia	excess flow
-rhea	flowing

8.3 Medical Terms

The medical terms composing this list may be broken down into word elements or parts. This translation will give the approximate meaning of the complete medical term. If further explanation is needed beyond the literal translation, a medical dictionary may be used.

Examples
My/o = muscle
pathy = disease
Myopathy = disease of the muscle

Erthr/o = red
cyte = cell
Erythrocyte = red cell (referring to bloold)

Certain terms or word elements that are consistently associated with the major body systems are listed below:

Terms	Definition	System
Cardi/o	Heart	Cardiovascular
Arteri/o	Arteries	Cardiovascular
Ven/o	Veins	Cardiovascular
Phleb/o	Veins	Cardiovascular
Or/o	Mouth	Digestive
Esophag/o	Esophagus	Digestive
Gastro/	Stomach	Digestive
Enter/o	Small Intestine	Digestive
Col/o	Large Intestine	Digestive
Hepat/o	Liver	Digestive
Pancreat/o	Pancreas	Digestive
Adren/o	Adrenals	Endocrine

Terms	Definition	System
Gonad/o	Gonads	Endocrine
Hem/o	Blood	Hematologic
Hemat/o	Blood	Hematologic
Hidr/o	Sweat Glands	Integumentary
Seb/o	Sebaceous Glands	Integumentary
Dermato/o	Skin	Integumentary
Cutane/o	Skin	Integumentary
Lymph/o	Lymphatic Structures/Fluids	Lymphatic
Splen/o	Spleen	Lymphatic
My/o	Muscles	Musculoskeletal
Fasci/o	Fascia	Musculoskeletal
Ten/o	Tendons	Musculoskeletal
Oste/o	Bones	Musculoskeletal
Arthro/o	Joints	Musculoskeletal
Chondr/o	Cartilage	Musculoskeletal
Neru/o	Nerve	Nervous
Encephla/o	Brain	Nervous
Myel/o	Spinal Cord	Nervous
Ocul/o	Eyes	Nervous/Sense
Ophthalm/o	Eyes	Nervous/Sense
Ot/o	Ears	Nervous/Sense
Oophor/o	Ovaries	Reproductive/Female
Hyster/o	Uterus	Reproductive/Female
Metr/o	Uterus	Reproductive/Female
Metri/o	Uterus	Reproductive/Female
Uter/o	Uterus	Reproductive/Female
Nephr/o	Kidneys	Urinary
Ureter/o	Ureters	Urinary
Cyst/o	Bladder	Urinary
Vesic/o	Bladder	Urinary
Urethro/o	Urethra	Urinary

9 Information Utilized by Physicians

The following referenced is from "Reference Guide on Medical Terminology" pgs. 452 – 484 and available on line at:

http://www.fjc.gov/public/home.nsf/autogoogle?openform&url=http://google.fjc.gov/search?q=manual&btnG=Search&sort=date%3AD%3AL%3Ad1&output=xml_no_dtd&ie=UTF-8&oe=UTF-8&client=www&proxystylesheet=www&site=www

While physicians dealing with diagnosis and treatment tend to think in terms of both internal and external causation, courts are usually asked to determine the role of causes that are external to the individual. Generally, physicians focus on causal elements that can be addressed through medical treatment or through changes in lifestyle or diet; courts focus primarily on causal elements for which a litigant or other party might be held responsible. For example, a workers' compensation case might concern the role of physiological stress at work in causing underlying heart disease, or the role of carbon monoxide in triggering a specific heart attack.[46] Identification of those kinds of causes depends on information concerning quantification of risks in the workplace environment, as well as on the medical literature on causation, including the psychological, toxicological, and epidemiological literature.[47] To determine general causation, the expert must review the pertinent literature, as familiarity with this literature is key to expert opinion. For example, since many cardiologists advise patients on returning to work after a heart attack, they will often be familiar with the literature on work-based risks and cardiovascular disease, whereas most other physicians, who deal with this question less frequently, would need to devote some time to study before evaluating such a special consideration.

III. Information Utilized by Physicians

Physicians rely on the following diverse sources of information in arriving at a diagnosis, determining a course of treatment, and exploring causation: the patient history (information derived directly from the patient), patient records, physical examination, and diagnostic tests.[48]

A. Patient History (from the Patient)

The patient history is one of the primary and most useful tools in the practice of clinical medicine. It is usually divided into present illness (including both subjective reports and medical documentation) and past medical problems, with or without medical documentation.[49]

As obtained by the examining physician, the patient history is extremely important in evaluating the patient's condition, determining what medical tests may be warranted, arriving at a diagnosis, and recommending an appropriate

46. *See, e.g.*, Fiore v. Consolidated Freightways, 659 A.2d 436 (N.J. 1995) (truck driver's workers' compensation case claiming that his heart disease was caused by occupational exposure to carbon monoxide fumes remanded so that parties could provide more reliable exposure evidence).

47. *See* Cullen et al., *supra* note 19, at 220–21.

48. *See* Jerome P. Kassirer & Richard I. Kopelman, Learning Clinical Reasoning 4 (1991).

49. Barbara Bates et al., A Guide to Physical Examination and History Taking 2–3 (6th ed. 1995).

course of treatment. Even in this era of sophisticated medical testing protocols, it is estimated that 70% of significant patient problems can be identified, although not necessarily confirmed, by a thorough patient history.[50]

A thorough patient history includes not only present illness and past medical problems, but also aspects of medical, occupational, personal, and familial background that are relevant to the present problem. Moreover, patient histories may identify common patterns of illness among individuals with a common lifestyle or exposure element, such as reproductive problems in individuals occupationally exposed to lead. Although patient histories are important in determining a diagnosis, and useful in epidemiological studies of both acute and chronic diseases, there is no validated and widely used patient history questionnaire with which to begin the diagnostic process,[51] perhaps because the history-taking process is so iterative and intertwined with hypothesis testing.

Despite the absence of a standard patient history questionnaire, there is general agreement that a useful adult patient history includes the following information:

1. identification (e.g., name, sex, age);
2. chief complaint and history of the present illness;
3. medical history (e.g., injuries, medical conditions and diseases, surgical procedures);
4. lifestyle characteristics (e.g., use of nicotine, alcohol, and other drugs; exposures in the home);
5. familial health (e.g., medical conditions and diseases of relatives); and
6. occupational history (e.g., present and previous employment, exposures).[52]

While more recent events or those that more directly appear pertinent to the particular presenting symptoms of a patient will usually be given the most attention, historic events or familial history may provide insight into diagnosis and prognosis.[53] This is particularly true when the physician is considering exposure–disease relationships with a long latency, such as in asbestos-related disease or inherited predispositions for malignancy.[54]

1. Symptomatology

Symptoms are by definition subjective, since they are self-reported by the patient in his or her own words. Because symptoms that preoccupy the patient are not always the most relevant to diagnosis, the physician will often need to ask

50. *See* Mark H. Swartz, Textbook of Physical Diagnosis: History and Examination 667 (3d ed. 1998).

51. Office of Tech. Assessment, U.S. Congress, Reproductive Health Hazards in the Workplace app. B at 365 (1985).

52. *See, e.g.*, Bates et al., *supra* note 49, at 3–7, 16–17.

53. *See, e.g., id.* at 637–39.

54. *See* Thomas E. Andreoli et al., Cecil Essentials of Medicine 152 (3d ed. 1993).

the patient about symptoms that are particularly useful for diagnosis, but not of particular concern to the patient. Generally, patients will be asked to characterize symptoms by their location, intensity, frequency, exacerbating factors, ameliorating factors, and novelty.[55]

As a report of the patient's own experience, symptoms are uniquely valuable, but they are also subject to various sources of bias and error, both intentional and unintentional. A competent diagnostician can take sources of error into account, but for some symptoms, such as severity of pain, or when the first severe attack of shortness of breath occurred, it is usually not possible to objectively verify the patient's reports. The physician's skill, knowledge, and experience with the particular area of concern is critical in obtaining an accurate and meaningful history.[56] Physicians are accustomed to reaching a subjective conclusion regarding the quality and reliability of the history they obtain from the patient.

2. Environmental and Occupational History

Consideration of occupational and environmental causation in diagnosis has long been recommended to physicians, but more specific attention to the environmental and occupational history as part of the medical workup has recently been emphasized, with the degree of detail depending on the clinical situation.[57]

If the medical workup indicates a potential occupational or environmental disease, the physician should explore the patient's potential exposures in more detail.[58] Although the physician often will not have measures of environmental exposure, information about the level of exposure can be inferred in certain instances from the description of the workplace and work processes; the duration of exposure; correlates, such as eye irritation, headache, or odor; the size of a room or other enclosure; the presence of windows or other ventilation; and other activities occurring nearby.[59]

55. *See, e.g.*, Bates et al., *supra* note 49, at 635, 645–47.

56. *See* Anthony S. Fauci et al., *The Practice of Medicine, in* 1 Principles of Internal Medicine, *supra* note 42, at 1, 2; Lee Goldman, *Quantitative Aspects of Clinical Reasoning, in* 1 Principles of Internal Medicine, *supra* note 42, at 9, 9.

57. *See* Hu & Speizer, *supra* note 42, at 19; Environmental Medicine: Integrating a Missing Element into Medical Education 5–11 (Andrew M. Pope & David P. Rall eds., 1995).

58. Establishing exposure is usually deemed necessary to a plaintiff's toxic injury claim, and the existence or degree of exposure to the agent is often at issue. *See, e.g., In re* Paoli R.R. Yard PCB Litig., 916 F.2d 829 (3d Cir. 1990) (environmental exposure to polychlorinated biphenyls (PCBs) contested), *cert. denied*, 499 U.S. 961 (1991).

59. *See* Hu & Speizer, *supra* note 42, at 19; Frank E. Speizer, *Environmental Lung Diseases, in* 2 Principles of Internal Medicine, *supra* note 42, at 1429, 1429–30; Peter Casten, Jr., & Katherine Loftfield, *The Eyes and Vision, in* Environmental Medicine, *supra* note 19, at 240, 242. Exposure to chemical agents typically found in certain work environments can sometimes be inferred based on industrial hygiene studies of particular occupations. For example, employment as an asbestos insulator has been associated with significant levels of asbestos exposure.

Information about exposure may also be available from workplace industrial hygiene records or a police report. Other sources of information may include governmental agency or private consultant records and insurance inspections. However, physicians usually have to evaluate environmental or occupational diseases in the absence of quantitative exposure levels. Even in situations in which there are measurements of personal breathing-zone exposures, such data may not take into account various other factors, such as the level of a patient's exertion, which may change the actual dose to make it greater or lower than theoretical calculations; the performance of ventilation equipment; or the fit of a respirator.[60]

3. Other Risk Factors

In addition to information about environmental and occupational exposures, a patient's history should include information about other known risk factors, such as the patient's family history, smoking history, amount of exercise, alcohol use, use of medications or illicit drugs, and exposures to chemicals in the home or from hobbies.[61]

B. Past and Present Patient Records and Exposure-Related Records

Although time-consuming and bureaucratically cumbersome, an examination of patient records from former treating physicians, clinics, and hospitals can often be crucial for accurate diagnosis, for determination of the onset of an illness or symptom, and to provide information about external exposures. Patient records may reveal the course of an illness and the results of prior tests, and they can help gauge the reliability of patient-reported information. Unfortunately, because obtaining multiple patient medical records from various institutions in a timely manner is often difficult, much medical care is rendered in their absence. More complete records are often gathered once litigation has begun.

C. Physical Examination[62]

The physical examination is a routine procedure for evaluating the patient and determining a diagnosis. The physical examination identifies approximately 20%

60. For the effect of exercise, see, e.g., Joseph D. Brain et al., *The Effects of Exercise on Inhalation of Particles and Gases, in* Variations in Susceptibility to Inhaled Pollutants: Identification, Mechanisms, and Policy Implications 204, 210 (Joseph D. Brain et al. eds., 1988); for other variables affecting an individual's exposure and response to inhaled gases or particles, see, e.g., Speizer, *supra* note 59, at 1430.

61. *See* Bates et al., *supra* note 49, at 16–19; Speizer, *supra* note 59, at 1429–30.

62. Courts sometimes attach importance to the physician–witness's examination of the patient. *See, e.g., In re* Paoli R.R. Yard PCB Litig., 35 F.3d 717, 771 (3d Cir. 1994) (physician's testimony on causation admitted as to patients the witness examined), *cert. denied*, 513 U.S. 1190 (1995); *In re* "Agent Orange" Prod. Liab. Litig., 611 F. Supp. 1223, 1235, 1243–47 (E.D.N.Y. 1985), *aff'd*, 818 F.2d 187

of significant medical problems.[63] The physical exam has standard components with which physicians, depending on their degree of specialization, may be more or less proficient. For example, while most physicians will hear a loud heart murmur or identify a severe tremor, subtle signs of heart disease or neurological disease may be missed by those without specialty training in cardiology[64] or neurology, respectively. Greater proficiency can be expected from a specialist, because doctors in specialized fields focus their examinations on the system in question, do more tests within an area, are more skilled in performing the exam, and are better able to distinguish between significant and insignificant deviations from normal.

The findings from the physical exam as well as radiographic imaging studies, noninvasive functional tests, and blood tests are referred to as "signs" of illness, as contrasted with symptoms, which are subjectively reported by the patient. Although signs are more objective than symptoms, they still depend on the physician's skill and objectivity, degree of attention to detail, and level of concern. Physical signs assume enhanced significance when they demonstrate the presence of a functional or structural change already suggested by the patient history.[65]

A thorough physical exam begins with the taking of vital signs (temperature, heart rate, respiratory rate, and blood pressure). Next is a description of the patient's general appearance and whether the patient was able to cooperate with the exam. This is followed by examination of each region and organ system (skin, head, ears, eyes, nose, mouth and throat, neck, chest, lungs, heart and cardiovascular system, abdomen, genitourinary system, extremities and musculoskeletal system, and nervous system). Psychological assessments are sometimes then provided.[66] However, many specialists may perform only a portion of the exam; and, because of time constraints, many practitioners focus on only one aspect of a patient at a given time.[67]

Physicians are taught to record their findings on a physical exam in a routinized but not necessarily standardized fashion. A thorough exam will include

(2d Cir. 1987), *cert. denied*, 487 U.S. 1234 (1988). Courts have also recognized that physicians may present testimony based on examinations and tests performed by others, as well as on medical records. *See, e.g.*, Kannankeril v. Terminix Int'l, Inc., 128 F.3d 802, 809 (3d Cir. 1997); Sementilli v. Trinidad Corp., 155 F.3d 1130 (9th Cir.) (per curiam) (physician could present testimony on plaintiff's condition based on medical records and knowledge, experience, training, and education), *dissenting opinion amended*, 162 F.3d 1015 (9th Cir. 1998).

63. *See* Swartz, *supra* note 50, at 667.
64. *See, e.g.*, Feinstein, *supra* note 40, at 2.
65. *See* Fauci et al., *supra* note 56, at 2.
66. *See* Bates et al., *supra* note 49, at 118–21.
67. *Id.* at 117.

"findings" as opposed to merely notes indicating that an observation was "within normal limits" or "negative." However, the emphasis is on the accuracy of the observation, rather than the degree of detail that may be presented. How the findings of the physical exam fit into context with other data in the case is a key item in assessing the exam's reliability.[68]

As discussed above, specialists are generally better able than generalists to elicit patient history information, ascertain physical findings, and interpret lab results within their area of expertise. Findings that may have limited clinical meaning but may inform decisions regarding external causation in legal proceedings, such as the bilateral asymptomatic stable pleural thickening in someone with a history of asbestos exposure, are sometimes not mentioned by a treating physician, such as a radiologist. Thus, the absence of such findings from the treating physician's records should not necessarily be taken as an indication of disagreement between the treating physician and the specialist.

D. Diagnostic Tests

For diagnosis of more serious conditions, especially cancer, physicians are taught always to seek a tissue biopsy.[69] This is often referred to as a gold standard, because it is regarded as highly accurate or at least the most definitive indicator of a particular condition. For other conditions, the definitive test may be a radiological test (e.g., a pulmonary angiogram for diagnosis of pulmonary embolism)[70] or a microbiological test (e.g., a sputum culture for diagnosis of tuberculosis).[71]

Sometimes physicians and patients will be satisfied with a diagnosis even though the gold standard test for that diagnosis was not performed. There may be too much risk associated with such a test (e.g., if it is invasive or involves intentional exposure to a possible allergen), its costs may outweigh the benefit of achieving a more definitive diagnosis, or it may be superfluous because other data are so consistent and convincing.[72] As always, the various cost–benefit and risk–benefit equations are interpreted relative to the individual patient, physician, and medical circumstances, as well as institutional capabilities.

68. *Id.* at 649–52.

69. *See, e.g.*, Dan L. Longo, *Approach to the Patient with Cancer, in* 1 Principles of Internal Medicine, *supra* note 42, at 493, 494.

70. *See* Steven E. Weinberger & Jeffrey M. Drazen, *Diagnostic Procedures in Respiratory Disease, in* 2 Principles of Internal Medicine, *supra* note 42, at 1417, 1418.

71. *See* Matthew E. Levinson, *Pneumonia, Including Necrotizing Pulmonary Infections (Lung Abscess), in* 2 Principles of Internal Medicine, *supra* note 42, at 1437, 1440.

72. *See* Kassirer & Kopelman, *supra* note 48, at 217–22.

In modern medical practice, tests and procedures are critical to confirming most diagnoses. These include radiological examination, laboratory tests, physiological tests of lung or nerve function, pathological examination of tissue, and invasive diagnostic tests, such as cardiac catheterization. A physician's decision whether to order a diagnostic test for specified clinical indications should take into consideration expense, risk, accuracy, and predictive value. Tests are limited by their inherent sensitivity and specificity, the fallibility of the instrumentation, and the variation in skills of the individuals who perform or interpret the tests. Error rates for diagnostic tests, as discussed below,[73] in terms of sensitivity and specificity are generally available, but the all-important predictive values[74] vary with the particular disorder and with the population (i.e., demographics, background rate of disease) on whom the test is performed or the population from which a tested individual is derived. While pathological examination of tissue biopsies is considered the gold standard of diagnostic tests, even it has an error rate.[75]

In general, laboratory tests do not have a paramount role in establishing the external etiology of many chronic and acute illnesses. Major exceptions to this are microbiological evaluations for causes of infectious diseases, and cases of toxic substance intoxication, such as lead poisoning or alcohol or drug poisoning.[76]

Depending on the diagnosis being considered and whether the exposure truly leaves a reliable "signature" or "residue,"[77] a biopsy may or may not have great utility for exogenous causal diagnosis. Invasive tissue biopsies are not routinely performed for purposes of establishing causation because of the risk involved with the procedure to obtain the tissue. Sometimes such test results are incidentally available because they may have been used to establish the diagnosis, particularly in the case of lung disorders.

73. *See infra* note 105 and accompanying text.

74. *See infra* notes 107–108 and accompanying text.

75. *See* Fauci et al., *supra* note 56, at 3; Goldman, *supra* note 56, at 10; Kassirer & Kopelman, *supra* note 48, at 23.

76. *See* Christopher H. Linden & Frederick H. Lovejoy, Jr., *Poisoning and Drug Overdose, in* 2 Principles of Internal Medicine, *supra* note 42, at 2523, 2523–25.

77. Certain persistent toxic agents can sometimes be detected in laboratory tests. *See, e.g.*, Hose v. Chicago Northwestern Transp. Co., 70 F.3d 968 (8th Cir. 1995) (laboratory tests showed elevated manganese in plaintiff's body; MRI indicated manganese in brain). The interpretation of such tests has been at issue in a number of cases. *See, e.g., In re* Paoli R.R. Yard PCB Litig., 35 F.3d 717 (3d Cir. 1994) (dispute over whether PCB levels in plaintiffs' adipose tissue exceeded background levels), *cert. denied*, 513 U.S. 1190 (1995); Wright v. Willamette Indus., Inc., 91 F.3d 1105 (8th Cir. 1996) (presence of wood dust fibers at plaintiffs' residence and in tissue samples insufficient to establish exposure to formaldehyde at levels known to cause plaintiffs' symptoms).

1. Laboratory Tests

Laboratory tests are usually tests in which a specimen, usually blood or another body fluid, is submitted to a laboratory for a chemical or microbiological analysis. For many of the routine chemical assays for levels of proteins, fats, electrolytes, enzymes, or hormones in blood, there are established normal ranges for a given laboratory or test manufacturer, and for given subpopulations (e.g., men or women, children or adults). The results are interpreted as being either within or outside of normal limits. Not all deviation from normal limits is pathological, particularly if the individual is otherwise without complaint. For example, the results of liver function tests often fluctuate outside of the normal range in those without liver disease or hepatotoxin exposure. Based on standard statistical techniques for defining normal ranges, one in twenty test results can be expected to be abnormal (i.e., outside the normal range) in a healthy individual.[78]

Common laboratory tests include x-rays, routine blood chemistries, and blood counts. More specialized tests include computerized axial tomography (CAT) scans, magnetic resonance imaging (MRIs), and angiograms.[79] All of these tests are used in one of three ways as part of the diagnostic process. The first and most common use is to clarify a disease process or pathology or pathophysiology.[80] A second and less common use of laboratory tests is for estimation of exposure to potentially toxic substances. These tests include measures of an agent in the body (e.g., blood lead levels) or in an excretory product (e.g., urine mercury). Understanding that such tests only determine exposure and not disease or health effect is critical.[81] A third and fairly uncommon type of laboratory test is used to substantiate an exposure–effect relationship.[82] Many, if not most, such tests are actually tests of allergic sensitization (e.g., to a metal or other potential cause of allergic asthma). The expert should be clear about what type of information is being inferred from a given test and about the basis in the literature for using the test for that purpose.[83]

78. *See* Cullen et al., *supra* note 19, at 223–24. For an overview of available blood tests, fluid analysis studies, and urinalyses, see, e.g., Kathleen Deska Pagana & Timothy James Pagana, Mosby's Manual of Diagnostic and Laboratory Tests 7–9, 557, 859–73 (1998).

79. *See* Fauci et al., *supra* note 56, at 3; for uses of laboratory tests in environmental disease, see Cullen et al., *supra* note 19, at 222–23 and Arthur Frank, *The Environmental History, in* Environmental Medicine, *supra* note 19, at 232. *See also In re* Paoli R.R. Yard PCB Litig., 916 F.2d 829 (3d Cir. 1990), *cert. denied*, 499 U.S. 961 (1991).

80. For a case involving the use of laboratory tests in diagnosis, see *Cella v. United States*, 998 F.2d 418 (7th Cir. 1993).

81. *See, e.g.*, Linden & Lovejoy, *supra* note 76, at 2523.

82. *See* Cullen et al., *supra* note 19, at 223.

83. *See id.* at 228. For an example of laboratory tests used to rule out alternative diagnoses and causes, see *Hose v. Chicago Northwestern Transportation Co.*, 70 F.3d 968, 973, 975 (8th Cir. 1995) (supporting a diagnosis of manganese encephalopathy, medical witnesses cited a positron emission tomography (PET) scan to rule out alcoholism, stroke, and Alzheimer's disease, and an MRI to exclude copper, calcium, and other harmful exposures).

Physicians are taught to think about clinical testing in terms of the clinical significance (particularly, predictive value) of a given test in a given situation. Small or inconsistent changes in values do not necessarily indicate a clinically important effect and should be confirmed by repeat testing before being otherwise investigated. On the other hand, important effects may not drive an individual's values outside of the population reference range. For instance, someone previously at the upper limit of the normal range exposed to a chlorine leak might suffer a reduction in rate of airflow. Although the subsequent rate was within the normal range, it would not be normal in this individual.[84] Unfortunately, baseline data on an individual prior to exposure are usually not available. Thus, making inferences from other diagnostic and exposure information may be useful in understanding the impact of exposure on that individual.

2. Pathology Tests

Pathology tests are conducted by taking a sample of body tissue (obtained during surgery or a biopsy) and submitting it for microscopic evaluation by a specialist physician (pathologist). The pathologist makes a determination as to whether the tissue appears normal for the organ from which it was taken. If it does not appear normal, then a determination of the pattern of abnormality, such as inflammation, malignancy, or scarring, is sought.[85]

Sometimes the etiology of the abnormality is apparent, as when special stains are used for determination of the presence of microorganisms that can cause a given infection. On the other hand, most cancers, whether of lung or breast or bone marrow, have no features that allow the histopathologist to discern a toxic, viral, or hereditary etiology. Clues from molecular biology analysis have been experimentally reported, but are not yet available clinically.[86]

Pathology, typically felt to be the gold standard, often is found wanting when external etiology needs to be determined. Some conditions, such as neuropsychiatric diseases that may be related to metal or solvent exposure, do not have established pathological abnormalities.[87]

3. Clinical Tests

Clinical tests are physiological determinations of organ function. Common examples are pulmonary (lung) function tests, which have well-established normal

84. Cullen et al., *supra* note 19, at 223.

85. For specific examples, see Ivan Damjanov, Histopathology: A Color Atlas and Textbook 23–24, 36, 58, 64 (1996).

86. *See* Bernard D. Goldstein & Mary Sue Henifin, Reference Guide on Toxicology § IV, in this manual.

87. *See* Howard Hu, *Heavy Metal Poisoning, in* 2 Principles of Internal Medicine, *supra* note 42, at 2564, 2565–66.

ranges, but are quite dependent on patient effort; nerve and muscle function tests, which are largely effort-independent and have reasonably well-established reference ranges, but are sensitive to interlaboratory variation, and electrocardiograms (EKGs), which are interpreted with a combination of objective measures and more subjective recognition of patterns resulting from inidividual expertise.[88]

All tests have strengths and limitations for their use in reaching a certain diagnosis or making a causal inference. The expert should be able to address strengths and weaknesses of various approaches based on the situation at hand. Why was one test chosen or preferable to another? If available, what is the sensitivity, specificity, and validity for the test in general, and what are its predictive values in the population (characterized by age group, gender, comorbid diseases, workplace exposures) from which the individual comes?[89]

Mostly these predictive values will be available in the medical literature, but there are many disappointing gaps. Given inevitable inconsistencies in the patient's data, a qualified expert will usually be able to interpret and explain these inconsistencies in a satisfactory manner.

IV. Physician Decision Making

A. Introduction

For the treating physician, "[c]linical reasoning is the essential function of the physician; optimal patient care depends on keen diagnostic acumen and thoughtful analysis of the trade-offs between the benefits and risks of tests and treatments."[90] Beyond assessing the presence or absence of disease, and defining appropriate treatment or prevention, the physician must be able to skillfully communicate information to the patient and other interested parties.[91]

Moreover, a physician may be asked to determine the causation of disease, in order, for example, to offer a patient advice on continuing activities that may cause, contribute to, or exacerbate or ameliorate the disease. The physician may also be asked to determine causality as an expert in a legal proceeding.[92] In undertaking all of these activities, the physician is grounded in the art and science of clinical reasoning, which we describe below in general terms.

88. For specific tests of pulmonary, nerve and muscle function, and electrocardiography, respectively, see Pagana & Pagana, *supra* note 78, at 1016–21, 490–92, 486–89, 478–82.

89. *See infra* § IV and accompanying footnotes.

90. Kassirer & Kopelman, *supra* note 48, at 2.

91. *See* Cullen et al., *supra* note 19, at 217.

92. *See* Hu & Speizer, *supra* note 42, at 19, 20.

The physician is trained to recognize diseases as coherent deviations from normal structure or function that affect a certain part of the body or type of tissue. Physicians recognize the characteristic symptoms, signs, and laboratory manifestations of given diseases, although a relatively small number of discrete symptoms and signs are shared by a much larger number of coherent diseases. In fact, diseases result from one or a combination of only ten or so general pathophysiological processes (congenital, infectious, neoplastic, toxic, genetic, vascular, immunologic, inflammatory, endocrine, and traumatic). The goal of the physician is to distinguish which specific type of disorder (disease) is causing a patient's symptoms and signs.[93]

One of the difficulties in recognizing diseases is the absence of an accepted metric for establishing new disease entities. Thus, when a possible new set of characteristic symptoms, signs, and laboratory manifestations is described, there is no one method for developing consensus on whether a new disease entity exists.[94] For example, when the characteristic symptoms, signs, and laboratory test results of acquired immunodeficiency syndrome (AIDS) were first described in the early 1980s, prior to the identification of the human immunodeficiency virus (HIV), there was considerable controversy over whether a new disease entity had manifested itself. Development of a test for infection with the specific virus cemented recognition of the disease. There have also been analogous, but largely unresolved, controversies over chronic fatigue syndrome, fibromyalgia, multiple-chemical sensitivity, and Gulf War syndrome.[95]

93. For an example of how a symptom may be common to a number of diseases, compare Jeffrey A. Gelfand & Charles A. Dinarello, *Fever and Hyperthermia, in* 1 Principles of Internal Medicine, *supra* note 42, at 84, 88 tbl.17-1; Elaine T. Kaye & Kenneth M. Kaye, *Fever and Rash, in* 1 Principles of Internal Medicine, *supra* note 42, at 90, 91–96 tbl.18-1; Robert B. Daroff & Joseph B. Martin, *Faintness, Syncope, Dizziness, and Vertigo, in* 1 Principles of Internal Medicine, *supra* note 42, at 100, 100 tbl.20-1; Patrick T. O'Gara & Eugene Braunwald, *Approach to the Patient with a Heart Murmur, in* 1 Principles of Internal Medicine, *supra* note 42, at 198, 199 tbl. 34-1.

94. *See, e.g.*, Khalida Ismail et al., *Is There a Gulf War Syndrome?*, 353 Lancet 179, 179 (1999) ("For an illness to be recognised as a new disorder it must be sufficiently different from other recognised disorders . . . . There is no formal process to investigate whether a set of symptoms are unique to a new illness."). For an explication of several methods that can be used to determine whether a new disease entity exists, see also David H. Wegman et al., *Invited Commentary: How Would We Know a Gulf War Syndrome If We Saw One?*, 146 Am. J. Epidemiology 704 (1997).

95. The recognition of multiple-chemical sensitivity as a disease was at issue in *Zwillinger v. Garfield Slope Housing Corp.*, No. CV 94-4009, 1998 WL 623589 (E.D.N.Y. Aug. 17, 1998). *See also* Howard M. Kipen & Nancy Fiedler, *Invited Commentary: Sensitivities to Chemicals—Context and Implications*, 150 Am. J. Epidemiology 13 (1999).

B. Diagnosis

Clinical diagnosis has been described as a process of "iterative hypothesis testing." It relies on both analysis and synthesis of data. When making a diagnosis, a clinician makes inferences about types of malfunctions of the patient's organs or chemistry that would lead to the observed abnormalities. The basis for the inferences are facts (information) that have been collected about the patient. The clinician applies inferential (also known as inductive) reasoning, considering the specific historical, physical, and laboratory facts, until a diagnosis that coherently describes the patient's condition can be hypothesized. Such a working diagnosis is sometimes called, or corresponds to, a syndrome, which is a clustering of signs and symptoms of abnormal function.[96] Syndromes and working diagnoses do not identify precise underlying internal causes. To arrive at an underlying internal cause, the physician must process the multiple symptoms and signs from the working diagnosis into a single diagnosis or disease, such as multiple vascular strokes as an explanation for dementia.

In the process of performing a differential diagnosis, the physician determines which of two or more diseases with similar clinical findings is the one that the patient is suffering from.[97] The physician does this by developing a list of all of the possible diseases that could produce the observed signs and symptoms, and then comparing the expected clinical findings for each with those exhibited by the patient.[98]

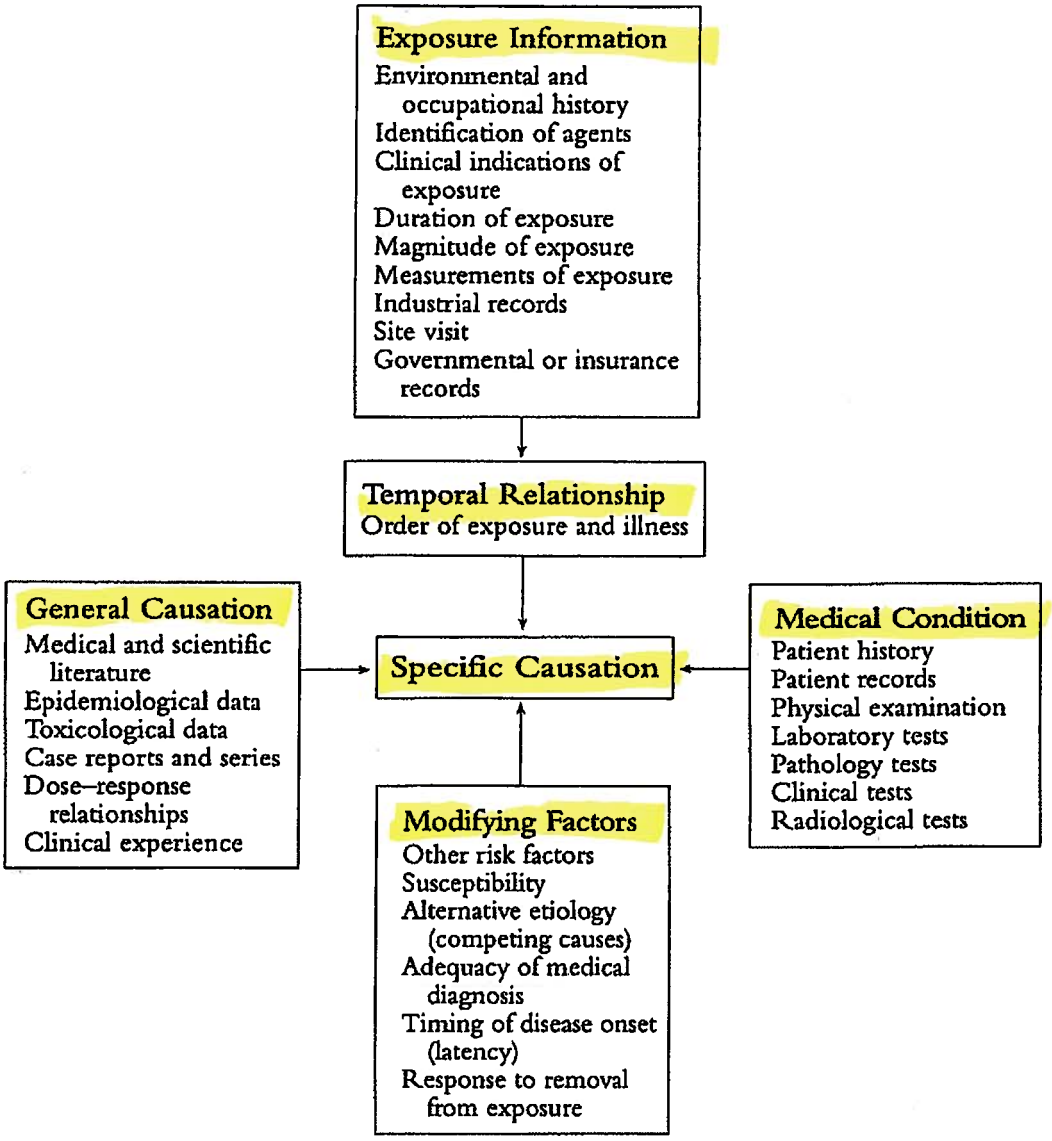
While working through a differential diagnosis, the clinician will often have generated a number of diagnostic hypotheses of what specific underlying diseases might be the cause of the patient's problem. Initially these hypotheses are colored by the patient's demographic characteristics (e.g., age, gender, race) as well as appearance and chief (or presenting) complaints, because all of these

96. For example, dementia is a syndrome of impaired memory, thinking, language, and judgment (all of which are symptoms that can actually also be measured as signs) related to destruction or malfunction of specific parts of the brain. In congestive heart failure, shortness of breath (symptom), trouble lying down flat (symptom), swollen ankles (symptom or sign), weight gain (sign), swollen neck veins (sign), crackling noises heard in the lungs (sign), and galloping heart sounds (sign) are attributable to one pathophysiological dysfunction—inadequate pumping of blood by the heart. In Cushing's syndrome, an abnormally round face (moon face), diabetes mellitus (high blood sugar causing a syndrome of its own), bone thinning (osteoporosis), and high blood pressure are all due to excessive amounts of certain hormones, glucocorticoids, resulting from either excess glandular secretion by the body or overuse as a medication. Fauci et al., *supra* note 56, at 3.

97. *See* Stedman's Medical Dictionary 474 (26th ed. 1995) (definition of *differential diagnosis*); Kassirer & Kopelman, *supra* note 48, at 16.

98. Diagnosis is at issue in many kinds of cases, including medical malpractice and other personal injury claims. *See, e.g.*, Bates et al., *supra* note 49, at 635–48; Samuels v. Secretary of Dep't of Health & Human Servs., No. 91-127V, 1995 WL 809884 (Fed. Cl. Aug. 1, 1995) (diagnosis of a neurological disorder at issue in claim under the National Vaccine Injury Compensation Program); Alex v. Dr. X, 692 So. 2d 499 (La. Ct. App. 1997) (diagnosis of tuberculosis at issue).

Figure 1. Determining External Causation



must establish the characteristics of the medical condition. Second, he or she carefully defines the nature and amount of the environmental exposure. The third step is to demonstrate that the medical and scientific literature provides evidence that in some circumstances the exposure under consideration can cause the outcome under consideration. This step is synonymous with establishment of general causation. As part of this step, the clinician attempts to establish the relationship between dose and response, including whether thresholds exist, ultimately defining the clinical toxicology of the exposure. The fourth step is to

explanation of all of the patient's signs and symptoms with a single underlying condition or disease process is desirable. Of course, some patients, especially the elderly, may have more than one underlying disease (e.g., heart disease, osteoporosis, and chronic renal failure). Sometimes two common conditions will be a more logical explanation than one complex and unusual disease that could also explain all of the observed manifestations. Physicians also consider competing hypotheses, to ascertain that no other disease is present that better explains the current hypothesis or findings.[103]

All diagnostic hypotheses represent probabilistic judgments that are based on observed medical facts that have variable probabilities of being correct. Each fact (symptom, sign, or test abnormality) also has only a variable probability of being found in a given condition that is typically characterized by its presence. If the diagnosis is based on inconsistent records or observations, the physician should explain how the inconsistencies affected the assessment being offered.[104]

C. Probabilistic Basis of Diagnosis

Medical diagnosis is not an exact science. As indicated above, physicians make probabilistic judgments on a day-to-day basis, even when they can supplement a patient's history and physical with the results of extensive laboratory tests. Laboratory, clinical, and physiological tests are important for any given disease and may be characterized in terms of their "sensitivity" and "specificity," which indicate the usefulness of the test results in making a particular disease diagnosis. For a given test, sensitivity, which is also known as the true positive rate, is the percentage of positive tests in patients who actually have the disease. Test results in those who have a disease but are incorrectly identified as not having the disease because of the test's insensitivity are "false negatives." Thus, a test that is positive in 80% of actual cases of asthma (80% sensitivity) will fail to indicate asthma, or be falsely negative, in 20% of actual cases.

Specificity is the percentage of negative test results in individuals who are free of a given disease, also known as the true negative rate. Test results in those who are free of the disease who are incorrectly identified as having the condition are "false positives." Thus, a test that indicates abnormal bronchial reactivity in 15% of individuals without asthma would have a false positive rate of 15%; their test results were positive, but they are free of the condition.[105] For example, a physician may order a chest x-ray as a test to rule out lung cancer for a 60-year-old man who just began to cough up flecks of blood but has a normal physical exam.

103. *See id.*

104. *See id.* at 16; Bates et al., *supra* note 49, at 635–74.

105. *See* Bates et al., *supra* note 49, at 641; Goldman, *supra* note 56, at 10–11; Kassirer & Kopelman, *supra* note 48, at 18–19; Michael D. Green et al., Reference Guide on Epidemiology § V.H, and David H. Kaye & David A. Freedman, Reference Guide on Statistics §§ III.A.3, IV.B.2, IV.C, in this manual.

If the x-ray does not show any evidence of lung cancer (is negative for a finding consistent with lung cancer), that diminishes the probability of lung cancer, but it does not rule it out. A cancer may actually be present but not show up on the x-ray because it is too small or because it is in an unobservable location. The physician will be aware of the possibility of such a false-negative result and, especially for a high-risk individual (see below), may order a follow-up exam in a few months or immediately order a more sensitive test, such as a CAT scan or bronchoscopy. A false-positive result that was due to the imperfect specificity of the chest x-ray would occur if the x-ray showed an abnormality that suggested cancer, but when biopsied (the gold standard of tissue diagnosis) turned out to be an old scar resulting from a dormant injection.

Sensitivity and specificity provide information about the usefulness of a piece of data (a symptom, sign, or test) for diagnostic reasoning in any population of patients. However, they do not give complete information for predicting or excluding disease in individual patients. For that, information about the patient, and the population that he or she represents, must be incorporated.[106]

Physicians must interpret the predictive value of a test in assessing the presence or absence of disease in a specific patient. The predictive value of a test for a specific individual is based not only on the sensitivity and specificity of the test, but also on the prevalence of disease in the population from which the patient comes, such as age group, gender group, racial group, and groups with occupational exposures.[107] In the previous example, if the 60-year-old man was a smoker and had been occupationally exposed to a lung carcinogen, such as asbestos, a negative x-ray might be viewed more suspiciously than if he was free of additional risks.

If sensitivity and specificity are known in general for a particular test, sign, or symptom, and the overall prevalence of the condition is known for the population group from which the patient comes, then one can actually calculate a good approximation of the predictive value of the test, sign, or symptom for that person and condition according to a rule known as Bayes' theorem. These calculations have actually been translated into nomograms (tables) for general use.[108] Few clinicians actually calculate such probabilities, but they use an analogous reasoning process on a routine basis. This Bayesian reasoning is a major tool of

106. *See* Bates et al., *supra* note 49, at 645–46.

107. "Positive predictive value" is the frequency of disease among patients with positive results, and "negative predictive value" is the frequency of absence of disease among individuals with negative test results. For a test with a given sensitivity and specificity, positive predictive value is higher when a condition is common in a population, and negative predictive value is higher when the condition is rare. Bates et al., *supra* note 49, at 642. *See also* David H. Kaye & David A. Freedman, Reference Guide on Statistics §§ III.A.3, IV.C, in this manual.

108. *See* Swartz, *supra* note 50, at 675–76 & fig.25-3. *See generally* David H. Kaye & David A. Freedman, Reference Guide on Statistics § IV.D, app., in this manual.

physicians in thinking through a differential diagnosis. For instance, heart attacks are very rare in 25-year-olds and relatively more common in 75-year-olds. In analyzing a patient with chest pain and borderline abnormal EKG changes, the physician is much more likely to suspect a heart attack as the cause of the pain in the 75-year-old, and admit the patient to a hospital, at least for monitoring.[109]

Diagnostic reasoning is usually more complex than the examples given because it is simultaneously based on multiple symptoms, signs, and test results (e.g., family history, physical exam). These findings are not all truly independent of one another, thus preventing straightforward addition of the probabilities as in a Bayesian model. This lack of independence limits the ability of physicians to make accurate calculations of the results of multiple simultaneous predictive values. However, physicians must routinely make such estimations, albeit often implicitly and without numerical quantification, as part of clinical care. Thus, physicians frequently rely on the principles of Bayesian reasoning when deciding on a diagnosis.[110] Doctors combine probabilities of disease (prevalence) with their knowledge of the frequency of signs and symptoms in a given disease and competing diseases to progressively modify and ultimately arrive at their view of the likelihood of the disease under consideration.

D. Causal Reasoning

During the diagnostic process, the physician employs causal reasoning to integrate the various clinical variables into an understanding of the cause-and-effect relationships among them, based on an understanding of how the various systems of the human body interact and react to external stressors. Causal reasoning allows the clinician to conceptualize the possible course of the patient's disease and predict the effects of treatment, and is important in evaluating the coherency of a diagnosis. For example, if the patient is experiencing chest pain on exertion and has a history of high blood cholesterol levels, the physician might posit a causal model that involves cholesterol plaque substantially obstructing coronary arteries, resulting in inadequate blood flow to the heart muscle during exercise causing chest pain. This model might then suggest that the physician first investigate the degree of occlusion in the coronary arteries, and second

109. The positive predictive value of a symptom of chest pain for a heart attack is very low in a 25-year-old because advanced atherosclerotic cardiovascular disease is rare in this age group and other causes of chest pain are more common. Similarly, interstitial fibrosis on a chest x-ray, whatever the x-ray's sensitivity and specificity for a true underlying finding of pathologic fibrosis, has a much higher predictive value for a diagnosis of asbestosis in a person known to come from an asbestos-exposed population than in someone with no known occupational exposure to asbestos.

110. *See* Kassirer & Kopelman, *supra* note 48, at 19–24; Steven N. Goodman, *Toward Evidence-Based Medical Statistics. 2: The Bayes Factor*, 130 Annals Internal Med. 1005, 1011 (1999).

consider measures such as smoking cessation, dietary modification, medications, and even angioplasty or surgery if the level of occlusion proves to be substantial and a likely explanation for the pain.

As the process of refinement of diagnostic hypotheses unfolds, the consideration of several causal models may be necessary, because consistency of the model with observed findings does not necessarily prove that a model is correct. In the example above, another model that would explain the findings is exposure to high levels of carbon monoxide from a faulty furnace at home, producing a blood carboxyhemoglobin level of 18% (the normal for a nonsmoker is less than 1%) and reducing the blood's oxygen-carrying capacity. In conjunction with only mild coronary artery obstruction by plaque, this exposure then leads to inadequate oxygen delivery to the heart muscle and chest pain. The model combines general causation models for coronary artery disease with information on the levels of carbon monoxide and coronary artery obstruction specific to this patient. Thus, the physician applies general medical knowledge about the relationship of various factors to symptoms and then refines the appropriate causal model in accordance with the specific patient's condition. Although carbon monoxide intoxication can cause chest pain that is due to inadequate oxygen delivery to the heart, it requires a blood carboxyhemoglobin level of at least 5% to 10%, and its impact is enhanced by the presence of underlying mechanical obstruction of the coronary arteries. Hence, the physician must usually consider and assess alternative and more specific causal models before accepting a particular model as the preferred explanation. Like the probabilistic reasoning described above, this kind of reasoning is rarely made explicit.

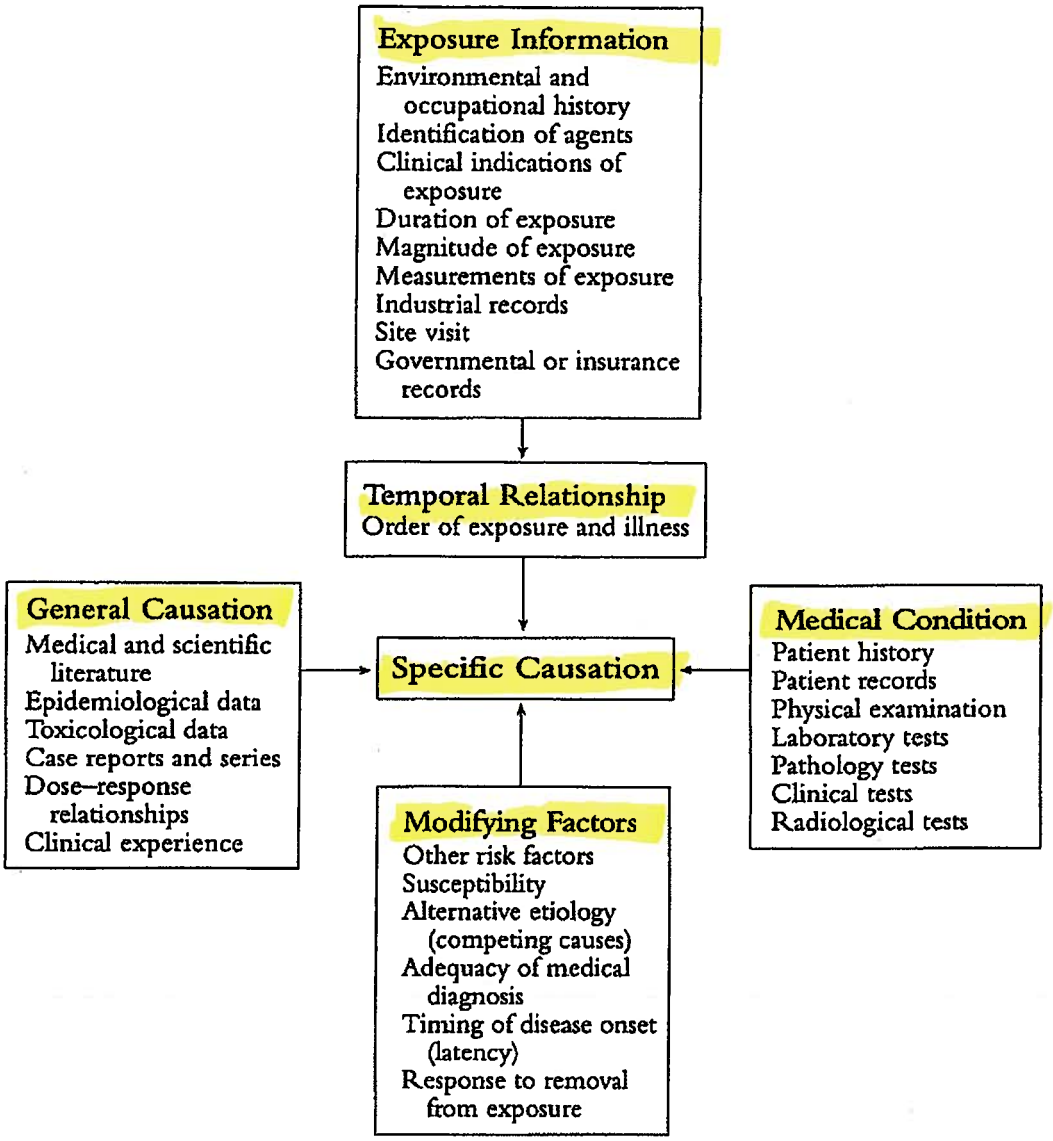
E. Evaluation of External Causation

For the physician, both causal and probabilistic reasoning are the basis for establishing external causation, which is the relationship between environmental factors (work, chemical exposures, lifestyle, medications) and illness, as well as for making the more common analysis of internal causation as discussed earlier in section IV.B. The physician may be asked to determine external causation by the patient or a third party, such as a lawyer, insurance company, or governmental agency. A key element of determining causation is gaining access to all information available about the patient's condition.

Figure 1 provides examples of the diverse types of information that may be available for review in determining external causation. In any given case, much of the listed information is normally not available.[111] Determining external causation also generally occurs in a stepwise fashion. In the first step the physician

111. For a somewhat different illustration of the interaction of such factors, see Cullen et al., *supra* note 19, at 230 fig.18-2.

Figure 1. Determining External Causation



must establish the characteristics of the medical condition. Second, he or she carefully defines the nature and amount of the environmental exposure. The third step is to demonstrate that the medical and scientific literature provides evidence that in some circumstances the exposure under consideration can cause the outcome under consideration. This step is synonymous with establishment of general causation. As part of this step, the clinician attempts to establish the relationship between dose and response, including whether thresholds exist, ultimately defining the clinical toxicology of the exposure. The fourth step is to

apply this general knowledge to the specific circumstances of the case at hand, incorporating the specifics of exposure, mitigating or exacerbating influences, individual susceptibilities, competing or synergistic causes, and any other relevant data.[112]

Many conditions resulting from toxic exposures are similar or identical in clinical manifestations to conditions arising from nontoxic causes.[113] Physicians rely on their training and expertise as clinicians and scientists when considering the medical and scientific literature as well as information about a patient's condition to best determine causality in a particular patient. Definitive tests for causality are actually rare,[114] and physicians must almost always use an element of judgment in determining the relationship between exposure and disease in a

112. Many cases involving issues of external causation have involved witnesses who testify to having arrived at an opinion on cause through a process of ruling out or eliminating other causes, a process frequently referred to by the courts and witnesses as "differential diagnosis" or "differential etiology" (for explanation of the differences between medical and legal uses of terminology, see section I.B., *supra*). Not infrequently, this form of testimony is implicitly or explicitly offered to satisfy the applicable burden of proof on causation. The relationship between the "more probable than not burden of proof" and "differential diagnosis" was discussed in *Cavallo v. Star Enterprise*, 892 F. Supp. 756 (E.D. Va. 1995), *aff'd in part, rev'd in part*, 100 F.3d 1150 (4th Cir. 1996), *cert. denied*, 522 U.S. 1044 (1998), a case in which the witness opined on whether a spill of aircraft fuel caused the plaintiff's rash. The court explained, "The process of differential diagnosis is undoubtedly important to the question of 'specific causation.' If other possible causes of an injury cannot be ruled out, or at least the probability of their contribution to causation minimized, then the 'more likely than not' threshold for proving causation may not be met." *Id.* at 771 (footnote omitted).

Courts differ on whether opinion based on such "differential diagnosis" or "differential etiology" of cause is admissible. *Compare* Westberry v. Gummi, 178 F.3d 257, 263 (4th Cir. 1999) (reliable "differential diagnosis" provides a valid basis for an expert opinion), Anderson v. Quality Stores, Inc., 181 F.3d 86 (4th Cir. 1999) (per curiam) (opinion on spray paint causing pulmonary problems should have been admitted based on "differential diagnosis" and temporal relationship), *In re* Paoli R.R. Yard PCB Litig., 35 F.3d 717 (3d Cir. 1994) (approving opinion based on "differential diagnosis"), *cert. denied*, 513 U.S. 1190 (1995), McCullock v. H.B. Fuller Co., 61 F.3d 1038, 1042–44 (2d Cir. 1995) (accepting opinion based on "differential etiology"), *and* Zuchowicz v. United States, 140 F.3d 381, 387–91 (2d Cir. 1998) (accepting witness's "differential etiology" opinion of causes of pulmonary hypertension), *with* Raynor v. Merrell Pharms., Inc., 104 F.3d 1371, 1375–76 (D.C. Cir. 1997) ("differential diagnosis" of cause of birth defect inadmissible where general causation proof absent), Cavallo v. Star Enter., 892 F. Supp. 756, 771–73 (E.D. Va. 1995) ("differential diagnosis" of cause inadmissible where general causation not established), *aff'd in part, rev'd in part*, 100 F.3d 1150 (4th Cir. 1996), *cert. denied*, 522 U.S. 1044 (1998), Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1412–14 (D. Or. 1996) ("differential diagnosis" and specific causation require proof of general causation; witness did not explain how he ruled out other causes), Haggerty v. Upjohn Co., 950 F. Supp. 1160, 1166–67 (S.D. Fla. 1996) ("differential diagnosis" testimony inadmissible where another cause could explain all of plaintiff's symptoms), *aff'd*, 158 F.3d 588 (11th Cir. 1998) (unpublished table decision), *and* Austin v. Children's Hosp. Med. Ctr., 92 F.3d 1185 (6th Cir. 1996) (unpublished table decision) (text at No. 95-3880, 1996 WL 422484, at *3 (6th Cir. July 26, 1996)) (expert unable to show that defendant, rather than other sources, "more likely than not" infected plaintiff's son with fatal virus).

113. *See, e.g.*, Herbert Y. Reynolds, *Interstitial Lung Disease*, *in* 2 Principles of Internal Medicine, *supra* note 42, at 1460, 1460–63 & tbl.259-1.

114. For a discussion of the difficulty of establishing causation, see Feinstein, *supra* note 40, at 266–74.

given patient. For instance, if a substance is suspected to cause an allergic or toxic condition, it may be necessary for diagnostic purposes to remove a patient from the workplace on a trial basis. On the other hand, determinations of external causation in patients with cancer may be irrelevant to treatment decisions as treatment is usually unaffected by assignment of cause.[115]

Physicians use both causal and probabilistic reasoning in determining both internal and external causation in regard to a particular illness. Methods for determination of some special external causes of disease may be found in occupational and environmental medical texts and journals[116] and generally are analogous to methods used for assessment of internal disease causation.[117] The difference is essentially in the body of medical, toxicological, epidemiological, and industrial hygiene knowledge that is relevant and needs to be incorporated.

For instance, in an elderly patient with chronic shortness of breath, the treating physician may use differential diagnosis to determine that chronic bronchitis is the best explanation as the underlying cause of symptoms, having excluded heart disease, anemia, lung fibrosis, and emphysema. The treating physician will rarely consider the external causes of the chronic bronchitis, beyond consideration of whether the patient smoked cigarettes.[118] The specific contribution of environmental or workplace exposures is rarely assessed as a part of clinical care in an elderly nonworking patient, since it does not affect diagnosis, treatment, and prognosis of this particular disease.[119] However, such determination of external causation may be essential to determination of a contested workers' compensation award.[120]

The key factor for the courts to recognize is that, while similar underlying reasoning is used in determination of both internal and external causation, and

115. However, exceptions may be cited, including the need to determine if there is a genetic (familial) risk of cancer that may require notification and screening of family members (e.g., certain forms of colon cancer and breast cancer), or if other family members or workers may be at remediable risk.

116. *See, e.g.*, Howard Hu & Frank E. Speizer, *Specific Environmental and Occupational Hazards, in* 2 Principles of Internal Medicine, *supra* note 42, at 2521, 2521–22; Linden & Lovejoy, *supra* note 76, at 2523–25; Hu, *supra* note 87, at 2565–67.

117. *See, e.g.*, peer review case studies published by the Agency for Toxic Substances and Disease Registry (ATSDR), a branch of the Centers for Disease Control and Prevention. For the most part, these case studies discuss the diagnosis and treatment of environmental illness, and in a number of instances discuss the reasoning involved in assessing the causal role of an environmental exposure. Selected ATSDR case studies are included in Environmental Medicine: Integrating a Missing Element into Medical Education, *supra* note 57, at app. C.

118. *See* Eric G. Honig & Ronald H. Ingram, Jr., *Chronic Bronchitis, Emphysema, and Airways Obstruction, in* 2 Principles of Internal Medicine, *supra* note 42, at 1451, 1452.

119. In a working patient, the contribution of workplace conditions may be taken into account in advising the patient on the advisability of returning to or remaining in the work environment if there are conditions present that may exacerbate the patient's respiratory condition. *Id.* at 1456.

120. *See, e.g.*, Fiore v. Consolidated Freightways, 659 A.2d 436 (N.J. 1995).

physicians routinely make limited determinations of external causation, many of the facts relevant to a determination of external causation rely on a body of scientific literature that is not routinely used by treating physicians. As a corollary, an expert's opinion on diagnosis and his or her opinion on external causation should generally be assessed separately, since the bases for such opinions are often quite different.

1. Exposure

Critical to a determination of causation is characterizing exposure. Exposure to a toxic substance can sometimes be established by a review of the patient's history and various available indicators of exposure, as discussed in section III. There are four "cardinal" pieces of exposure information:

1. The material or agent in the environmental exposure should be identified.
2. The magnitude or concentration of an exposure should be estimated, including use of clinical inference.
3. The temporal aspects of the exposure should be determined—whether the exposure was short-term and lasted a few minutes, days, weeks, or months, or was long-term and lasted for years. Similarly, the latency between exposure and disease onset is often critical.
4. If possible, the impact on disease or symptoms should be defined.[121]

In many instances, the desired information will be incomplete,[122] but it can often be inferred from the literature that a given amount of time in a particular industry is well associated with disease-producing potential. Progressive pulmonary fibrosis (accelerated silicosis) can develop in as little as ten months in workers involved in manufacturing abrasive soaps, tunneling in rock that has a high quartz content, or carrying out sandblasting in small, enclosed spaces, although

121. *See* Cullen et al., *supra* note 19, at 224.

122. The courts vary in the degree of certainty they require in exposure estimates. Many courts accept exposure evidence as sufficient without proof of specific levels. *See, e.g.*, Kannankeril v. Terminix Int'l, Inc., 128 F.3d 802, 808–09 (3d Cir. 1997). Other courts have required more particularized proof. *See, e.g.*, Curtis v. M&S Petroleum, Inc., 174 F.3d 661, 671–72 (5th Cir. 1999) (exposure evidence sufficient for opinion on causation where expert testified that refinery workers were exposed to at least 100 parts per million (ppm), and probably several hundred ppm, of benzene). Based on these measurements, *Curtis* distinguishes another Fifth Circuit case, *Moore v. Ashland Chemical, Inc.*, 151 F.3d 269 (5th Cir. 1998) (en banc), *cert. denied*, 119 S. Ct. 1454 (1999), in which exposure evidence was found insufficient to support an opinion on causation because the expert had a "'paucity of facts'" on which to base an opinion and did not testify to any specific levels of exposure. 174 F.3d at 670 (quoting *Moore*, 151 F.3d at 279 n.10). Exposure levels have been at issue in a number of other cases. *See, e.g., In re* Paoli R.R. Yard PCB Litig., 916 F.2d 829 (3d Cir. 1990), *cert. denied*, 499 U.S. 961 (1991); *In re* "Agent Orange" Prod. Liab. Litig., 611 F. Supp. 1223 (E.D.N.Y. 1985), *aff'd*, 818 F.2d 187 (2d Cir. 1987), *cert. denied*, 487 U.S. 1234 (1988).

simple silicosis is much more commonly a chronic illness resulting from years of exposure.[123] In other situations, exposure estimates will be based on methods beyond the scope of medical expertise, such as physical or chemical analyses, or chemical fate-and-transport modeling (i.e., using mathematical models to project the movement of chemicals in air, water, and soil).

In determining causation, the physician may have particular insight into clinical clues related to exposure, such as clinical indicators of degree of exposure, temporal relationships, and the effect of removal from the toxic substance.[124] The physician also has particular insight into the role that preexisting illnesses may play in causing an exacerbation, recurrence, or complication of a clinical condition independent of any exposure to toxic products, or in concert with a toxic exposure.[125]

2. Reviewing the Medical and Scientific Literature

After characterizing exposure and the nature of the patient's disease, the physician expert witness must determine if the medical and research literature supports a determination of environmental causation.[126] The research literature in-

123. *See* Speizer, *supra* note 59, at 1431–32.

124. An appropriate temporal relationship—the time that elapsed between exposure and onset of disease or symptoms—is a necessary but often insufficient basis for an opinion on causation. Courts frequently warn against reasoning based on the premise "*post hoc, ergo propter hoc.*" *See, e.g.*, Whiting v. Boston Edison Co., 891 F. Supp. 12, 23 n.52 (D. Mass. 1995) (rejecting opinion on cause of acute lymphocytic leukemia following radiation exposure). In some cases, courts have permitted opinions on causation based primarily on temporal proximity between exposure and development of the disease, but many of these cases involved symptoms or diseases that closely followed the exposure asserted to be the cause. *See, e.g.*, Curtis v. M&S Petroleum, Inc., 174 F.3d 661, 670 (5th Cir. 1999); Anderson v. Quality Stores, Inc., 181 F.3d 86 (4th Cir. 1999) (unpublished table decision) (text at No. 98-2240, 1999 WL 387827, at *2 (4th Cir. June 14, 1999) (per curiam)). Other courts have excluded opinions on causation based primarily on temporal proximity. In *Moore v. Ashland Chemical, Inc.*, 151 F.3d 269, 278 (5th Cir. 1998) (en banc), *cert. denied*, 119 S. Ct. 1454 (1999), for example, the Fifth Circuit found that the expert's reliance on the temporal relationship between the exposure and the onset of symptoms was entitled to little weight in the absence of supporting medical literature. *See also* Rosen v. Ciba-Geigy Corp., 78 F.3d 316, 319 (7th Cir.) (rejecting expert testimony on nicotine patch as cause of heart attack that occurred after three days of wearing patch), *cert. denied*, 519 U.S. 819 (1996); Porter v. Whitehall Labs., Inc., 9 F.3d 607, 614 (7th Cir. 1993) (rejecting clinical observations and temporal relationship between drug ingestion and renal failure as bases for opinion on causation where scientific studies unavailable). On occasion, a temporal relationship that does not fit the expected pattern may be a basis for ruling out the suspected cause. *See, e.g.*, Heller v. Shaw Indus., Inc., 167 F.3d 146, 157–58 (3d Cir. 1999) (temporal relationships may be important in supporting an opinion on causation, but expert's reliance on temporal relationship is flawed in this case). *See generally* Speizer, *supra* note 59, at 1429–36; Honig & Ingram, *supra* note 118, at 1452, 1456.

125. *See* Cullen et al., *supra* note 19, at 227.

126. The courts differ on the question whether the witness giving an opinion on causation must support his or her opinion with references to medical or scientific studies supporting a causal link between the toxic exposure and the plaintiff's disease. A number of courts have answered this question in the affirmative. *See, e.g.*, Moore v. Ashland Chem., Inc., 151 F.3d 269, 277–78 (5th Cir. 1998) (en banc), *cert. denied*, 119 S. Ct. 1454 (1999); Rosen v. Ciba-Geigy Corp., 78 F.3d 316, 319 (7th Cir.)

cludes epidemiological studies and toxicology studies. The physician should be guided by the methods set forth in the Reference Guides on Epidemiology and Toxicology in evaluating this literature and its relevance to the patient's exposure and condition.[127]

Physicians also have access to case reports or case series in the medical literature. These are reports in medical journals describing clinical events involving one individual or a few individuals. They report unusual or new disease presentations, treatments, or manifestations, or suspected associations between two diseases, effects of medication, or external causes of diseases. For example, the association between asbestos and lung cancer was first reported in a 1933 case report, although the first controlled epidemiological study on the association was not published until the 1950s.[128] There are a number of other instances in which epidemiological studies have confirmed associations between a specific exposure and a disease first reported in case studies (e.g., benzene and leukemia; vinyl chloride and hepatic angiosarcoma),[129] but there are also instances in which controlled studies have failed to substantially confirm the initial case reports (e.g., the alleged connection between coffee and pancreatic and bladder cancer or the infectious etiology of Hodgkins disease).[130]

(witness cited no scientific or medical literature, or other explanation of asserted causal relationship between nicotine patch and heart attack), *cert. denied*, 519 U.S. 819 (1996); Porter v. Whitehall Labs., Inc., 9 F.3d 607, 615 (7th Cir. 1993) (medical literature did not establish link between ibuprofen and plaintiff's kidney ailment; medical theories had not been tested). Other courts have upheld the admission of medical opinion based solely on clinical observations and reasoning, sometimes with reference to the physician's experience with similar kinds of patients or cases. *See, e.g.*, Heller v. Shaw Indus., Inc., 167 F.3d 146, 153–57 (3d Cir. 1999); Westberry v. Gummi, 178 F.3d 257, 262–66 (4th Cir. 1999) (affirmed trial court's admission of expert testimony on talc as cause of plaintiff's sinus problems despite absence of supporting medical literature); Fadelalla v. Secretary of the Dep't of Health & Human Servs., No. 97-05730, 1999 WL 270423, at *6 (Fed. Cl. Apr. 15, 1999) (while clinical experience may be sufficient to establish causal relationship, in this case expert had insufficient clinical experience on which to base an opinion on causation); Becker v. National Health Prods., Inc., 896 F. Supp. 100, 103 (N.D.N.Y. 1995) (absence of published literature on relationship between diet supplement and diverticulosis not fatal to plaintiff's case where expert relied on "differential etiology").

127. *See* Michael D. Green et al., Reference Guide on Epidemiology, §§ V–VII, and Bernard D. Goldstein & Mary Sue Henifin, Reference Guide on Toxicology, §§ III–V, in this manual.

128. *See* Michael Gochfeld, *Asbestos Exposure in Buildings*, *in* Environmental Medicine, *supra* note 19, at 438, 440.

129. *See* Michael Gochfeld, *Chemical Agents*, *in* Environmental Medicine, *supra* note 19, at 592, 600 (vinyl chloride); Howard M. Kipen & Daniel Wartenberg, *Lymphohematopoietic Malignancies*, *in* Textbook of Clinical Occupational and Environmental Medicine 555, 560 (Linda Rosenstock & Mark R. Cullen eds., 1994) (benzene).

130. Kristin E. Anderson et al., *Pancreatic Cancer*, *in* Cancer Epidemiology and Prevention 725, 740–41 (David Schottenfeld & Joseph F. Fraumeni, Jr., eds., 2d ed. 1996); Debra T. Silverman et al., *Bladder Cancer*, *in* Cancer Epidemiology and Prevention, *supra*, at 1156, 1165–66.

Case reports lack controls and thus do not provide as much information as controlled epidemiological studies do.[131] However, case reports are often all that is available on a particular subject because they usually do not require substantial, if any, funding to accomplish, and human exposure may be rare and difficult to study. Causal attribution based on case studies must be regarded with caution. However, such studies may be carefully considered in light of other information available, including toxicological data.[132]

3. Clinical Evaluation of Information Affecting Dose–Response Relationships

Assessing the role of external causes in the patient's condition requires the integration of the information described in the preceding sections, with particular attention to dose–response relationships. The toxicological law of dose–response, that is, that "the dose makes the poison," refers to the general tendency for greater doses of a toxin to cause greater severity of responses in individuals, as well as greater frequency of response in populations.[133] Clinically, there are some instances in which the general rule does not hold. For agents that cause an allergic response through an immunologic mechanism, the dose–response relationship is often less straightforward. Many people who are not prone or able to develop an allergic reaction, for genetic or other reasons, will not respond adversely to the substance at any dose. However, those who are susceptible are more likely to become specifically reactive (sensitized) to the specific agent as the dose increases. After sensitization has occurred, severe reactions may occur with exposures that are much lower than the previous level required for sensitization.[134]

Although some diseases (e.g., pneumonia that is due to influenza) are frequently considered to be unifactorial, the possibility of multiple causes of a clini-

131. *See generally* Michael D. Green et al., Reference Guide on Epidemiology § II.A, in this manual.

132. *See* Cullen et al., *supra* note 19, at 226. Courts have given varying treatment to case reports. *Compare* Haggerty v. Upjohn Co., 950 F. Supp. 1160, 1165 (S.D. Fla. 1996) (case reports are "no substitute for a scientifically designed and conducted inquiry" (citing Casey v. Ohio Med. Prods., 877 F. Supp. 1380, 1385 (N.D. Cal. 1995))), *aff'd*, 158 F.3d 588 (11th Cir. 1998) (unpublished table decision), *and* Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1411 (D. Or. 1996) (case reports "cannot be the basis of an opinion based on scientific knowledge"), *with* Pick v. American Med. Sys., Inc., 958 F. Supp. 1151, 1160–62, 1178 (E.D. La. 1997) (case studies on gel implants admissible in case on penile implant; theory developed by single physician not admissible), Glaser v. Thompson Med. Co., 32 F.3d 969, 975 (6th Cir. 1994) (ordering trial based on witness who relied on case reports and his own research in rendering opinion on diet pills as cause of intracranial bleeding and fall), *and* Cella v. United States, 998 F.2d 418, 426 (7th Cir. 1993) (in claim under Jones Act, medical opinion on cause of polymyositis based in part on case reports).

133. *See* Michael Gochfeld, *Principles of Toxicology*, *in* Environmental Medicine, *supra* note 19, at 65, 71–72.

134. *See* Cullen et al., *supra* note 19, at 228–29.

cal condition is a critical concern. At some level most diseases have multiple host and environmental factors that contribute to their presence. A commonly held misconception is that the presence of a nontoxic or other toxic cause for a condition automatically excludes a role for the toxin being considered as an external cause.[135] While this is sometimes true, in reality the converse can also be true. For example, epidemiology studies dealing with occupational asbestos exposure and cigarette smoking indicate that together they result in much higher rates of lung cancer than either one causes on its own.[136] Thus, two toxic agents have been found to interact in a synergistic manner so that their combined effects are much greater than even the sum of their individual effects.[137]

Even if causal factors do not interact synergistically, several may contribute in an incremental fashion to a disease and should not be assumed to be mutually exclusive.[138] Accordingly, the common statement that "alternative causes of disease must be ruled out" before causation is attributed can be more accurately refined to say that "the role of other causes must be adequately considered." If there is a significant rate of disease of unknown etiology (i.e., other causes or risk factors have not been identified), the determination of external causation

135. Some courts have stated that the plaintiff must offer a "differential diagnosis" to rule out other causes, whereas other courts have rejected such a requirement. *Compare* Wheat v. Pfizer, Inc., 31 F.3d 340, 342 (5th Cir. 1994) (witness failed to rule out hepatitis C and another drug as causes of plaintiff's liver disease), Mancuso v. Consolidated Edison Co., 967 F. Supp. 1437, 1446 (S.D.N.Y. 1997) ("differential diagnosis" required to rule out other possible causes; plaintiff's complaints were commonplace ailments), *and* National Bank of Commerce v. Dow Chem. Co., 965 F. Supp. 1490 (E.D. Ark. 1996) (case dismissed because, inter alia, plaintiffs failed to exclude other causes), *aff'd*, 133 F.3d 1132 (8th Cir. 1998), *with* Curtis v. M&S Petroleum, Inc., 174 F.3d 661, 670–72 (5th Cir. 1999) (rejecting requirement of "differential diagnosis" to rule out other causes), *and* Heller v. Shaw Indus., Inc., 167 F.3d 146, 153–57 (3d Cir. 1999) (existence of possible alternative causes goes to weight, not admissibility).

136. Occupational asbestos exposure in nonsmokers increases the risk of lung cancer by a factor of about five, from about 11 per 100,000, for nonsmoking industrial workers not exposed to asbestos to about 58 per 100,000 for nonsmoking asbestos workers; a significant smoking history increases the rate of lung cancer by a factor of at least ten. *See* U.S. Surgeon Gen., U.S. Dep't of Health & Human Servs., The Health Consequences of Smoking: Cancer and Chronic Lung Disease in the Workplace 216 (1985); *see also* Rodolfo Saracci, *The Interactions of Tobacco Smoking and Other Agents in Cancer Etiology*, 9 Epidemiologic Revs. 175, 176–80 (1987). Because the effects of smoking and asbestos are multiplicative for lung cancer, the population of smoking asbestos workers has a lung cancer incidence of 5 times 10, or 50 times the background rates, rather than the 15-fold increase predicted by adding the separate risks. *See* U.S. Surgeon Gen., U.S. Dep't of Health & Human Servs., *supra*, at 216–17.

137. *See* Gochfeld, *supra* note 133, at 73.

138. For example, both occupational asthma and smoking can lead to impairment of pulmonary function, and the presence of one does not rule out a causal role for the other. *See* John H. Holbrook, *Nicotine Addiction*, *in* 2 Principles of Internal Medicine, *supra* note 42, at 2516, 2518; E.R. McFadden, Jr., *Asthma*, *in* 2 Principles of Internal Medicine, *supra* note 42, at 1419, 1419–21. *Cf.* Wheat v. Pfizer, Inc., 31 F.3d 340 (5th Cir. 1994), which involved a victim who died of hepatitis after taking two drugs known to cause liver damage. As to her claim against Pfizer, the manufacturer of one of the drugs, the court found the evidence inadequate, in part, for failing to exclude the possibility that her disease was caused by the other drug. *Id.* at 343. The plaintiff's witness offered the possibility that the hepatitis

cal condition is a critical concern. At some level most diseases have multiple host and environmental factors that contribute to their presence. A commonly held misconception is that the presence of a nontoxic or other toxic cause for a condition automatically excludes a role for the toxin being considered as an external cause.[135] While this is sometimes true, in reality the converse can also be true. For example, epidemiology studies dealing with occupational asbestos exposure and cigarette smoking indicate that together they result in much higher rates of lung cancer than either one causes on its own.[136] Thus, two toxic agents have been found to interact in a synergistic manner so that their combined effects are much greater than even the sum of their individual effects.[137]

Even if causal factors do not interact synergistically, several may contribute in an incremental fashion to a disease and should not be assumed to be mutually exclusive.[138] Accordingly, the common statement that "alternative causes of disease must be ruled out" before causation is attributed can be more accurately refined to say that "the role of other causes must be adequately considered." If there is a significant rate of disease of unknown etiology (i.e., other causes or risk factors have not been identified), the determination of external causation

135. Some courts have stated that the plaintiff must offer a "differential diagnosis" to rule out other causes, whereas other courts have rejected such a requirement. *Compare* Wheat v. Pfizer, Inc., 31 F.3d 340, 342 (5th Cir. 1994) (witness failed to rule out hepatitis C and another drug as causes of plaintiff's liver disease), Mancuso v. Consolidated Edison Co., 967 F. Supp. 1437, 1446 (S.D.N.Y. 1997) ("differential diagnosis" required to rule out other possible causes; plaintiff's complaints were commonplace ailments), *and* National Bank of Commerce v. Dow Chem. Co., 965 F. Supp. 1490 (E.D. Ark. 1996) (case dismissed because, inter alia, plaintiffs failed to exclude other causes), *aff'd*, 133 F.3d 1132 (8th Cir. 1998), *with* Curtis v. M&S Petroleum, Inc., 174 F.3d 661, 670–72 (5th Cir. 1999) (rejecting requirement of "differential diagnosis" to rule out other causes), *and* Heller v. Shaw Indus., Inc., 167 F.3d 146, 153–57 (3d Cir. 1999) (existence of possible alternative causes goes to weight, not admissibility).

136. Occupational asbestos exposure in nonsmokers increases the risk of lung cancer by a factor of about five, from about 11 per 100,000, for nonsmoking industrial workers not exposed to asbestos to about 58 per 100,000 for nonsmoking asbestos workers; a significant smoking history increases the rate of lung cancer by a factor of at least ten. *See* U.S. Surgeon Gen., U.S. Dep't of Health & Human Servs., The Health Consequences of Smoking: Cancer and Chronic Lung Disease in the Workplace 216 (1985); *see also* Rodolfo Saracci, *The Interactions of Tobacco Smoking and Other Agents in Cancer Etiology*, 9 Epidemiologic Revs. 175, 176–80 (1987). Because the effects of smoking and asbestos are multiplicative for lung cancer, the population of smoking asbestos workers has a lung cancer incidence of 5 times 10, or 50 times the background rates, rather than the 15-fold increase predicted by adding the separate risks. *See* U.S. Surgeon Gen., U.S. Dep't of Health & Human Servs., *supra*, at 216–17.

137. *See* Gochfeld, *supra* note 133, at 73.

138. For example, both occupational asthma and smoking can lead to impairment of pulmonary function, and the presence of one does not rule out a causal role for the other. *See* John H. Holbrook, *Nicotine Addiction*, *in* 2 Principles of Internal Medicine, *supra* note 42, at 2516, 2518; E.R. McFadden, Jr., *Asthma*, *in* 2 Principles of Internal Medicine, *supra* note 42, at 1419, 1419–21. *Cf.* Wheat v. Pfizer, Inc., 31 F.3d 340 (5th Cir. 1994), which involved a victim who died of hepatitis after taking two drugs known to cause liver damage. As to her claim against Pfizer, the manufacturer of one of the drugs, the court found the evidence inadequate, in part, for failing to exclude the possibility that her disease was caused by the other drug. *Id.* at 343. The plaintiff's witness offered the possibility that the hepatitis

may be complicated.[139] In general, if a patient is not subject to other known risk factors for a disease, it is more likely that the external cause is a factor in causing the patient's illness.[140]

Differences in individual susceptibility are commonly cited as the reason why one person gets sick from an environmental exposure while other persons are not affected. True individual susceptibility is based on genetic differences, such as immunologic reactivity, enzyme metabolism, and gender.[141] A number of other acquired factors, such as age, body mass, interacting simultaneous exposures, and preexisting disease, may also contribute to susceptibility.[142] Reliable and accurate information is available about the effects on some diseases of age, body mass, gender, and other factors; however, information on genetic susceptibility is available for only a few diseases, and information on the relation between genetic susceptibility and particular toxic exposures, for even fewer.[143]

resulted from the combined action of the two drugs, which the court rejected because the witness cited no study of the combined effects of the drugs. *Id.* The court also faulted the plaintiff for failing to rule out hepatitis C as a cause of the liver damage, though there was no test for the condition at that time. *Id.* at 342. *But see* Benedi v. McNeil-PPC, Inc., 66 F.3d 1378, 1384 (4th Cir. 1995) (upholding plaintiff's recovery for liver damage caused by Tylenol and alcohol consumption).

139. The problem of unidentified risks (often termed "background cases of unknown etiology") has been recognized in a number of decisions. For example, in *In re Breast Implant Litigation*, 11 F. Supp. 2d 1217 (D. Colo. 1998), the court disapproved of a physician's identification of silicone as the cause of the plaintiff's disease through "differential diagnosis," stating: "As a practical matter, the cause of many diseases remains unknown; therefore, a clinician who suspects that a substance causes a disease in some patients very well might conclude that the substance caused the disease in the plaintiff simply because the clinician has no other explanation." *Id.* at 1230. *See also* National Bank of Commerce v. Dow Chem. Co., 965 F. Supp. 1490 (E.D. Ark. 1996) (rejecting testimony that pesticide caused birth defect where witness acknowledged that causes are unknown for 70% to 80% of birth defects), *aff'd*, 133 F.3d 1132 (8th Cir. 1998); Whiting v. Boston Edison Co., 891 F. Supp. 12 (D. Mass. 1995) (in case alleging radiation caused power plant worker's acute lymphocytic leukemia, witness's acknowledgement that 90% of cases are of unknown cause cast doubt on "differential diagnosis" of cause); *In re* "Agent Orange" Prod. Liab. Litig., 611 F. Supp. 1223, 1250 (E.D.N.Y. 1985) ("Central to the inadequacy of plaintiffs' case is their inability to exclude other possible causes of plaintiffs' illnesses—those arising out of their service in Vietnam as well as those that all of us face in military and civilian life."), *aff'd*, 818 F.2d. 187 (2d Cir. 1987), *cert. denied*, 487 U.S. 1234 (1988). The plaintiff may be able to rely on inferences from epidemiological, toxicological, or other evidence, however. *See* Michael D. Green et al., Reference Guide on Epidemiology, and Bernard D. Goldstein & Mary Sue Henifin, Reference Guide on Toxicology, in this manual; *In re* Hanford Nuclear Reservation Litig., No. CV-91-3015-AAM, 1998 WL 775340 (E.D. Wash. Aug. 21, 1998).

140. This kind of reasoning is discussed in *In re Paoli Railroad Yard PCB Litigation*, 35 F.3d 717, 760 n.30 (3d Cir. 1994), *cert. denied*, 513 U.S. 1190 (1995).

141. *See* Stuart M. Brooks et al., *Types and Sources of Environmental Hazards, in* Environmental Medicine, *supra* note 19, at 9, 15–17; Daniel W. Nebert et al., *Genetic Epidemiology of Environmental Toxicity and Cancer Susceptibility: Human Allelic Polymorphisms in Drug-Metabolizing Enzyme Genes, Their Functional Importance, and Nomenclature Issues*, 31 Drug Metabolism Revs. 467 (1999); Maurizio Taningher et al., *Drug Metabolism Polymorphisms as Modulators of Cancer Susceptibility*, 436 Mutation Res. 227 (1999).

142. *See* Karen Reiser, *General Principles of Susceptibility, in* Environmental Medicine, *supra* note 19, at 351, 351–52, 358.

143. *See id.* at 357.

In almost all instances, integration of all the above factors into an opinion on causality cannot be reduced to mathematical formulas. There are inevitable gaps in information, as well as lack of knowledge regarding individual characteristics, such as susceptibility and resistance. Thus, clinical judgment is critical to opinions on diagnosis and causation for the individual patient even when the scientific population basis for general causation may be quite strong.

V. Treatment Decisions

Following diagnosis, most physicians are concerned with applying appropriate treatment to either cure or ameliorate a patient's condition. Such treatment may be surgical (e.g., removal of a diseased organ), ablative (e.g., radiotherapy aimed at a tumor), chemotherapeutic (e.g., use of pharmacological agents with a host of different actions), rehabilitative (e.g., physical therapy), interdictive (e.g., removal of the patient from a toxic or allergenic exposure), behavioral (e.g., counseling), or something else.[144] Some of the recommended therapies for different conditions found in the textbooks and professional literature are reified as practice guidelines by various organizations and the government. Some recommended therapies have demonstrated their effectiveness in randomized controlled trials, whereas others, both old and new, have much less scientific support.

Treatment options for an individual patient must be assessed in light of the nature and severity of the particular disease (e.g., people whose lung cancer is metastatic are not often candidates for removal of the primary tumor), and the likelihood of unacceptable complications from the treatment (e.g., removal of a lung to cure cancer in someone with severe emphysema may not leave enough remaining lung tissue to allow the patient to walk, even if his or her cancer is cured).[145] Prediction of the effects, both positive and negative, of a course of therapy is based on the professional literature and consideration of a patient's specific situation. For example, a patient with underlying kidney disease may not be an appropriate candidate for some radiographic tests and therapies that use dye that runs a high risk of causing further damage to the kidneys. Use of an effective antibiotic to which a patient "may possibly" have had a previous aller-

144. *See* Kassirer & Kopelman, *supra* note 48, at 11, 32–33.

145. A physician's selection of appropriate treatment is often at issue in medical malpractice cases (*see supra* notes 31–32 and accompanying text), but it also is at issue in other kinds of cases, including claims that medical treatment was "necessary" and therefore covered in insurance litigation under ERISA (*see, e.g.*, McGraw v. Prudential Ins. Co., 137 F.3d 1253, 1258–1263 (10th Cir. 1998)), claims that treatment was improperly withheld from prisoners under the Eighth Amendment (*see, e.g.*, Kulas v. Roberson, 202 F.3d 278 (9th Cir. 1999) (unpublished table decision) (text at No. 98-16954, 1999 WL 1054663 (9th Cir. Nov. 19, 1999) (mem.)), and medical monitoring claims (*see, e.g., In re* Paoli R.R. Yard PCB Litig., 916 F.2d 829, 852 (3d Cir. 1990), *cert. denied*, 499 U.S. 961 (1991)).

gic reaction should be weighed against the use of alternative antibiotics that may be less effective against the infection. The physician may also consider the likely severity of a reaction and the ability to prevent or treat it with additional medication. Thus, although treatment recommendations are often written down as a precise series of sequential decisions (often called algorithms), making decisions for actual patients is generally more complex and requires consideration of many individual factors.

VI. Medical Testimony: Looking to the Future

It is likely that medical testimony will continue to be one of the most common forms of expert testimony in the future. While many commentators have focused attention on medical testimony in toxic injury cases, particularly testimony offered on issues of external causation, a growing number of cases concern ERISA suits challenging coverage under health care plans and claims of unlawful discrimination under the Americans with Disabilities Act. As the health care system continues to evolve, there will be growing numbers of cases, particularly on coverage issues, requiring medical testimony. Also, advances in the medical sciences, including medical genetics and biotechnology, will present new challenges to courts in cases requiring medical testimony.

With this forecast, courts will continue to grapple with issues of admissibility of medical testimony for the foreseeable future. As the cases we have used to illustrate this chapter demonstrate, there are great and unresolved differences in how various courts treat the admissibility of medical testimony. While this reference guide does not propose legal standards to govern admissibility of medical evidence,[146] it does provide a framework for legal analysis by describing the scientific and professional practices of physicians as they perform their professional duties and offer opinions on diagnosis, treatment, and internal and external causation. It is challenging to encourage consistent use of medical terminology and make explicit the extensive knowledge base and reasoning process that physicians implicitly employ in evaluating medical problems. Further work in these areas will improve the transferability of medical knowledge into the courts and other arenas.

146. *See supra* note 30.

Glossary of Terms

adequacy of diagnostic hypothesis. Diagnostic sufficiency. To be considered adequate, a diagnostic hypothesis must explain the patient's normal findings as well as abnormal findings.

attending physician. A physician formally attached to (credentialed at) the hospital in which the patient is being treated.

Bayes' theorem. An algebraic formula that allows the pretest and posttest clinical data to be expressed in terms of probabilities. By integrating the pretest probability of a disease or set of diseases with the result of a given test (and taking into account the sensitivity and specificity of that test), the physician is able to calculate a posttest probability of a disease or set of diseases. This approach can be useful in certain circumstances, but many clinical situations can be so complex that it is impractical to apply Bayes' theorem.

case report/case series. The most basic type of descriptive study of an individual (case report) or a series of individuals (case series), usually including such factors as gender, age, and exposure or treatment, but without controlled assessment of the relationship between exposure or treatment and disease or outcome.

clinical tests. Noninvasive tests of the function of an organ system, including tests of pulmonary function, muscle function, endurance, and heart function.

coherency of a diagnostic hypothesis. In a coherent diagnostic hypothesis, the patient's findings (signs, symptoms, test results), risk factors, and complications match the expectations for the disease.

consulting physician. A physician brought in to give an expert opinion or a second opinion, who may or may not be involved in treatment. He or she may rely on information contained in the patient's medical records, patient history, laboratory tests, x-rays, and so forth, or may combine these facts with his or her own examination of the patient and any additional tests considered advisable.

diagnosis. The determination of which disease is most likely present in a given patient, as indicated by the patient's various symptoms, signs, and test results.

diagnostic hypothesis. One or more disease entities, conditions, or syndromes postulated to be responsible for causing a patient's clinical presentation. See working diagnosis.

diagnostic tests. Any tests (clinical, laboratory, or pathologic) whose results may assist the physician in making his or her diagnosis.

differential diagnosis. The term used by physicians to refer to the process of determining which of two or more diseases with similar symptoms and signs the patient is suffering from, by means of comparing the various competing diagnostic hypotheses with the clinical findings.

differential etiology. A term used on occasion by expert witnesses or courts to describe the investigation and reasoning that leads to a determination of external causation, sometimes more specifically described by the witness or court as a process of identifying external causes by a process of elimination.

disease. Coherent deviation from normal in structure or function that affects a certain part or parts of the body or type of tissue.

dose–response relationship. The general tendency to observe greater responses in individuals when they are given greater doses of a drug or toxic substance. The presence of such a relationship supports an inference of a causal relationship between exposure and response (disease).

external causation. As used herein, an underlying cause of a given disease in a given individual that stems from a source outside the individual's body. A hereditary disease such as Tay-Sachs disease or hemophilia would not be due to external causation; cirrhosis of the liver resulting from excessive alcohol intake or ataxia resulting from lead poisoning would be due to external causation.

general causation. General causation is established by demonstrating (usually by reference to a scientific publication) that exposure to the substance in question causes (or is capable of causing) disease; for example, smoking cigarettes causes lung cancer.

inductive reasoning. See inferential reasoning.

inferential reasoning. The reasoning process by which a physician assimilates the various findings on a given patient and forms hypotheses that lead to testing and further hypotheses until a coherent diagnosis is reached.

invasive procedure. A procedure (surgery, test, etc.) in which the body of the patient is invaded by an instrument of some sort. Invasive procedures may be as minimal as the biopsy of a lesion on the skin or as traumatic as open-heart surgery.

laboratory tests. Analyses of fluids or other substances collected from the body of the patient, including blood samples, urine samples, and fecal samples.

multiplicative interaction. A process that occurs when two toxic agents (or two disease states) interact in the patient in such a manner that the magnitude of their combined effects is equal to the product of the effect of each agent (or disease) working in isolation. This is a special instance of synergism.

noninvasive procedure. A procedure (usually a test procedure) that does not invade the body of the patient, including exercise and stress tests, electrocardiograms, CAT scans, and MRIs.

parsimony in a diagnostic hypothesis. A preference for the simplest way to coherently and adequately explain all of the patient's findings, normal and abnormal.

pathogenesis. The mode of origin or development of any disease or morbid process.

pathology test. Microscopic analysis of a piece of body tissue obtained during surgery or by biopsy, in which an expert determines whether the tissue appears to be normal for the organ form from which it was taken. If it does not appear normal, the expert then attempts to determine what the pattern of abnormality is (scarring, malignancy, inflammation, etc.)

pathophysiology. The derangement of function seen in disease; alteration in function as distinguished from structural disease.

patient history. An interview conducted by the treating physician with the patient, in which the physician elicits from the patient the symptoms he or she is suffering from, as well as information about past and present medical history and treatment, personal information on family status and lifestyle, environmental information about habitation and employment, and the like.

physical exam. A noninvasive, largely external examination of the patient's body in which the physician looks for signs of normal and abnormal function. The physician may do a physical examination of a healthy individual to fulfill the requirements of an employer or insurance company, or of a patient who is ill to substantiate or refute the symptoms obtained from a patient during the taking of the patient history.

predictive value. The extent to which a given test will predict the presence or absence of a given disease. The positive predictive value of a test or observation refers to the proportion of all positive results that are "true" positive test results in a particular population. The negative predictive value of a test or observation refers to the proportion of "true" negative results in a population.

sensitivity. The percentage of patients with positive test results for a disease who actually have the disease (called a "true positive" result). Test results for those who have a disease but are incorrectly identified as not having the disease because of the test's insensitivity are called "false negatives." A test with high sensitivity given to people suffering from the disease it tests for will have a high proportion of true positives and only a few false negatives. A test with low sensitivity will reveal a considerable number of false negatives and fewer true positives.

sensitization. The initial exposure of a person to a specific antigen (any substance that is capable of inducing an immune reaction in an individual and of reacting with the products of that response); repeated exposure to the same antigen may then result in a much stronger immune response (e.g., an individual stung by a bee on one occasion may have a stronger response if stung again, and if subjected to sufficient numbers of bee stings, may eventually react by going into anaphylactic shock).

sign. A physical condition observed in a patient by the physician in the course of a physical examination, such as fever, cardiac murmur, enlarged lymph nodes, suspicious breast mass.

specific causation. Specific, or individual, causation is established by demonstrating that a given exposure is the cause of an individual's disease (for example, that a given plaintiff's lung cancer was caused by smoking).

specificity. The percentage of negative test results in individuals who are free of a given disease, also known as the "true negative" rate. Test results in those who are free of the disease who are incorrectly identified as having the condition are called "false positives." Thus, a test that indicates abnormal bronchial reactivity in 15% of individuals without asthma would have a false positive rate of 15%; their test results were positive, but they are free of the condition.

susceptibility. The propensity of an individual to be harmed by an agent (e.g., a person who has a high susceptibility to irritant gases will suffer from bronchitis or asthma more than a person with a low susceptibility). Susceptibility tends to be influenced by age, gender, and genetics as well as the individual's state of health and history of prior exposure.

symptom. A patient's subjective report of physical abnormality as described to the physician during the taking of the patient history. Symptoms may include reports of pain in various parts of the body, sensations such as dizziness or fatigue, fever or chills, or swelling or suspicious nodules. If a symptom, such as fever or the existence of a suspicious breast nodule, is verified by the physician during the physical exam, it is considered a sign.

syndrome. A clustering of the symptoms, signs, and laboratory findings that indicate a specific disease state.

synergistic interaction. The joint action of two or more agents such that their combined effect is greater than the sum of the effects of each agent working separately. See multiplicative interaction.

threshold. The lowest dose of any substance at which a measurable response occurs. For a substance that produces more than one effect, the threshold may vary according to the effect. For instance, with a neurotoxin that can

produce dizziness, convulsion, coma, and death, the thresholds for the different effects can vary from quite low for dizziness to relatively high for death.

treating physician. A physician in charge of diagnosis and therapy for a given patient. The treating physician is likely to be an attending physician at the hospital to which the patient has been admitted. Many physicians will act as treating physicians with patients for whom they provide primary care, but may be called upon to act as consulting physicians at the request of colleagues or the patients of other physicians.

working diagnosis. A diagnostic hypothesis sufficiently convincing to form the basis for planning the next step in patient management. A working diagnosis may provide a rationale for the physician to order further tests, to forecast a likely clinical course for the patient, to refrain from further testing and simply to observe the patient for a given time, or to initiate a course of treatment. If a working diagnosis proves to be correct, either by subsequent testing or by patient response, it may become the final diagnosis.

References on Medical Testimony

Thomas E. Andreoli et al., Cecil Essentials of Medicine (3d ed. 1993).

Barbara Bates et al., A Guide to Physical Examination and History Taking (6th ed. 1995).

Joan E. Bertin & Mary S. Henifin, *Science, Law, and the Search for the Truth in the Courtroom,* 22 J.L. Med. & Ethics 6 (1994).

Environmental Medicine (Stuart M. Brooks et al. eds., 1995).

1 & 2 Harrison's Principles of Internal Medicine (Anthony S. Fauci et al. eds., 14th ed. 1998).

Alvan R. Feinstein, Clinical Judgment (1967).

Michael D. Green, Bendectin and Birth Defects: The Challenges of Mass Toxic Substances Litigation (1996).

Jerome P. Kassirer & Richard I. Kopelman, Learning Clinical Reasoning (1991).

Susan R. Poulter, *Medical and Scientific Evidence of Causation: Guidelines for Evaluating Medical Opinion Evidence, in* Expert Witnessing: Explaining and Understanding Science 186 (Carl Meyer ed., 1999).

Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?* 7 High Tech. L.J. 189 (1992).