Abstract

The National Institutes of Health Consensus Development Conference on Diagnosis and Management of Asymptomatic Primary Hyperparathyroidism brought together endocrinologists, surgeons, radiologists, epidemiologists, and primary health care providers as well as the public to address indications for surgery in asymptomatic patients with hyperparathyroidism (HPT) and how patients not operated on should be monitored and managed to minimize the risk of complications of HPT. Following 1 1/2 days of presentations by experts and discussion by the audience, a consensus panel weighed the evidence and prepared their consensus statement.

Among their findings, the panel concluded that (1) a diagnosis of HPT is established by demonstrating persistent hypercalcemia together with an elevated serum parathyroid hormone concentration; (2) current and acceptable treatment for HPT is surgery to cure the condition; (3) the diagnosis of HPT in an asymptomatic patient does not in all cases mandate referral for surgery; conscientious surveillance may be justified in patients whose calcium levels are only mildly elevated and whose renal and bone status are close to normal; and (4) preoperative
localization in patients without prior neck operation is rarely indicated and not proven to be cost-effective.

The full text of the consensus panel's statement follows.

Introduction

Hyperparathyroidism is increasingly being recognized as a result of the detection of hypercalcemia by widespread use of multiphasic screening. Women are affected twice as often as men, and the incidence of hyperparathyroidism increases with age. Approximately 100,000 new cases occur each year in the United States. Because the disease is now known to be more common than previously appreciated, physicians are increasingly interested in the correct diagnosis and proper management of patients with hyperparathyroidism.

The increased recognition of hyperparathyroidism by screening tests has disclosed a population of patients in whom symptoms are subtle or absent. A new clinical profile of hyperparathyroidism that is characterized by mild hypercalcemia has emerged. Presentation with bone disease that is evident on standard radiographs, nephrolithiasis, or other complications is now uncommon, yet it is not clear that incidentally discovered hyperparathyroidism is benign.

Studies of the natural history of hyperparathyroidism are yielding information about how often and over what time course the mild syndrome remains benign. Silent loss of bone mass and changes in skeletal architecture in this asymptomatic population are being assessed with sensitive new techniques. The clinical significance of changes in bone density is uncertain, but it seems likely that progressive parathyroid-dependent bone loss is an additional risk factor for fractures. The potential for mild hyperparathyroidism to cause or accelerate hypertension, renal deterioration, peptic ulcer disease, and psychiatric symptoms also is being evaluated.

Parathyroidectomy is a highly successful treatment when performed by experienced surgeons. As there is evidence that some patients with asymptomatic primary hyperparathyroidism may have a prolonged benign course, it is possible that such patients can be managed without operative intervention. If patients are not operated on, they must be monitored to detect progression of the disease. For these patients, the principal issue is how this can best be accomplished, balancing the need to identify skeletal, renal, or other complications that are indications for operation against the burdens and expense of long-term monitoring.

Evaluation of the long-term consequences of asymptomatic
hyperparathyroidism with and without surgical treatment will answer questions about optimal management of this condition. Predictive factors, if they can be discerned, would help to distinguish subpopulations of patients who will develop adverse effects from those who tolerate mild hyperparathyroidism without complications. Identification of such factors would have a significant impact on the justification for operative or nonoperative management.

To address these issues, on October 29-31, 1990, the National Institute of Diabetes and Digestive and Kidney Diseases, together with the Office of Medical Applications of Research of the National Institutes of Health, convened a Consensus Development Conference on Diagnosis and Management of Asymptomatic Primary Hyperparathyroidism. Following a day and a half of presentations by experts in the relevant fields and discussion from the audience, a consensus panel comprising specialists and generalists from the medical and other related scientific disciplines considered the evidence and formulated a consensus statement in response to the following six previously stated questions:

- What is the most accurate, cost-effective method of diagnosing hyperparathyroidism?
- Are there patients with asymptomatic hyperparathyroidism who can safely be followed? Should they be?
- If not operated on, how should asymptomatic patients be monitored and managed?
- What are the indications for surgery in patients with asymptomatic hyperparathyroidism?
- What is the role of gland localization technology in management of patients with hyperparathyroidism?
- What research should be done to clarify issues in diagnosis and management of hyperparathyroidism?

**What is the Most Accurate, Cost-Effective Method of Diagnosing Hyperparathyroidism?**

The diagnosis of primary hyperparathyroidism (HPT) can best be established by demonstrating persistent hypercalcemia together with an elevated serum parathyroid hormone (PTH) concentration.

Measurement of total serum calcium concentration is a sensitive and cost-effective method for screening for primary HPT. When an elevated total serum calcium concentration is encountered, the clinician should first confirm this finding under conditions that minimize the likelihood of false positive values. The repeat blood sample should be obtained with minimal venous occlusion and preferably with the patient fasting. Drugs
such as thiazide diuretics that can increase serum calcium concentration should be discontinued for several days.

Because small elevations in serum calcium may be clinically significant, clinicians should know the stated normal range for the laboratories used. Total calcium measurements may be misleading in patients with decreased serum albumin, a problem that can be resolved by the use of an ionized serum calcium determination.

Additional pertinent data may be available from multiphasic screening results. Low serum phosphorous, high chloride, low bicarbonate, and high alkaline phosphatase concentrations are consistent with primary HPT but are not diagnostic; urea nitrogen and creatinine help in evaluating renal function.

Immunoassays for intact PTH using double antibody methods represent a major advance in diagnosis. The majority of patients with primary HPT have unequivocal elevations with these assays; the remainder have minimally elevated or high normal values. Patients with hypercalcemia due to other causes such as malignancy and sarcoidosis have low normal or suppressed PTH values. Because only rare instances of true ectopic secretion of PTH by malignant tumors have been reported, this possibility need only be considered in patients with elevated PTH and evidence of malignancy or in whom neck exploration for primary HPT is unsuccessful.

Borderline elevations or high normal values for intact PTH may be found in familial hypocalciuric hypercalcemia (FHH), an uncommon, benign condition in which neck exploration is contraindicated. In this syndrome, hypercalcemia often is detected at an early age and is associated with low urinary calcium excretion. Definitive diagnosis of FHH can be made by measuring serum and urine calcium in family members. Family studies also are important for detecting kindreds with multiple endocrine neoplasia (MEN) and familial HPT.

**Are There Patients With Asymptomatic Hyperparathyroidism Who Can Safely Be Followed? Should They Be?**

The consensus panel agrees that there may be a subgroup of patients with primary HPT that can be safely followed. All primary HPT patients should be considered candidates for surgery. Some uncomplicated asymptomatic patients, however, may be considered for judicious nonsurgical medical monitoring. To identify those patients who qualify for such management, physicians must have a clear understanding of "asymptomatic" primary HPT and undertake a rigorous evaluation and selection process to identify candidates who can be followed without
We use "asymptomatic primary HPT" to describe the clinical profile of patients with documented primary HPT without symptoms or signs commonly attributable to the disease. These patients are usually detected incidentally by multiphasic screening. Some patients may have one or several vague symptoms that cannot be definitively attributed to primary HPT but may instead be nonspecific or arise from a coexisting condition. Nevertheless, for purposes of this conference, such patients were considered "asymptomatic." In contrast, patients who present significant bone, renal, gastrointestinal or neuromuscular symptoms typical of primary HPT are defined as "symptomatic" and require surgery.

Our uncertainty regarding the natural history of asymptomatic primary HPT can be likened to the understanding of hypertension or hypercholesterolemia before large-scale epidemiological and clinical studies. There are no clinical signs or absolute laboratory criteria that can be used to identify patients who are likely to develop complications. Decisions regarding surgical or medical management must remain founded on clinical judgment on a case-by-case basis. The only acceptable treatment for these patients other than surgery is conscientious long-term medical surveillance.

**Indications for Medical Monitoring**

To qualify for nonsurgical management, a patient must have a serum calcium that is only mildly elevated, no previous episodes of life-threatening hypercalcemia, and normal renal and bone status.

**Indications for Surgical Treatment**

Conversely, some asymptomatic patients will have objective manifestations of primary HPT that are indications for surgery:

- Markedly elevated serum calcium.
- History of an episode of life-threatening hypercalcemia.
- Reduced creatinine clearance.
- Presence of kidney stone(s) detected by abdominal radiograph.
- Markedly elevated 24-hour urinary calcium excretion.
- Substantially reduced bone mass as determined by direct measurement.

The mean bone density often is below normal in patients with primary HPT. This diminished bone mass is most consistently observed at sites
of cortical bone. Sparse long-term data are available regarding bone loss in asymptomatic primary HPT patients. Furthermore, no published data or study presented to the conference had the requisite power, in terms of numbers of patients or duration of followup, to compare fracture rates in patients with asymptomatic primary HPT to normals. Because low bone mass in postmenopausal women is associated with increased risk of fracture, we assumed that this relationship is likely to be valid in patients with primary HPT, although this assumption remains to be established.

The data were not sufficient to justify precise quantitative recommendations for surgery for any of the above listed tests. Nevertheless, panel members felt some examples should be offered as possible guidelines. The values mentioned below are ones that panel members perceived as warranting operation. It is clear, however, that many physicians will recommend for less prominent elevations that we cite below. Examples of values on which there was consensus regarding need for operation include serum calcium elevations 1 to 1.6 mg/dL (0.25 to 0.4 mmol/L) above the accepted normal range, i.e., 11.4 to 12 mg/dL (2.85 to 3.0 mmol/L), given a normal range of 8.8 to 10.4 mg/dL (2.2 to 2.6 mmol/L); creatinine clearance reduced by 30 percent compared with age-matched normals; confirmed 24-hour urine calcium excretion >400 mg; and bone mass more than two standard deviations below age-, gender-, and race-matched controls.

In addition, surgery is indicated in those patients in whom medical surveillance is neither desirable nor suitable:

- Patient requests surgery.
- Consistent followup is unlikely.
- Coexistent illness complicates management.
- Patient is young (<50 years old).

Surgery is recommended for younger patients because the outcome of several decades of primary HPT is not known. In addition, for such patients, long-term compliance may be inadequate to ensure a safe outcome, and the cumulative expense and time invested in rigorous monitoring greatly outweigh the expense and time of an operation. Care should be taken to avoid surgery in FHH; in such patients, surgery is inappropriate.

Despite this outline for management, some patients will decline recommended surgery. They should be followed at least as intensively as uncomplicated asymptomatic patients in the manner described in the next section.
If Not Operated on, How Should Asymptomatic Patients Be Monitored and Managed?

Monitoring Procedures

When it is decided to follow a patient with asymptomatic hyperparathyroidism, that patient must understand that a decision to forgo parathyroid surgery is considered safe only if the patient and the physician remain committed to conscientious long-term monitoring. The goals of such followup include the early recognition of worsening hypercalcemia, the deterioration of bone, renal impairment, or the appearance or growth of renal stones.

The patient should be seen at least semiannually until the lack of progression of the disease has been established. Once stability of the various parameters has been established over 1 to 3 years, the intervals between these various observations can be safely extended. The patient should be specifically queried regarding neuromuscular weakness, depression, and symptoms related to the skeletal, gastrointestinal, and renal systems, and the following determinations are recommended at each visit:

- Blood pressure.
- Serum calcium.
- Serum creatinine and creatinine clearance.

In addition, we recommend the following:

- Abdominal radiographs annually.
- 24-hour urinary calcium in selected patients.
- Repeat bone mass measurement after 1 to 2 years.

The panel suggests that a second determination of bone mass be sought after an interval of time that is adequate to assess whether there has been significant loss of bone. The appropriate interval will depend on the precision of the instrument available. It is acknowledged that there is inadequate information identifying the ideal methodology for monitoring changes in bone mass in patients with asymptomatic HPT. There is some indication that measurement of the forearm bone density with single photon absorptiometry may be useful to monitor changes in bone density. The development of alternative densitometric methodology such as dual energy x-ray absorptiometry may soon become preferable to identify subtle changes in cortical bone. Although a recommendation for a change to surgical therapy can be made solely on the basis of an abnormally low value of bone density, such a decision remains
Management During Surveillance

There are certain aspects of management that should be advised for all patients being followed; the patients should avoid dehydration, immobilization, and a diet with restricted or excess calcium. Loop or thiazide diuretics should be used with caution. Because of the risk of a hypercalcemic crisis, patients should be advised to seek immediate medical care with the appearance of a medical illness that may produce dehydration (e.g., vomiting, diarrhea, etc.). There should be adequate treatment of hypertension, even when it is mild.

Many physicians prescribe estrogen therapy for postmenopausal women, because of the beneficial actions of estrogens on postmenopausal osteoporosis and cardiovascular risk factors. While there is evidence that estrogen therapy can reduce the action of PTH on bone and lower serum calcium without causing PTH levels to rise, there are limited data on long-term therapy with estrogens in postmenopausal patients with asymptomatic hyperparathyroidism.

Other drugs such as the bisphosphonates, oral phosphate, calcitonin, or mithramycin, which modify the PTH-induced stimulation of bone resorption, are not presently indicated in patients with asymptomatic HPT. However, bisphosphonates or oral phosphate may be considered in the rare patient with symptomatic hyperparathyroidism who is not a surgical candidate because of severe concurrent diseases.

What Are the Indications for Surgery in Patients With Asymptomatic Hyperparathyroidism?

During monitoring of asymptomatic patients, the following developments may warrant consideration for operative intervention:

- Typical parathyroid-related symptoms involving skeletal, renal, or gastrointestinal systems.
- Sustained increase in serum calcium of greater than 1.0 to 1.6 mg/dL (0.25-0.4 mmol/L) above the normal range.
- Significant decline in renal function.
- Nephrolithiasis or worsening calciuria.
- Significant decline in bone mass.
- Significant neuromuscular or psychologic symptoms without other obvious cause.
- The inability or unwillingness of the patient to continue under medical supervision.
In addition to the absolute level of serum calcium, clinicians need to take into account the magnitude of the changes over time.

Assessment of renal function should be made by measurement of creatinine clearance. Although the panel could not define precise values, a confirmed decrease of more than 30 percent was considered significant.

The significance of declining bone mass is controversial and a decrease to two standard deviations below the mean for age-, sex-, and race-matched controls was considered sufficient to warrant operation, as already discussed.

The relationship of psychologic symptoms to hyperparathyroidism is uncertain. All investigators have suggested that neuromuscular symptoms are frequent and often reversed by successful parathyroidectomy while other less specific somatic symptoms are rarely improved by operation.

What Is the Role of Gland Localization Technology in Management of Patients With Hyperparathyroidism?

Imaging of the parathyroid glands before an initial neck exploration is not necessary.

In the past 4 years, extensive experience has been acquired in nonoperative methods for localization of abnormal parathyroid glands. Both noninvasive methods (ultrasound, computed tomography, thallium-technetium scanning, magnetic resonance imaging) and invasive methods (arteriography, venous sampling, needle aspiration) are available. Such methods may be useful when a previous operation has failed. However, because of their potential risks, invasive imaging techniques should never be employed before a first neck exploration.

The use of noninvasive imaging procedures before a first operation is controversial. Some surgeons never use these techniques, while others find them helpful in planning the sequence of an operation. The usefulness of all noninvasive imaging methods is diminished by their unreliability (about 15 percent false positives and only 60 percent true positives). By comparison, operative exploration of the neck by experienced surgeons has a demonstrated success rate of 95 percent. There is no evidence that preoperative imaging can significantly improve surgical therapy by: (1) shortening the time of operation or decreasing its cost, (2) decreasing complications of an operation, or (3) preventing failed operations. The results of imaging studies should seldom, if ever, be used as the basis of selecting patients for operative or nonoperative
What Research Should Be Done To Clarify Issues in Diagnosis and Management of Hyperparathyroidism?

Ultimately, the ability to predict outcomes in asymptomatic HPT and decide on operative versus nonoperative management will require a multicenter, randomized, controlled trial of sufficient size and duration to assess the long-term incidence and progression of complications. However, many specific issues were identified during the conference that need to be resolved before such a trial can be designed. In particular, it is important to define the neuromuscular, psychological, cardiovascular, and gastrointestinal effects of primary HPT. The effects of asymptomatic HPT on bone mass and structure are being defined in ongoing studies, but effects on bone strength and susceptibility to fracture also should be addressed.

The case-control method might be a feasible initial approach. Objective analysis could be carried out in patients with HPT before and after surgery and in carefully matched controls undergoing other elective surgical procedures such as thyroidectomy. Epidemiologic studies using existing databases such as the National Health and Nutrition Examination Survey might identify conditions associated with primary HPT, because the majority of subjects identified with hypercalcemia would have HPT. In addition, it may be desirable to organize and collect available data on fracture, change in bone mass and histomorphometry, and other complications from patients currently being followed in specialized centers.

A preliminary clinical trial comparing bone mass outcomes and biochemical measures of bone turnover in postmenopausal women with HPT randomly assigned to estrogen plus surgery versus estrogen alone for a limited time might establish feasibility and provide guidance for the design of a large multicenter trial. Such a trial would permit analysis of the effects of HPT without the confounding effects of estrogen deficiency yet provide access to the patient population with the highest incidence of HPT.

Basic studies are needed on the etiology of hyperparathyroidism and its molecular and cellular pathophysiology. Identification of the gene locus on the long arm of chromosome 11 for the MEN I gene, discovery of reciprocal translocations in parathyroid adenomas involving the parathyroid hormone gene, and evidence for deletions of one or both copies of the MEN I gene in hyperplasia and adenomas (suggesting the MEN I gene is an antioncogene) all provide an exciting opportunity to explore abnormal parathyroid function. Insight into pathogenesis and
complications also could be achieved by developing animal models of hyperparathyroidism.

Recent clinical studies using calcitriol and its analogs to treat secondary hyperparathyroidism suggest that parathyroid gland function may be controlled pharmacologically. Development of antagonists of PTH synthesis, secretion, and end organ effect would substantially increase therapeutic options for HPT and influence the design of clinical trials.

Conclusions and Recommendations

- The diagnosis of primary HPT is established by demonstrating persistent hypercalcemia together with an elevated serum parathyroid hormone concentration.
- Current and acceptable treatment following the diagnosis of primary HPT is operative intervention for cure.
- The diagnosis of primary HPT in the asymptomatic patient, however, does not in all cases mandate referral for imminent operative intervention; conscientious surveillance may be justified in patients whose calcium levels are only mildly elevated and whose renal and bone status are close to normal.
- During the long-term medical and nonoperative followup of these patients, a schedule of monitoring has been devised with assessment of specific symptoms, biochemical parameters, and measurement of bone mineral content. Management guidelines are devised to minimize the risk of deterioration of renal, skeletal, or gastrointestinal complications of HPT.
- Changes that may warrant operative intervention during monitoring include rising serum calcium, deterioration of renal function, decline in bone mass, and onset of parathyroid-related symptoms.
- Preoperative localization in patients without prior neck operation is rarely indicated and not proven to be cost effective.
- A randomized multicenter clinical trial is needed to compare operative versus nonoperative management of asymptomatic HPT. Pilot studies would be useful to define the multisystem effects of HPT. Further basic research is required to understand the pathogenesis and develop pharmacologic therapy for HPT.

Consensus Development Panel John T. Potts, Jr., M.D.
Panel and Conference Chairperson
Physician-in-Chief
Massachusetts General Hospital
Jackson Professor of Clinical Medicine
Harvard Medical School
Boston, Massachusetts Irving Paul Ackerman, M.D.
Internist, Endocrinologist
Southern California Permanente Medical Group
Los Angeles, California Clyde F. Barker, M.D.
Professor and Chairman
Department of Surgery
University of Pennsylvania Medical Center
Philadelphia, Pennsylvania Murray F. Brennan, M.D.
Chairman
Department of Surgery
Memorial Sloan-Kettering Cancer Center
New York, New York Jack W. Coburn, M.D.
Nephrology Section
West Los Angeles Veterans Administration Medical Center
Adjunct Professor of Medicine
UCLA School of Medicine
Los Angeles, California Siu L. Hui, Ph.D.
Professor of Medicine
Director
Division of Biostatistics
Indiana University School of Medicine
Indianapolis, Indiana G. Leland Melson, M.D.
Professor of Radiology
Chief of Diagnostic Ultrasound
Mallinckrodt Institute of Radiology
Washington University School of Medicine
St. Louis, Missouri Lawrence G. Raisz, M.D.
Professor of Medicine
Head
Division of Endocrinology and Metabolism
University of Connecticut Health Center
Farmington, Connecticut Michael Rosenblatt, M.D.
Senior Vice President for Research
Merck Sharp & Dohme Research Laboratories
West Point, Pennsylvania Clinton T. Rubin, Ph.D.
Associate Professor; Director
The Musculo-Skeletal Research Laboratory
Department of Orthopaedics
State University of New York
Stony Brook, New York Janet A. Schlechte, M.D.
Associate Professor
Director
Clinical Research Center
University of Iowa College of Medicine
Iowa City, Iowa John L. Townsend, M.D.
Professor of Medicine
Chief of Endocrinology and Metabolism
Department of Medicine
Howard University College of Medicine
Washington, D.C. Lawrence K. Wolfe, M.D.
Private Practice
Internal Medicine and Endocrinology
Associate Clinical Professor of Medicine
Vanderbilt University School of Medicine
Nashville, Tennessee Glenda L. Wong, Ph.D.
Associate Professor of Biology
University of Colorado
Colorado Springs, Colorado Speakers John P. Bilezikian, M.D.
"Characterization and Evaluation of Asymptomatic Primary Hyperparathyroidism"
Chief
Division of Endocrinology
Professor of Medicine
College of Physicians and Surgeons
Columbia University
New York, New York Orlo H. Clark, M.D.
"Diagnosis and Management of Asymptomatic Hyperparathyroidism: Safety, Efficacy, and Deficiencies in Our Knowledge"
Professor
Department of Surgery
Mount Zion Medical Center
University of California, San Francisco
"Sequential Studies of Bone Mass in Patients With Untreated Hyperparathyroidism"
Department of Endocrinology
The Royal North Shore Hospital
St. Leonards, Sydney
AUSTRALIA John L. Doppman, M.D.
"The Role of Localization Studies Prior to Initial Surgery for Hyperparathyroidism"
Director
Department of Diagnostic Radiology
Clinical Center
National Institutes of Health
Bethesda, Maryland David A. Heath, M.B., Ch.B., F.R.C.P.
"Longitudinal Studies on Untreated Primary Hyperparathyroidism"
Reader in Medicine
University of Birmingham
Department of Medicine
Queen Elizabeth Hospital
Edgbaston, Birmingham
ENGLAND Hunter Heath III, M.D.
"The Clinical Spectrum of Primary Hyperparathyroidism"
Consultant in Endocrine Research
Professor of Medicine
Mayo Clinic
Mayo Medical School
Rochester, Minnesota Jack H. Ladenson, Ph.D.
"Blood Calcium Determination in Primary Hyperparathyroidism"
Professor
Department of Pathology and Clinical Chemistry in Medicine
Washington University School of Medicine
St. Louis, Missouri Frederic W. Lafferty, M.D.
"Differential Diagnosis of Hypercalcemia"
Clinical Professor of Medicine
University Hospitals of Cleveland
Cleveland, Ohio Sverker Ljunghall, M.D., Ph.D.
"Longitudinal Studies of Mild Primary Hyperparathyroidism"
Professor and Chairman
Department of Internal Medicine
University Hospital
Uppsala
SWEDEN Robert Marcus, M.D.
"Estrogens and Progestins in the Management of Primary Hyperparathyroidism"
Professor of Medicine
Stanford University
Director
Aging Study Unit
Veterans Administration Medical Center
Palo Alto, California Stephen J. Marx, M.D.
"Etiology of Parathyroid Gland Dysfunction in Primary Hyperparathyroidism: Insights from Approaches as Diverse as Epidemiology and Molecular Biology"
Chief
Mineral Metabolism Section
Metabolic Diseases Branch
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland L. Joseph Melton III, M.D.
"Epidemiology"
Professor of Epidemiology
Mayo Clinic and Foundation
Rochester, Minnesota Robert M. Neer, M.D.
"Natural History of Asymptomatic Primary Hyperparathyroidism"
Associate Professor of Medicine
Harvard Medical School
Samuel R. Nussbaum, M.D.
"Sensitive Immunometric Assays for Parathyroid Hormone and Their Impact on the Diagnosis and Management of Hyperparathyroidism"

Susan M. Ott, M.D.
"Methods of Determining Bone Mass"

A. Michael Parfitt, M.B., B.Chir.
"Asymptomatic Primary Hyperparathyroidism Discovered by Multichannel Biochemical Screening. Clinical Course and Considerations Bearing on the Need for Surgical Intervention"

Munro Peacock, M.D.
"Interpretation of Bone Mass Determinations"

Don C. Purnell, M.D.
"Prospective Study of Asymptomatic Primary Hyperparathyroidism"

Elizabeth Shane, M.D.
"Medical Management of Asymptomatic Primary Hyperparathyroidism"

Allen M. Spiegel, M.D.
"Pathophysiology of Primary Hyperparathyroidism"

Samuel A. Wells, Jr., M.D.
"Surgical Therapy: Long-Term Benefits"
Chairman
Department of Surgery
Washington University School of Medicine
St. Louis, Missouri Planning Committee Judith E. Fradkin, M.D.
Planning Committee Chairperson
Chief
Endocrine and Metabolic Diseases Programs Branch
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland Gerald D. Aurbach, M.D.
Chief
Metabolic Diseases Branch
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland Linda Blankenbaker
Program Analyst
Office of Medical Applications of Research
National Institutes of Health
Bethesda, Maryland Benjamin T. Burton, Ph.D.
Associate Director for Disease Prevention and Technology Transfer
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland Joan Chamberlain
Deputy Director
Information Office
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland Jane Demouy
Technical Writer/Editor
Information Office
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland John H. Ferguson, M.D.
Director
Office of Medical Applications of Research
National Institutes of Health
Bethesda, Maryland Willis R. Foster, M.D.
Senior Staff Physician
Office of Disease Prevention and Technology Transfer
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland William H. Hall
Director of Communications
Office of Medical Applications of Research
National Institutes of Health
Bethesda, Maryland Hunter Heath III, M.D.
Consultant in Endocrine Research
Professor of Medicine
Mayo Clinic
Mayo Medical School
Rochester, Minnesota Stephen J. Marx, M.D.
Chief
Mineral Metabolism Section
Metabolic Diseases Branch
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland Susan M. Ott, M.D.
Associate Professor
Department of Medicine
University of Washington
Seattle, Washington John T. Potts, Jr., M.D.
Panel and Conference Chairperson
Physician-in-Chief
Massachusetts General Hospital
Jackson Professor of Clinical Medicine
Harvard Medical School
Boston, Massachusetts Samuel A. Wells, Jr., M.D.
Chairman
Department of Surgery
Washington University School of Medicine
St. Louis, Missouri Conference Sponsors National Institute of Diabetes and Digestive and Kidney Diseases
Phillip Gorden, M.D.
Director NIH Office of Medical Applications of Research
John H. Ferguson, M.D.
Director