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IMPOTENCE AND ITS MEDICAL AND PSYCHOSOCIAL CORRELATES: RESULTS OF THE MASSACHUSETTS MALE AGING STUDY

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ABSTRACT

We provide current, normative data on the prevalence of impotence, and its physiological and psychosocial correlates in a general population using results from the Massachusetts Male Aging Study. The Massachusetts Male Aging Study was a community based, random sample observational survey of noninstitutionalized men 40 to 70 years old conducted from 1987 to 1989 in cities and towns near Boston, Massachusetts. Blood samples, physiological measures, socio-demographic variables, psychological indexes, and information on health status, medications, smoking and lifestyle were collected by trained interviewers in the subject's home. A self-administered sexual activity questionnaire was used to characterize erectile potency.

The combined prevalence of minimal, moderate and complete impotence was 52%. The prevalence of complete impotence tripled from 5 to 15% between subject ages 40 and 70 years. Subject age was the variable most strongly associated with impotence. After adjustment for age, a higher probability of impotence was directly correlated with heart disease, hypertension, diabetes, associated medications, and indexes of anger and depression, and inversely correlated with serum dehydroepiandrosterone, high density lipoprotein cholesterol and an index of dominant personality. Cigarette smoking was associated with a greater probability of complete impotence in men with heart disease and hypertension.

We conclude that impotence is a major health concern in light of the high prevalence, is strongly associated with age, has multiple determinants, including some risk factors for vascular disease, and may be due partly to modifiable para-aging phenomena.

KEY WORDS: impotence, penile erection, elderly, aging

Impotence has been defined as the persistent inability to attain and maintain an erection adequate to permit satisfactory sexual performance.[1] Although regarded as a benign disorder, impotence has a profound impact on the quality of life of many men.[2] In the United States in 1985, impotence accounted for 400,000 outpatient visits to physicians and 30,000 hospital admissions, resulting in total direct costs of $146 million.[3]

Despite the prevalence, costs and implications of impotence for quality of life, current normative data on impotence in a healthy population are seriously lacking, particularly in the context of physiological and psychosocial variables.[4] The Kinsey survey, conducted more than 40 years ago in a social and medical context vastly different from that of the present day, remains the most extensive population-based source of normative data on male sexual behavior in the United States.[5] Subsequent studies have been restricted in scope.[6-15] No comprehensive new data from a healthy population exist to provide context for the considerable advances of the last decade in our understanding of the physiological mechanisms of erectile function and dysfunction.[2, 16, 17]

Impotence is commonly associated with aging. Putative causes and clinical correlates of impotence, many of them likewise associated with aging, include vascular insufficiency, hormonal derangement, interruption of neural pathways, diabetes, psychogenic factors and side effects of therapeutic drugs.[2] It has been argued that impotence, like cardiovascular disease and other age-related disorders, can be attributed at least partly to such modifiable para-aging phenomena.[18]

The Massachusetts Male Aging Study (MMAS), conducted from 1987 to 1989, was a community based, multidisciplinary survey of health and aging in men, including a private, self-administered questionnaire on sexual function and activity. We used MMAS data to measure the prevalence of impotence in a normal population of healthy, aging men, to describe the age trend of impotence in this population, to determine whether age-related health indexes account for the age trend in impotence and to identify the variables associated with impotence over and above the age trend.

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METHODS

Study sample. The study sample consisted of respondents to the MMAS, a cross sectional, random sample survey of health status and related variables in men 40 to 70 years old. The MMAS was conducted from 1987 to 1989 in 11 randomly selected cities and towns in the area of Boston, Massachusetts. The design of the MMAS has been described previously.[18-23] The MMAS sexual activity questionnaire included 9 items related to potency (see Appendix) to which 1,290 of the 1,709 MMAS subjects (75%) provided complete responses. These 1,290 men constitute the present study sample (table 1).

The 419 excluded men did not differ from the study sample with respect to body mass index, serum cholesterol, smoking habits, physical activity, prevalence of heart disease, hypertension, medication use as listed in table 1, ethnic distribution, religion or membership in social groups. In 291 cases the reason for exclusion was lack of a current sexual partner and consequent nonresponse to items 7 to 9 (see Appendix). The exclusion of men with no partner should bias the estimated prevalence of impotence downward if at all, since men without partners are likely to have a greater than normal rate of impotence. The low percentage of racial minority subjects in

TABLE 1. *Physical, medical and socio-demographic characteristics of 1,290 MMAS subjects included in study of impotence*

Physical measures:*	
Age (yrs.)	53.8 ± 8.5
Ht. (inches)	69.1 ± 3.0
Wt. (pounds)	186.7 ± 31.5
Body mass index (kg./m.^{2})	27.5 ± 4.4
Serum cholesterol (mg./dl.)	209 ± 49
Physical activity (kcal./kg./day)	48.2 ± 15.2
Cigarette smoking:	
Current smokers†	286 (22)
Cigarettes/day (smokers only)*	25.0 ± 15.1
Passive smoking at work†	475 (38)
Passive smoking at home†	366 (28)
Medical conditions:†	
Diabetes:	
Not treated	36 (3)
Treated	52 (4)
Heart disease:	
Not treated	65 (5)
Treated	90 (7)
Hypertension:	
Not treated	176 (14)
Treated	200 (16)
Arthritis:	
Not treated	228 (18)
Treated	76 (6)
Allergy:	
Not treated	261 (20)
Treated	67 (5)
Ulcer:	
Not treated	98 (8)
Treated	31 (2)
Medications:†	
Cardiac	112 (9)
Antihypertensive	140 (11)
Lipid-lowering	21 (2)
Hypoglycemic agents	49 (4)
Vasodilator	53 (4)
Sympathetic	28 (2)
Socio-demographics:†	
Married	1,082 (84)
Have sexual partner‡	1,290 (100)
Living alone	112 (9)
Employed	1,061 (82)
Depressed	120 (9)
Belonging to social groups	706 (55)
Emotional support available	1,196 (93)
Race:†	
White	1,240 (96)
Black	28 (2)
Other	22 (2)
Education:	
Below high school	125 (10)
Completed high school	205 (16)
Beyond high school	377 (29)
Bachelor's degree	174 (13)
Graduate study or degree	409 (32)

* Mean ± standard deviation.
† Number of patients (%).
‡ Criterion for inclusion in impotence substudy.

the sample (4%) was consistent with the composition of the Massachusetts population.

Data collection. A trained technician visited each subject in his home, and completed the interview, measurements and blood sampling within 4 hours of his awakening. Informed consent was obtained at the interview on a written form approved by an institutional review board.

Health status was ascertained by asking the subject to categorize a list of diseases as absent, treated or untreated. Height, weight and blood pressure were measured by standardized methods developed for large-scale field work.[24] Prescription and nonprescription medications were gathered and inventoried by the interviewer. Two blood samples were drawn 30 minutes apart, and pooled for analysis of lipids and sex steroid hormones.[20] The psychological instruments included measures of dominance,[25] anger[26] and depression.[27] The sexual activity questionnaire was completed in private and returned to the interviewer.[22]

Calibration study. The MMAS instrument was linked to a direct assessment of potency by means of a separate calibration study.[28] Men presenting at our university medical center urology clinic during a 6-month period in 1990 completed a questionnaire consisting of the 9 questions listed in the Appendix and an additional question asking the respondent to characterize himself as not impotent, minimally impotent, moderately impotent or completely impotent. Table 2 shows that self-rated impotence was reflected in MMAS questions as higher frequency of erectile difficulty during intercourse, lower monthly rates of sexual activity and erection, and lower satisfaction with sex life and partner. Accordingly, a quadratic discriminant formula, combining the 9 MMAS sexual activity responses to produce an estimate of the probability of each subject having nil, minimal, moderate or complete impotence, was constructed from the calibration data by standard methods.[29,30] In a cross-validation procedure, virtually all of the calibration subjects (293 of 303 or 97%) were reclassified into the correct or an adjacent category. The discriminant formula was applied to MMAS data (when a direct self-rating of potency was not available) to produce a set of probabilities, $p_1\ p_2\ p_3\ p_4$, describing for each MMAS subject the respective likelihood of nil, minimal, moderate or complete impotence.

Statistical analysis. Simple comparisons between the MMAS subjects included in our study and those excluded because of incomplete response were made by the t or chi-square test as appropriate. To relate impotence to subject age, health status and other candidate predictor variables, multivariate linear regression and multivariate analysis of variance were used with vector $p_1\ p_2\ p_3\ p_4$ as the dependent variable. Subject age was included as an obligatory covariate in every analysis so that each independent variable was evaluated for its ability to account for age effects, and its predictive power over and above that of age. Multivariate linear regression was used to assess the dependence of $p_1\ p_2\ p_3\ p_4$ on continuous independent variables, such as serum cholesterol. The multivariate null hypothesis (no association with any degree of impotence) was tested with a single F statistic (degrees of freedom 3, infinity), followed by examination of the univariate regressions of p_1, p_2, p_3, p_4 when a significant multivariate result was obtained. Multivariate analysis of variance was similarly used to compare $p_1\ p_2\ p_3\ p_4$ across categories of disease status, smoking behavior and medication use. Statistical Analysis System procedures for the general linear model were used for multivariate linear regression and multivariate analysis of variance.[30] When statistical tests indicated interaction between the effects of 2 predictors, pre-planned followup comparisons were made between appropriate adjusted subgroup means. All differences noted in the presentation of results were statistically significant at $p < 0.01$.

RESULTS

Prevalence. Summary statistics on the prevalence of nil, minimal, moderate and complete impotence are shown in table 3. In the entire sample of 1,290 men 40 to 70 years old the mean probability of some degree of impotence was 52.0 ± 1.3% (standard error). The largest category was moderate impotence with a 25.2% prevalence, the next largest was minimal impotence at 17.2% and the smallest was complete impotence at 9.6%.

Age dependence. The relationship of impotence probabilities to the age of MMAS subjects is illustrated in figure 1. Between subject ages of 40 and 70 years the probability of complete impotence tripled from 5.1 to 15%, while the probability of moderate impotence doubled from 17 to 34%. Within the same age range the probability of minimal impotence remained constant at approximately 17%. An estimated 60% of the men were not impotent at age 40 years, with a decrease to 33% not impotent at age 70 years.

Whenever age was tested by multivariate linear regression or multivariate analysis of variance in conjunction with another

TABLE 2. *Self-rated impotence in calibration sample (303 subjects) related to sexual activity questions from MMAS*

	Not Impotent	Minimally Impotent	Moderately Impotent	Completely Impotent
No. subjects	116	41	92	54
Sexual activity (median frequency/mo.)	8	7	3	0
Full erection (median frequency/mo.)	30	30	4	0
Awaken with erection (median frequency/mo.)	10	10	3	0
No activity within last 6 mos. (%)	2	7	20	61
Trouble getting erection (%)*	5	50	85	90
Trouble keeping erection (%)*	5	63	95	96
Satisfaction with frequency of activity (%)	65	46	15	17
Satisfaction with sex life†	1.6	3.0	4.1	4.6
Satisfaction with partner†	1.3	2.2	3.2	3.7
Partner satisfaction†	1.5	2.6	3.5	4.1

* Among those reporting some sexual activity within last 6 months.
† Mean on scale from 1 (extremely satisfied) through 5 (extremely dissatisfied).

TABLE 3. *Prevalence of impotence in MMAS*

Degree of Impotence	Prevalence (%)*
Not impotent	48.0 ± 1.3
Impotent:	52.0 ± 1.3
Minimally	17.2 ± 0.8
Moderately	25.2 ± 0.9
Completely	9.6 ± 0.7

* 100 × mean probability ± standard error, 1,290 subjects.

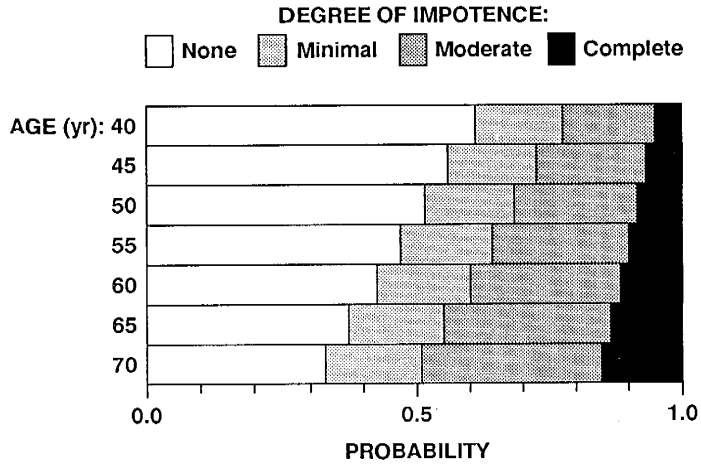

FIG. 1. Association of subject age with probability of impotence imputed by discriminant analysis in 1,290 respondents to sexual activity questionnaire of MMAS.

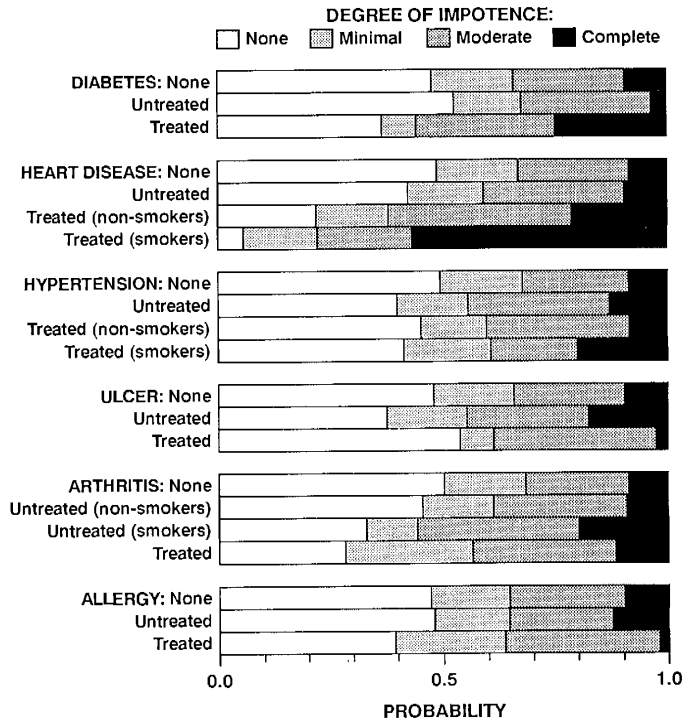

FIG. 2. Association of self-reported disease and treatment with age-adjusted probability of impotence imputed by discriminant analysis in 1,290 respondents to sexual activity questionnaire of MMAS.

predictor of impotence, age invariably proved to be statistically significant at $p < 0.0001$. No other variable, whether correlated with age or not, diminished the predictive power of simple age.

Disease. Certain treated medical conditions, including diabetes, heart disease and hypertension, were significantly associated with changes in the impotence probability pattern after adjustment for subject age (fig. 2). The age-adjusted probability of complete impotence was 28% in those with treated diabetes, 39% in those with treated heart disease and 15% in those with treated hypertension, compared with 9.6% in the entire sample. Untreated disease was associated with impotence in 3 other conditions: untreated ulcer (18%), untreated arthritis (15%) and untreated allergy (12%).

Lipids. The probability of impotence varied inversely with high density lipoprotein cholesterol. For the younger men (age 40 to 55 years), the age-adjusted probability of moderate impotence increased from 6.7 to 25% as high density lipoprotein cholesterol decreased from 90 to 30 mg./dl. (fig. 3). In the older men (age 56 to 70 years), the probability of complete impotence increased from near zero to 16% as high density lipoprotein cholesterol decreased from 90 to 30 mg./dl. Total serum cholesterol was not correlated with impotence probabilities.

Hormones. Of the 17 hormones measured in MMAS subjects, only the adrenal androgen metabolite dehydroepiandrosterone sulfate showed a strong correlation with impotence (fig. 3). The age-adjusted probability of complete impotence increased from 3.4 to 16% as dehydroepiandrosterone sulfate decreased from 10 to 0.5 μg./ml. Dihydrotestosterone and cortisol showed effects of small magnitude on minimal impotence only. No correlation with impotence was found for testosterone (either free, albumin-bound or total), sex hormone binding globulin (the major serum carrier of testosterone), various other androgens and metabolites (including androstenedione and androstanediol), estrogens, prolactin or the pituitary gonadotropins (follicle-stimulating hormone and luteinizing hormone).

Medications. Complete impotence was significantly more prevalent in men taking certain medications, including hypoglycemic agents (26%), antihypertensives (14%), vasodilators (36%) and cardiac drugs (28%), than in the sample as a whole (9.6%, fig. 4). The probability pattern in users of hypoglycemic agents closely resembled that in subjects reporting treated diabetes. Likewise, men taking cardiac drugs or antihypertensives showed a pattern of impotence probabilities similar to that in men reporting treated heart disease or hypertension. The probability of moderate (42%) as well as complete (36%) impotence was particularly great for men taking vasodilators.

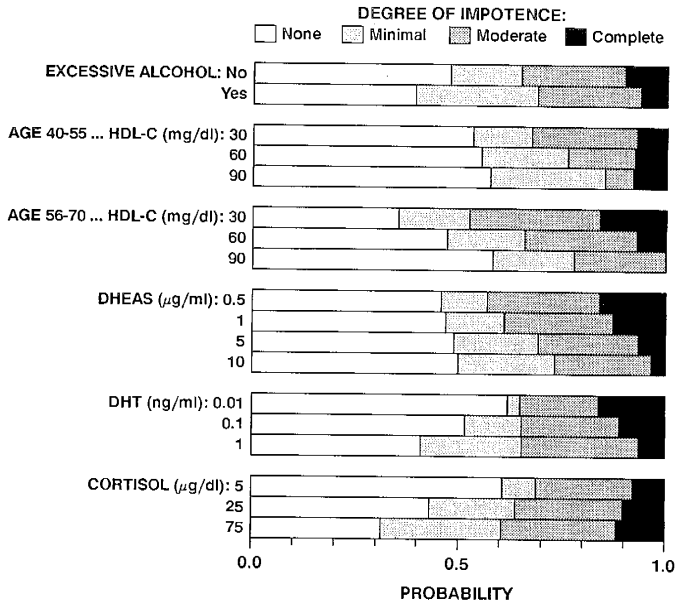

FIG. 3. Association of excessive alcohol consumption, high density lipoprotein cholesterol (*HDL-C*) and serum hormone levels with probability of impotence imputed by discriminant analysis in 1,290 respondents to sexual activity questionnaire of MMAS. Probabilities for high density lipoprotein cholesterol are stratified by age and other probabilities are age-adjusted by regression. *DHEAS*, dehydroepiandrosterone sulfate. *DHT*, dihydrotestosterone.

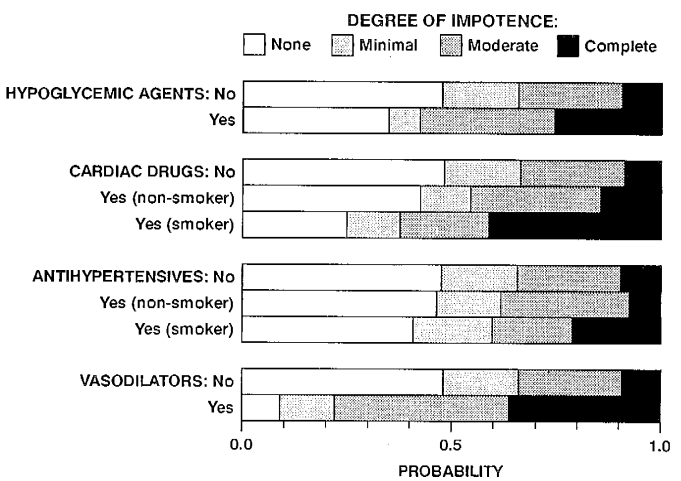

FIG. 4. Association of medications with age-adjusted probability of impotence imputed by discriminant analysis in 1,290 respondents to sexual activity questionnaire of MMAS.

No correlation was found between impotence and the use of lipid-lowering or sympathetic drugs.

Cigarette smoking. The association of impotence with certain risk factors was greatly amplified in current cigarette smokers. In subjects with treated heart disease the age-adjusted probability of complete impotence was 56% for current smokers, compared with 21% for current nonsmokers. Among treated hypertensives those who currently smoked cigarettes had an elevated probability of complete impotence (20%), whereas the nonsmokers (8.5%) were comparable to the general sample (9.6%). The current smokers with untreated arthritis had a significantly greater probability of complete impotence (20%) than the nonsmokers with that condition (9.4%).

Drug effects were also exacerbated by current smoking. Current cigarette smoking increased the age-adjusted probability of complete impotence in those taking cardiac drugs (from 14 to 41%), antihypertensive medications (from 7.5 to 21%) and vasodilators (from 21 to 52%).

A general effect of current cigarette smoking was not noted. The probability of complete impotence was 11% in smokers and 9.3% in nonsmokers ($p > 0.20$). Among current smokers, impotence probabilities showed no dependence on dosage (packs per day) or lifetime pack-years smoked. No significant difference was noted between subjects who reported exposure to cigarette smoke at home or at work and those who reported none.

Life-style. Probabilities of impotence were not dependent on body mass index (weight divided by height squared) or waist-to-hip ratio, an index of fat localization. The impotence probability pattern did not differ significantly between subjects who were greater than 120% of ideal weight[31] and those who were not. Excessive alcohol consumption (more than 600 ml. per week) was associated with increased probability of minimal impotence from 17 to 29% (fig. 3). A quantitative estimate of daily alcohol consumption was similarly correlated with minimal impotence, as was a measure of blood alcohol content based on daily consumption and normalized for body mass.

Psychological indexes. Suppression and expression of anger, as measured by the Spielberger scales, were correlated with higher probabilities of moderate and complete impotence (fig. 5). Men with maximum levels of anger suppression and anger expression showed an age-adjusted probability of 35% for moderate impotence and 16 to 19% for complete impotence, both well above the general level (9.6%).

The Jackson scale of dominance, measuring frequency of attempts to control one's environment and influence others, and the Center for Epidemiologic Studies depression scale, were strongly correlated with impotence (fig. 5). At the maximum level of dominance the age-adjusted probability of moderate impotence was 15% (compared with 25% generally) and the probability of complete impotence was 7.9% (compared with 9.6%). At the maximum degree of depression the combined age-adjusted probability of either moderate or complete impotence was nearly 90%, compared with 59% at mid scale and 25% at the least depressed extreme.

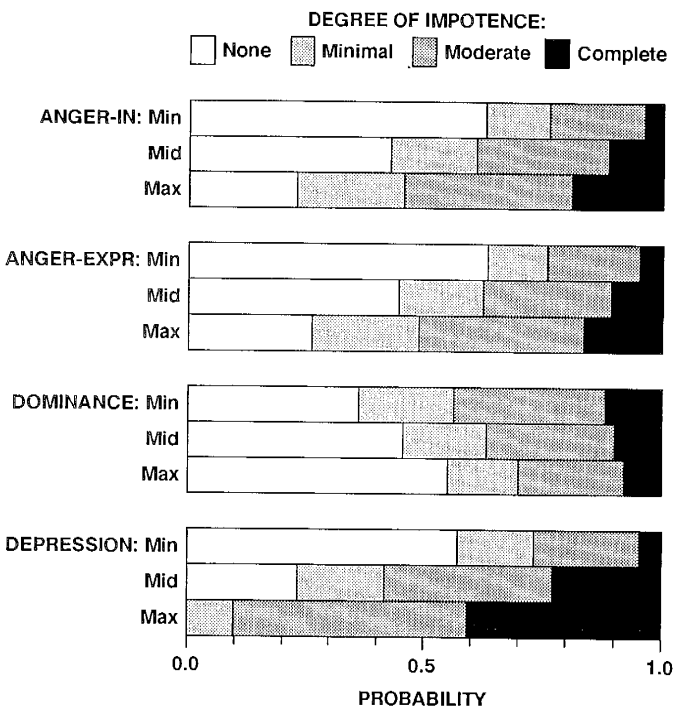

FIG. 5. Association of psychological indexes with age-adjusted probability of impotence imputed by discriminant analysis in 1,290 respondents to sexual activity questionnaire of MMAS. Labels refer to minimum (*Min*), mid range (*Mid*) and maximum (*Max*) values observed in impotence substudy subjects. *ANGER-IN*, suppression of anger. *ANGER-EXPR*, expression of anger.

DISCUSSION

Methodological merits. The MMAS was based on a random sample of noninstitutionalized men with age distributed uniformly between 40 and 70 years, only a fraction of whom were sick and undergoing medical care. By contrast, earlier studies of impotence were limited to men from outpatient clinics,[11] elderly populations[13-16,32] or other selective sources.[6-8] The MMAS response rate (53%) was similar to those achieved in other comparable epidemiological studies involving in-home phlebotomy. The representative nature of the MMAS data was confirmed in a followup study of 400 nonrespondents. The achieved sample size (1,709) was larger than that of any study in the 40 years since the Kinsey et al survey.[5] By making in-home measurements MMAS avoided artifactual biases that commonly result from interaction of the subjects with the medical system. The MMAS inventory of medication was more accurate than is possible in a clinic-based study. The comprehensive MMAS hormone and lipid profile represented basal values controlled for pulsatile emissions and diurnal variation.

The ascribed degree of impotence in this study was based on subject responses to a privately self-administered questionnaire, rather than on a designation by the physician. Potency was addressed as a subjective state,[1] as opposed to the more concretely defined phenomenon of erectile dysfunction. Recent data show that a large fraction of cases of erectile dysfunction are organic in origin and result from hemodynamic, neurological, endocrinological, drug-related or other undefined factors.[2] Potency, defined as satisfactory functional capacity for erection, may coexist with some degree of erectile dysfunction in the sense of submaximal rigidity or submaximal capability to sustain the erection.[33] Therefore, impotence is best defined by the individual's assessment of his own situation in simple terms of minimal, moderate or complete impotence as presented to a physician for treatment.[34] Statistical validity of this approach to categorizing impotence was established by cross-validation of our calibration data.[28]

Prevalence. In the MMAS sample the prevalence of impotence of all degrees was estimated at 52%. Projection of these results to 1990 population data would suggest that impotence affects 18 million American men 40 to 70 years old. Therefore, impotence should be considered as a major health concern.

Published information concerning the prevalence of impotence has been limited in scope and usefulness.[5-17] Among the few reports in the recent literature the criteria for defining impotence have varied, while the methods for obtaining study populations differed and prevalence figures were not stratified by age.[11,16,35] Slag et al found that among 1,180 men attending a medical outpatient clinic 34% reported impotence to their interviewers.[11] Schein et al reported, on the basis of detailed questionnaires, that the presence of impotence in 212 family practice patients with a mean age of 35 years was 27%.[7] Morley determined a prevalence of impotence of 27% in men more than 50 years old undergoing a general health screening.[12] Diokno et al reported a 40% impotence rate in 283 noninstitutionalized men older than 60 years.[16]

Age trend. In the MMAS sample, the probability of complete impotence tripled from 5 to 15% between subject ages of 40 and 70 years. No other variable, whether or not associated with impotence, could account for the effect of the simple age variable when both were tested jointly by regression analysis.

Prior studies have shown similar relationships between impotence and aging. Kinsey et al found impotence to be an age-dependent disorder with a prevalence of 1.9% at age 40 years and 25% at age 65 years.[5] Studies by Pearlman and Kobashi,[36] Frank et al[8] and others in the 1970s have shown impotence-related age dependency. The Baltimore Longitudinal Study of Aging reported that by age 55 years impotence was a problem in 8% of all healthy men, and that for ages 65, 75 and 80 years the prevalence increased to 25%, 55% and 75%, respectively.[37] Pfeiffer and Davis observed in 261 men that age was negatively correlated with current sexual function.[38] In 225 geriatric clinic patients, Mulligan et al found the rate of impotence to be 26% at age 65 years and younger, and 50% at age 75 years and older.[32] Keil et al, studying self-reported sexual function in the Charleston Heart Study Cohort, found the rate of sexual inactivity to be 30% in men aged 60 to 69 years and 60% at age 80 years and older.[13] Heightening the importance of these data is the fact that by the year 2030, given current trends, 20% of the United States population will be more than 65 years old.[39] Moreover, life expectancy for men attaining age 65 years has increased substantially in this century.[40]

Diabetes. Diabetes is a well recognized illness associated with impotence. In the MMAS sample the age-adjusted probability of complete impotence was 3 times greater in subjects reporting treated diabetes than in those without diabetes. Other investigators, primarily using exclusively diabetic populations, have repeatedly found a high prevalence of impotence associated with diabetes, with estimates ranging from 35 to 50% and up to 75%.[41,42] The prevalence of impotence in diabetes has been reported to increase from 15% at age 30 to 34 years to 55% at age 60 years.[43] Onset of impotence is reported to occur at an earlier age in persons with diabetes than in the general population, frequently leading to or following within 10 years the diagnosis of diabetes, whether of the insulin-dependent or noninsulin-dependent type.[44,45]

While researchers disagree as to which of the many aspects of diabetes is the direct cause of impotence, vascular disease is most frequently cited. Zemel reported that 40 to 80% of men with diabetes and hypertension noticed sexual dysfunction but suggested that it was due in part to their medications.[41] Several investigators have found high rates of vascular abnormalities in patients with diabetes[46,47] and impotent patients in particular.[48] Other factors implicated in diabetic impotence include associated autonomic neuropathy and gonadal dysfunction.[44] Saenz de Tejada et al showed that diabetes impairs neurogenic and endothelium mediated relaxation of penile smooth muscle.[49]

Cardiovascular disease. Heart disease and 2 associated risk factors, hypertension and low serum high density lipoprotein, were significantly correlated with impotence in the MMAS sample. It may be inferred that vascular compromise, as reflected in cardiovascular or cerebrovascular disease, shares common determinants with vascular impairment of the erectile mechanism. The association of impotence with vascular disease in the literature is strong. Impairments in the hemodynamics of erection have been demonstrated in patients with myocardial infarction,[34] coronary bypass surgery,[50] cerebrovascular accidents[51] and peripheral vascular disease.[52] These studies were performed primarily in impotent and ill subjects. Oaks and Moyer, for example, reported that 8 to 10% of all untreated hypertensive patients were impotent at diagnosis of hypertension.[53] Wabrek and Burchell reported that among 131 men 31 to 86 years old who were hospitalized for an acute myocardial infarction 64% were impotent.[34] In a study of patients who underwent coronary artery surgery 57% were mostly impotent.[50] Several studies in impotent men reported that the number of abnormal penile vascular findings significantly increased when history included myocardial infarction or vascular risk factors, such as hypertension and cigarette smoking.[54-56]

Other diseases. Blake et al reported impotence among patients with arthritis.[57] In MMAS the association was specific to cigarette smokers. No data exist in the literature concerning impotence in connection with ulcer or allergy, both of which showed small but significant effects in the MMAS sample.

Hormones. The effect of androgens on libido and sexual behavior is well established[58] but their effect on the erectile mechanism and their contribution to impotence remain unclear.[59-61] The value of routine hormone screening remains controversial.[59-64] In MMAS, of 17 hormones measured only

dehydroepiandrosterone sulfate was strongly associated with impotence. Possibly relevant is the reputed predictive value of dehydroepiandrosterone sulfate for cardiovascular disease[65] or the fact that serum dehydroepiandrosterone sulfate concentration decreases more rapidly with age than many other androgens.[19]

Medications. Most studies documenting drug-induced impotence have been subjective, and based on case reports, uncontrolled studies and clinical impressions.[2,66] Morley noted that 16 of the 200 most widely prescribed drugs in the United States have been reported to cause impotence.[12] Slag et al found a 25% prevalence of drug-associated impotence in a medical outpatient population.[11] Whitehead and Klyde reported that hypoglycemia resulting from insulin or oral hypoglycemic agents can cause erectile or orgasmic dysfunction.[44] Male sexual dysfunction has been associated with virtually every available antihypertensive agent, including sympatholytics, β-adrenoceptor blocking agents, vasodilators and diuretics.[67-69]

In the MMAS data, which included a complete in-home medication inventory, impotence was statistically associated with antihypertensive, vasodilator, cardiac and hypoglycemic medications. In this observational setting the drug associations were necessarily confounded with the underlying medical conditions. Wein and van Arsdalen recommended that the diagnosis of drug-induced sexual dysfunction be restricted to a reproducible dose-related effect that disappears on discontinuing the drug.[68] A much larger survey or a controlled study in a clinical population would be required to establish any suspect medication as causative.

Life-style. Cigarette smoking has been shown to be an independent risk factor for vasculogenic impotence.[70] In MMAS subjects current cigarette smoking exacerbated the risk of impotence associated with cardiovascular diseases and medications. The influence on sexual function of other behavior patterns, such as excessive alcohol consumption, obesity and physical inactivity, is unclear.[71,72] We found no correlation between impotence and obesity, and only a slight association with alcohol in MMAS subjects.

Psychological factors. Psychological explanations for impotence, which are common in popular conceptions and in case histories,[73] have a specific physiological basis. While psychogenic stimuli normally facilitate erection, cerebral signals can produce impotence equally well by inhibiting reflex activation of the parasympathetic dilator nerves that enhance inflow of blood to the penis.[2]

In MMAS the psychological factors strongly associated with impotence included depression, low levels of dominance and anger either expressed outward or directed inward. Clinical surveys consistently document decreased interest in sexual activity in 50 to 90% of all depressed individuals.[74] Hostility and expression of anger are associated with many organic diseases, such as peptic ulcer and coronary disease.[75,76] Excessive sympathetic arousal is mediated by chronic anxiety, abnormal personality traits and problems with expression of emotions, particularly anger.[75] Pathways of mutual influence clearly connect these psychological factors with sexual performance. Excessive sympathetic outflow or elevated blood catecholamine levels in an anxious individual may produce vasoconstriction and increase penile smooth muscle tone, opposing the events necessary for erection.[2] Conversely, a man who has experienced a recent pattern of erectile dysfunction may be expected to be anxious, depressed and lacking self-esteem and self-confidence.

Implications. Impotence is a highly prevalent health problem with considerable impact on the quality of life of aging men. Our study produced normative data detailing a strong trend in the prevalence of impotence between ages 40 and 70 years. Several variables, including cigarette smoking and other risk factors for vascular disease, showed statistically significant associations with impotence over and above the age trend. We infer that impotence may be due partly to modifiable para-aging phenomena. Public health policy and clinical management directed at the modifiable factors associated with impotence could result in significant alleviation of a costly and burdensome problem of aging.

Dr. John W. Rowe and Dr. Christopher Longcope contributed to the development and conduct of the Massachusetts Male Aging Study.

APPENDIX

Sexual activity questions related to potency, from self-administered instrument included in MMAS

1. In an average week, how often do you usually have sexual intercourse or activity?
   ☐ (Enter number in box.)
2. During an average 24-hour day, how often do you have a full hard erection?
   ☐ (Enter number in box.)
3. During the last 6 months have you ever had trouble getting an erection before intercourse begins?
   a. No.
   b. Yes.
   c. Have not had sexual intercourse within last 6 months.
4. During the last 6 months have you ever had trouble keeping an erection once intercourse has begun?
   a. No.
   b. Yes.
   c. Have not had sexual intercourse within last 6 months.
5. How frequently do you awaken from sleep with a full erection?
   a. Daily.
   b. 2 or 3 times per week.
   c. Once a week.
   d. 2 or 3 times per month.
   e. Once a month.
   f. Less than once per month.
   g. Not at all within the last 6 months.
6. How satisfied are you with your sex life?
   a. Extremely satisfied.
   b. Somewhat satisfied.
   c. Neither satisfied nor dissatisfied.
   d. Somewhat dissatisfied.
   e. Extremely dissatisfied.
7. How satisfied are you with your sexual relationship with your present partner or partners?
   a. Extremely satisfied.
   b. Somewhat satisfied.
   c. Neither satisfied nor dissatisfied.
   d. Somewhat dissatisfied.
   e. Extremely dissatisfied.
8. How satisfied do you think your partner(s) is (are) with your sexual relationship?
   a. Extremely satisfied.
   b. Somewhat satisfied.
   c. Neither satisfied nor dissatisfied.
   d. Somewhat dissatisfied.
   e. Extremely dissatisfied.
9. Has the frequency of your sexual activity with a partner been:
   a. as much as your desire?
   b. less than you desire?
   c. more than you desire?

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