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# Family Practice Grand Rounds

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## Care of the Elderly, Demented, and Dying Patient

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DR. RICHARD B. SALMON (*Assistant Professor, Department of Family Medicine*): Today we focus on the case of a demented, elderly man who became acutely ill and died in the hospital. Initially the patient was brought by his family for evaluation of his dementia to the University Hospitals Center for Assessment and Care of the Elderly (UHCACE), an ambulatory assessment unit that includes a family physician, a psychiatrist, an internist, a neurologist, a nurse clinician, and a social worker. The case illustrates several important themes in the care of demented, elderly patients: the value of a team approach oriented toward the family, the severity of adjustment and grief reactions, the decision to withhold extraordinary care, and the difficulty that the health care team experiences in caring for the patient. The case discussants include several members of the Center for Assessment and Care of the Elderly and Dr. Robert Kohn, a pathologist, who will discuss the implications of aging as a cause of death in the elderly.

Mr. M. was an 88-year-old white retired sheet-metal worker whose wife had recently died of pancreatic cancer after a short terminal illness. Mrs. M. had been successfully covering up Mr. M.'s deficiencies, and after her death the family became aware of an unsuspected degree of dementia in Mr. M. Although Mr. M. knew his intimate family members by name, he did not know most of the people at his wife's funeral. He had little awareness of her death and kept asking where she was.

During this time his agitation increased, and his previous physician prescribed haloperidol in low doses. He was still able, for the most part, to care for himself. He spent his time either listening to the radio or watching television. Approximately two weeks before the evaluation process began, the family had placed him in a rest home for the elderly. He initially seemed to adjust well to that location and had started forming relationships with the staff.

His past medical history was significant for heart disease including congestive heart failure, atrial fibrillation, and chest pain for which he was treated with digoxin, isosorbide dinitrate, and hydrochlorothiazide without functional impairment. There was no history of alcohol or tobacco abuse, and the family history was negative for Alzheimer's disease. Physical examination revealed a well-developed, well-nourished white man who was cooperative and pleasant. His blood pressure was 145/92 mmHg and his heart rate was 82 beats/min. The cardiorespiratory examination revealed no signs of failure. The neurologic examination was without focal findings. Mental status examination showed that he did not know the day, date, year, current president of the United States, his address, or his age. He did know his birth date and that he was in a physician's office. He could remember two of three objects after two minutes. He knew how an apple and an orange were alike; however, he did not know how a car and a boat were alike. He could not add a penny, a nickel, and a dime, and he was concrete with his interpretations of proverbs.

During the examination he had an episode of nausea and diaphoresis lasting several minutes accompanied by new T-wave depressions in ECG leads II, III, aV<sub>F</sub>, V<sub>4</sub>, V<sub>5</sub>, and V<sub>6</sub>. The patient

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## Motrin® Tablets (ibuprofen)

**Contraindications:** Anaphylactoid reactions have occurred in individuals hypersensitive to Motrin Tablets or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin, iodides, or other nonsteroidal anti-inflammatory agents.

**Warnings:** Peptic ulceration and GI bleeding, sometimes severe, have been reported. Ulceration, perforation and bleeding may end fatally. An association has not been established. Use Motrin Tablets under close supervision in patients with a history of upper gastrointestinal tract disease, after consulting ADVERSE REACTIONS. In patients with active peptic ulcer and active rheumatoid arthritis, try nonulcerogenic drugs, such as gold. If Motrin Tablets are used, observe the patient closely for signs of ulcer perforation or GI bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity with papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with Motrin Tablets.

**Precautions:** Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin Tablets and the patient should have an ophthalmologic examination, including central visual fields and color vision testing.

**Fluid retention and edema** have been associated with Motrin Tablets; use with caution in patients with a history of cardiac decompensation or hypertension. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of Motrin Tablets safety in patients with chronic renal failure have not been done.

Motrin Tablets can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, skin rash, weight gain, or edema.

Patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin Tablets are added.

The antipyretic, anti-inflammatory activity of Motrin Tablets may mask inflammation and fever.

As with other nonsteroidal anti-inflammatory drugs, borderline elevations of liver tests may occur in up to 15% of patients. These abnormalities may progress, may remain essentially unchanged, or may be transient with continued therapy. Meaningful elevations of SGPT or SGOT (AST) occurred in controlled clinical trials in less than 1% of patients. Severe hepatic reactions, including jaundice and cases of fatal hepatitis, have been reported with ibuprofen as with other nonsteroidal anti-inflammatory drugs. If liver disease develops or if systemic manifestations occur (e.g. eosinophilia, rash, etc.), Motrin should be discontinued.

**Drug interactions.** Aspirin: used concomitantly may decrease Motrin blood levels.

**Coumarin:** bleeding has been reported in patients taking Motrin and coumarin.

**Pregnancy and nursing mothers:** Motrin should not be taken during pregnancy or by nursing mothers.

**Adverse Reactions:** The most frequent type of adverse reaction occurring with Motrin is gastrointestinal of which one or more occurred in 4% to 16% of the patients.

### **Incidence Greater than 1% (but less than 3%)—Probable Causal Relationship**

**Gastrointestinal:** Nausea,\* epigastric pain,\* heartburn,\* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence); **Central Nervous System:** Dizziness,\* headache, nervousness; **Dermatologic:** Rash\* (including maculopapular type), pruritus; **Special Senses:** Tinnitus; **Metabolic/Endocrine:** Decreased appetite; **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

### **Incidence less than 1%—Probable Causal Relationship\*\***

**Gastrointestinal:** Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests; **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma; **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme; Stevens-Johnson syndrome, alopecia; **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS); **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit; **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations; **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting; anaphylaxis; bronchospasm (see CONTRAINDICATIONS); **Renal:** Acute renal failure in patients with pre-existing significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria; **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

### **Incidence less than 1%—Causal Relationship Unknown\*\***

**Gastrointestinal:** Pancreatitis; **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri; **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions; **Special Senses:** Conjunctivitis, diplopia, optic neuritis; **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction; **Cardiovascular:** Arrhythmias (sinus tachycardia, sinus bradycardia); **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schönlein vasculitis; **Renal:** Renal papillary necrosis.

\*Reactions occurring in 3% to 9% of patients treated with Motrin. (Those reactions occurring in less than 3% of the patients are unmarked.)

\*\*Reactions are classified under "Probable Causal Relationship (PCR)" if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under "Causal Relationship Unknown" if seven or more events have been reported but the criteria for PCR have not been met.

**Overdosage:** In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine so alkaline diuresis may be beneficial.

**Dosage and Administration:** Rheumatoid arthritis and osteoarthritis. Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Do not exceed 2400 mg per day. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary.

**Caution:** Federal law prohibits dispensing without prescription.

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## ELDERLY DYING PATIENT WITH DEMENTIA

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agreed to hospitalization, and after talking with his daughter, he was admitted to the Coronary Monitoring Unit. Serial enzymes, thyroid function tests, VDRL, B<sub>12</sub>, and folate level were normal. An electroencephalogram (EEG) showed diffuse slowing without any focal findings. A computerized axial tomography (CT) scan was unsuccessful because of the patient's agitation. We concluded that Alzheimer's disease was the most likely cause of the dementia. Day by day he was less able to tolerate the acute hospital environment, and he was medicated with haloperidol with increasing frequency. A chest roentgenogram performed prior to discharge showed a right lower lobe infiltrate. Since he was afebrile and asymptomatic, he was treated with oral erythromycin.

On the patient's return to the rest home, the family thought that he did well initially. He repeatedly asked for his wife, and he was told that she had died. Eventually, his appetite decreased, and he became progressively more lethargic. In retrospect the family felt that this change correlated with his understanding his wife's death. Two weeks after discharge from the hospital, he was found to be poorly responsive and was readmitted.

Physical examination revealed a temperature of 39.9°C, a blood pressure of 150/80 mmHg, and bilateral rales. There were no other signs of congestive heart failure, and neurologic examination was without focal findings. Laboratory examination showed new elevations in the white blood cell count (WBC) ( $14 \times 10^3/\mu\text{L}$  with 71 percent segmented cells and 20 percent band cells), BUN (53 mg/dL), and creatinine (1.7 mg/dL). The albumin was decreased (2.4 mg/dL) and the ECG was without change. Sputum examination revealed gram-negative rods and gram-positive cocci in chains. A spinal tap was negative, and a CT scan showed only atrophy. The portable chest roentgenogram showed increased densities in both lower lung fields consistent with infection or pulmonary edema. The patient was skin-test negative for tuberculosis, mumps, and candida.

Nafcillin and tobramycin were begun, and when the sputum culture grew Klebsiella, cefamandole was substituted. Because of the 80 percent mortality from gram-negative pneumonia in the elderly,<sup>1</sup> I warned the family that the patient was seriously ill. The daughter agreed that the patient was not to be resuscitated or electively transferred to an intensive care unit.

Mr. M. initially responded to rehydration and antibiotics with an improvement in mental status and a decrease in his BUN, creatinine, and white blood cell count. Because of inadequate oral intake, on the fifth day peripheral hyperalimentation was started. He continued to improve, and the family began the process of selecting a nursing home.

On the ninth hospital day Mr. M. pulled out six intravenous catheters. He was given cefamandole by intramuscular injection and extra efforts were made to encourage oral intake. By the 12th hospital day we became concerned because he continually called out for his wife, his oral intake was still poor, he had developed a sacral decubitis and a peripheral phlebitis, and his WBC had increased. Local therapy was begun for the decubitis and the phlebitis. The pulmonary consultant advised that Mr. M.'s persistent bilateral infiltrates were more likely due to heart failure than infection. Cefamandole was stopped and the dosage of isosorbide dinitrate was increased.

At this point the house staff questioned the appropriateness of continued active medical treatment of this depressed, demented patient. Also, they felt that antidepressant medication was urgently needed. I felt that we were obligated to take reasonable steps to find and treat infection, which might account for this patient's acute deterioration. I also felt that provision of a sitter for the patient during the day and eventual placement in a good nursing home were the main methods of treatment of Mr. M.'s adjustment reaction. I spoke with Mr. M.'s daughter daily at this point to include her in these decisions.

A private-duty nurse was hired, and the patient began to improve. By the 18th hospital day he recognized his daughter and was able to joke with her. Discharge to a nursing home was again anticipated. However, on the 21st hospital day he became more lethargic. By the 24th hospital day he was responsive to pain only, his weight had decreased, and his WBC had increased. His chest x-ray examination results had not changed. At that time the patient clearly suffered from multiple system failure (brain, lung, heart, renal, immune), all of which had deteriorated despite a substantial period of reasonable care. After consulting his daughter, we therefore decided not to actively intervene. The patient died that day.

I interviewed the daughter two months later, and she stated that in retrospect, she felt Mr. M.'s

overall deterioration started when he realized his wife had died. Also, she felt she would have been happier if we had been less aggressive with his treatment.

DR. LAWRENCE BRESLAU (*Associate Professor, Department of Psychiatry*): This is an instructive case because of the controversial issues it raises in the care of patients with Alzheimer's disease. Generally, at the time of their initial presentation, patients with Alzheimer's disease are mostly concerned with their memory loss and their relationships with other people. They may seek help from their own physician, a social worker, or the Alzheimer's Society. Some work is necessary at this point to make a correct diagnosis, to educate the patient and family, and to help them to establish a stable care system. The family begins to behave differently toward the patient by being more available and by better understanding the patient's needs. In turn, the patient is comforted and reassured by the care system.

Ultimately a second crisis develops when some alteration occurs in the ability of the care system to meet the patient's needs. For example, the patient's disability may grow beyond the capabilities of the care system either because of the natural progression of his disease or because of a superimposed acute brain syndrome resulting from such things as toxicity of medications, infections, or fluid and electrolyte disorders. In another example, as in this case, a member of the care system is no longer available because of illness, death, or other commitments. Under these circumstances the patient may develop any number of psychiatric symptoms depending as much on the character of the patient as on the precipitating event or the extent of memory loss. Those symptoms range from depression to paranoia or a confusional state that is due to the marginal function of the cerebral cortex. The patient is frequently hospitalized because of these symptoms. At that time, the patient is seen by the hospital staff through a "clinical window," which may not adequately allow preceding events to become visible. An understanding of what has happened depends largely upon understanding what has happened to the patient's care system over the long term. This case illustrated this point nicely. This patient became depressed not because his wife died, but because his care system fell apart. At this point, the issue of who was to care for him was much more important

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**ALDOMET® (Methyldopa)MSD**

Tablets, containing 125, 250, or 500 mg methyldopa; Oral Suspension, containing 250 mg methyldopa per 5 ml and alcohol 1%.

**Contraindications:** Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

**Warnings:** It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions. With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood. At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstated. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, occasionally with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstated in such patients. Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

**Pregnancy and Nursing:** Use of any drug in women who are or may become pregnant or intend to nurse requires that anticipated benefits be weighed against possible risks; possibility of fetal injury or injury to a nursing infant cannot be excluded. Methyldopa crosses the placental barrier, appears in cord blood, and appears in breast milk.

**Precautions:** Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: urinary uric acid by the phosphotungstate method, serum creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

**Adverse Reactions:** *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression. *Cardiovascular:* Bradycardia, prolonged carotid sinus hypersensitivity, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear) *Gastrointestinal:* Nausea, vomiting, distention, constipation, flatus, diarrhea, colitis, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis. *Hepatic:* Abnormal liver function tests, jaundice, liver disorders. *Hematologic:* Positive Coombs test, hemolytic anemia. Bone marrow depression, leukopenia, granulocytopenia, thrombocytopenia. Positive tests for antinuclear antibody, LE cells, and rheumatoid factor. *Allergic:* Drug-related fever, lupus-like syndrome, myocarditis. *Dermatologic:* Rash as in eczema or lichenoid eruption; toxic epidermal necrolysis. *Other:* Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, hyperprolactinemia, amenorrhea, impotence, decreased libido, mild arthralgia, myalgia.

**Note:** Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third months of therapy; increased dosage or adding a diuretic frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

For more detailed information, consult your MSD Representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, PA 19486

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than the loss of a loving relationship. His wife died and he was placed in an inadequately staffed rest home.

MS. INA POWELL (*Nurse Clinician, Department of Family Medicine*): I visited the rest home and found Mr. M. immobile in a chair in the corner. No one was available with the expertise to help him with his physical or emotional problems. Although the family visited him, I think that he perceived them as saying, "We don't care enough to have you in a place where you would receive excellent care."

DR. BRESLAU: The radical change in his care system caused him to become anxious and depressed. This concept guides the decision concerning use of antidepressant medication. Antidepressants may cause cardiotoxicity and confusion and may have limited efficacy; therefore, in this case, the appropriate course was to increase his care in the hospital by hiring a private-duty nurse and to work toward eventual placement in a high-quality nursing home.

AUDIENCE PHYSICIAN: I always feel somewhat reticent about being aggressive and preserving a man whose family is making clear signals that they are not interested. I don't know if that is a fair conclusion, but being left in the corner of a nursing home does not seem appealing.

DR. BRESLAU: It's often hard to be convinced, but a patient such as this man may get better in a week or two and live another ten years. Many significantly demented patients do require long-term nursing home placement. Placement is not necessarily catastrophic. There are many people in nursing homes whose families still enjoy them and they still enjoy their families. Both the patient and the family often require intensive professional support. When one speaks legitimately about letting a patient die, I think one speaks about someone whose quality of life is very much worse than the patients one sees under these circumstances.

DR. ROBERT KOHN (*Professor, Department of Pathology*): In terms of debility and disease, this is quite a classic case. I would disagree with Dr. Breslau somewhat, although I qualify my disagreement with the knowledge that anytime we make a judgment about an individual on the basis of statistics, we are taking a chance. I think that an 88-year-old man with this history has probably

reached the end of his life. As his signs and symptoms evolved, I think it was clear that he was going to die. We may not always be able to predict the death-precipitating mechanism, but that probably isn't so important as the family dynamics. I am led to this conclusion by my findings from a recent review of autopsies on 20 persons over 85 years of age that I will describe.<sup>2</sup> First, however, I would like to make some general comments about the aging process.

Aging is a normal process, occurring in all members of the population; it is progressive and irreversible. There are three categories of age-related diseases. First are diseases that are aging processes themselves. The best example is atherosclerosis, which plays a role in disease of the heart, blood vessels, central nervous system, and kidneys. Other examples are osteoarthritis, emphysema, and osteoporosis. A second category of age-related disease consists of those diseases in which the incidence increases with age, such as hypertension. Deaths that are due to hypertension increase with age in a manner that parallels the increase in death due to all causes. This parallelism makes it appear that hypertension is an aging process in the American population. There are individuals, however, who never become hypertensive. The disease therefore lacks the inevitability of an aging process. Many neoplastic diseases are in this category. A third category of age-related diseases includes those diseases that are characterized not by universality or by increasing incidence in age, but rather by an increasing inability with age of the afflicted person to recover from them. Here we consider groups of physiologic insults that cause death in older people but have only minor consequences in younger people. Today's case is a perfect example. Pneumonia resulted in Mr. M.'s death, whereas a younger person might not even have been hospitalized. As individuals age, they can't react to stresses in the way they could at a younger age.

If we look at deaths per 100,000 as a function of age, the probability of dying doubles about every eight years after growth stops. If there were no deaths resulting from either cancer or atherosclerosis, there would be an increase of only about ten years' life expectancy at birth. Major causes of death would then be pneumonia and accidents.

DR. SALMON: Does the accident category include people who die after hip fractures?

DR. KOHN: Yes, and that question illustrates a

central problem. A typical case is an elderly woman who breaks her hip because of osteoporosis. She is hospitalized and ends up dying from pneumonia. Our vital statistics are unreliable because the physician has no clear guidelines on what to record on the death certificate.

The vital statistics do indicate that the peak rate for the major causes of death in white women, for example, is consistently around the age of 88 years, even though the causes of death are not directly related to each other. This information suggests to me that everyone has a fatal disease called aging, and what is listed on the death certificate is often a complication of aging rather than a primary disease process.

To pursue this suggestion further, and as the vital statistics (based on death certificates) are often unreliable, I reviewed the autopsy findings from 200 persons who had died after they reached the age of 85 years. I asked, "What was the cause of death in these people that would have caused death in a middle-aged person?" No acceptable cause of death was found in about 30 percent of the cases. Many of these people died, therefore, because every physiologic process concerned with the maintenance of homeostasis drops off in a roughly linear fashion after growth stops.<sup>3</sup> As humans age, the ability to react to physiologic insults decreases to a level at which life cannot be maintained in the face of rather trivial tissue injury.

Before applying this concept to individuals, we must remember that at each age there is a broad range of physiologic capacity. Nonetheless, it appears in the case of Mr. M. that he could not defend himself against infection. The issue of how far to push medical care in an individual case is a complex one. Many of the diseases of old people are treatable or otherwise modifiable; moreover, each physician must be aware of whether he is really doing something that will help the patient or something that will just make the physician feel better while making the patient and the patient's family miserable. I can't help wondering whether Mr. M. might not have been better off if he had never gone into a hospital, and, instead, had died at home surrounded by his loved ones a week or two earlier.

MS. POWELL: Often at the University Hospitals Center for Assessment and Care of the Elderly, families complain of just the opposite trend. They feel their previous physicians have not paid enough attention to their elderly family member

with dementia and other disabilities. The family has been told that the problems are due to "old age," with the implication that nothing can be done. While often there is no cure, there are frequently many appropriate treatments for these problems.

DR. AMASA B. FORD (*Associate Chairman, Department of Epidemiology and Community Health*): I came into contact with this patient about 24 hours before he died. At that time, it was clear we were dealing with someone in the process of dying.

Physicians have had very complicated relationships with death over many years. The tendency to say that this problem has fundamentally changed its complexion with modern increase in medical technology may not be accurate. Dr. Alfred Worcester<sup>4</sup> wrote three essays in the 1930s entitled "The Care of the Aged," "The Care of the Dying" and "The Care of the Dead." He gave a very detailed description of the physical and psychological aspects of the dying patient. He pointed out, for example, that while the dying patient often feels cold to the touch, the patient does not express being cold; therefore, efforts to warm the patient are misguided. His essays are thoughtful and largely applicable today, although they were written more than 50 years ago.

Dame Cicely Saunders,<sup>5</sup> a British physician, founded St. Christopher's Hospice in 1967. This hospice was the first devoted to the care of the dying patient. She has now gone beyond the idea of an institution for the care of the dying and fully supports the home hospice care concept that is beginning to spread in the United States. She believes that well-organized home care strongly supported by the family and other willing caretakers is the proper direction for the hospice movement to grow.

Although Dr. Balfour Mount,<sup>6</sup> Director of the Palliative Care Unit of the Royal Victoria Hospital in Montreal, is an expert in pain control, he promotes the concept of a multidisciplinary team approach with both hospital ward and home visiting teams. He points out the need for the psychological, emotional, and spiritual care of the patient and the family as a unit.

I bring these people to your attention to illustrate the movement in medicine to help health care providers confront their own feelings about the care of the dying patient and to learn to be comfortable and competent in caring for such patients.

At the right moment, we must not evade the necessity of shifting goals from saving the patient to seeing the patient through his final crisis. This decision should not be based on age alone; it must be founded on a thoroughly balanced evaluation of the patient.

MS. POWELL: Dr. Ford, at what time after the death of a patient do you suggest meeting with the family?

DR. FORD: One or two months. Around the time of the death there is much confusion. About six weeks later reality hits the family. How is Mr. M.'s daughter doing?

DR. SALMON: She is doing better now. With the rapid loss of both parents, she was depressed to the point of being unable to cope with her financial affairs. She is now socializing more and functioning better. I think it helped that she was well prepared for her father's death.

DR. DAVID SCHMIDT (*Director, Department of Family Practice, University Hospitals*): I would like to comment on Mr. M.'s dying shortly after his wife's death. I wonder whether his death was due to the loss of a loved one or loss of a caretaker. The pattern is not so strong with the death of a caretaker. The mechanism could be founded on the immune system. Studies have shown that about eight weeks after a spouse's death, a subject's cellular immune response diminishes, which might put him at increased risk of infection.<sup>7</sup> In this case we know Mr. M. was anergic.

DR. SALMON: I would like to conclude by saying that by the day prior to Mr. M.'s death everyone on the health care team and the family knew his death was inevitable. Whether that realization should have come earlier in his clinical course is subject to debate. However, everyone is in agreement that such assessments must not be avoided. Appropriate decisions must be made with the health care team in consultation with the family.

## References

1. Hocking WG, Golde DW: The pulmonary alveolar macrophage. *N Engl J Med* 301:580, 1979
2. Kohn RR: Cause of death in very old people. *JAMA* 247:2793, 1982
3. Strohler M: General theory of mortality and aging. *Science* 132:14, 1960
4. Worcester A: *The Care of the Aged and Dying and the Dead*. Springfield, Ill, CC Thomas, 1939
5. Saunders C, Summers DH, Teller N (eds): *Hospice: The Living Idea*. London, Edward Arnold, 1981
6. Mount BM: The problem of caring for the dying in a general hospital: The palliative care unit as a possible solution. *Can Med Assoc J* 115:119, 1976
7. Bartrop RW, Lazarus L, Luckhurst E, et al: Depressed lymphocyte function after bereavement. *Lancet* 1:834, 1977