# Family Practice Grand Rounds

# Mitral Valve Prolapse

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Dr. KENNETH B. FRISOF *(Family Practice Residency Director, Cleveland Metropolitan General Hospital; Assistant Professor, Department of Family Medicine):* Mitral valve prolapse has a special interest for me because it is a disease we did not recognize when I was in medical school. The 1970 edition of Hurst and Logue's *The Heart*[1] does not mention the entity by name, although much of the then-known data about it was contained in a three-page subsection with the unappealing title, "Late Systolic Murmurs, Nonejection Clicks and Systolic Honks."

Some historians of science claim that scientific progress is not so much a result of genius inspiration as the technological development of new investigational tools. This concept certainly applies to mitral valve prolapse. Echocardiography has become central to making the diagnosis and is the foundation for the recent advances in our knowledge about it.

In the early 1970s, the practicing physician first became aware of mitral valve prolapse as a possible explanation for atypical nonanginal chest pain, especially in the younger person. In the mid 1970s, physicians started reading about the high prevalence of mitral valve prolapse, with studies demonstrating more people who have it than would usually be picked up in practice. This discrepancy between academia and practice did not seem to have any great significance for several years until mitral valve prolapse was recognized as a risk factor for certain life-threatening conditions.

So where do we stand in the cost-conscious 1980s? Mitral valve prolapse is now known to be the most common valvular abnormality in America, affecting approximately 9 million people if one extrapolates from the Framingham data.[2] Symptoms can be ascribed to this valvular abnormality in only 10 to 20 percent of these 9 million; life-threatening complications undoubtedly occur in less than 0.1 percent.

We hope that today's presentation will not only provoke your interest in our current conceptualization of mitral valve prolapse but will also serve as a basis to follow the future evolution of this fascinating and common condition. Dr. Foldy will now present Mr. F.M., a patient he has cared for over the last two years.

Dr. SETH FOLDY *(Third-year Resident in Family Practice)*: F.M. is a 34-year-old white man who presented to the Cleveland Metropolitan General Hospital Emergency Room in July 1983 complaining of pain in the left chest. Findings on physical examination, chest radiograph, and electrocardiogram were normal. At that time he was taking atenolol, 50 mg daily, and prazepam, 20 mg at bedtime, for mitral valve prolapse and anxiety.

The patient recalled being told he had a heart murmur at the age of 15 years. He dated the onset of episodic chest pain to 1979, when his wife left him temporarily. The episodes abated when he and his family moved from Cleveland to a nearby farm, a period the entire family recalled with good

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feeling. In 1980 the patient witnessed a sudden myocardial infarction in his 46-year-old half-brother. Shortly thereafter he began experiencing severe, sharp chest pain, which led to his quitting work as a plumber. He also experienced numbness and tingling of the arms and hands. At that time an exercise stress test was normal, and an echocardiogram revealed prolapse of the mitral valve. Nerve conduction studies were suggestive of mild right carpal tunnel compression.

The patient complained of intermittent sharp substernal and left chest pain, often accompanied by palpitations, severe anxiety, lightheadedness, shortness of breath, and diaphoresis. Although symptoms might occur with exercise, occasionally exercise appeared to improve them. He also experienced episodes of bilateral forearm and hand pain with paresthesias, temporally unrelated to chest pain, occurring most often at night.

Further questions revealed two types of chest pain. One was of a burning nature and occurred most frequently at night, after meals, and after alcohol consumption, and was relieved with antacids. The other was a sharp apical pain with sudden onset that frequently lasted for hours and was often associated with states of heightened emotion.

Family history revealed that his father had had heart disease and his mother had undergone coronary artery bypass graft. One half-brother had suffered two myocardial infarctions by the age of 43 years, another had a double coronary bypass at 47 years, and a third had hypertension. The patient has been living with his wife, two teenage daughters, and a younger nephew. He smokes two packs of cigarettes daily and engages in nights of "binge" drinking two to three nights each month.

Physical examination revealed a muscular man of average build weighing 78 kg. Blood pressure was normal on several visits; intermittently systolic pressures were elevated to 160 to 170 mmHg; pulse was regular and of normal rate. On the initial examination a grade 1/6 midsystolic murmur was heard. Auscultatory findings on other visits were variable, at times revealing a mid- to late-systolic murmur at the apex, occasionally accompanied by a midsystolic click. Usually only normal heart sounds were heard. Tinnell's test was positive on the right only.

Serum chemistries were normal except for a cholesterol reading of 260 mg/dL. Chest x-ray films and electrocardiogram were normal. Exercise stress testing showed only borderline high systolic blood pressure response to exercise. Upper gastrointestinal series revealed spontaneous gastroesophageal reflux without stricture or esophagitis. A 24-hour Holter ambulatory monitor revealed a normal sinus rhythm.

Therapeutic interventions included education regarding the mitral valve prolapse syndrome, gastroesophageal reflux, and carpal tunnel syndrome. The patient was encouraged to distinguish his different forms of chest pain. Dietary and postural recommendations were offered for gastroesophageal reflux, and the patient improved with cimetidine and antacid foam. Atenolol was restarted, and hydroxyzine was used for anxiety. "Cock-up" wrist splints improved symptoms of hand paresthesias. The patient was referred for vocational rehabilitation and received relaxation therapy.

The family interview revealed a high degree of isolation of the patient from his wife and children since the acceleration of his symptoms. He was excluded from discussion of many family issues for fear of aggravating his chest pain. Family members were reassured of the benign nature of his physical condition, and full involvement of the patient in family affairs was prescribed. In light of the striking family history of arteriosclerotic heart disease, dietary counseling was done and cessation of smoking recommended. Thus far the patient has refused participation in an organized alcohol abstinence program.

Dr. FRISOF: Dr. Weinstein, in what ways, if any, does Mr. F.M. fit the "typical" picture of the mitral valve prolapse patient?

Dr. MARLENE WEINSTEIN *(Third-year Resident in Family Practice)*: Mr. F.M. is, as we shall see, an unlikely candidate for mitral valve prolapse. A study from Framingham published in 1983 comprehensively addresses how common this lesion is and who is apt to have it.[2]

Previous studies have cited prevalence rates ranging from 5 to 15 percent. Many of the initial studies were limited by small sample sizes and selection bias. In contrast, the Framingham study involves a large sample from a noninstitutionalized general population in Massachusetts. It obtained M-mode echocardiograms on 4,967 subjects—

2,013 from the original cohort first studied in 1951 (mean age of 70 years), and 2,931 from a younger cohort consisting of the offspring of the original cohort and their spouses (mean age 44 years). Weight, height, and subscapular skin thickness were measured. The Quetelet's Index, a measure of body habitus, was calculated for each subject. The overall prevalence rates are 3 to 7 percent—3 percent in the elderly cohort and 7 percent for the younger cohort—averaging 5 percent for the entire population.

The Framingham study conclusively demonstrated that as women increase in age, their prevalence of mitral valve prolapse declines—from 17 percent in women aged 20 to 29 years, to 7.5 percent in women aged 50 to 59 years, to 1.4 percent in women aged over 80 years. In contrast, for men, prevalence was constant through the life span, ranging from 2 to 4 percent.

The reasons for the high prevalence of mitral valve prolapse among young women and its decline with age remain unclear. There is some pathologic and echocardiographic evidence that prolapsed mitral leaflets tend to become fibrotic and scarred secondary to abnormal flow stress, eventually limiting their abnormal motion. This hypothesis, however, does not explain the constant prevalence with age among men.

Leaner subjects, as measured by Quetelet's Index (weight/height$^2$) and the skinfold thickness index, had a significantly higher prevalence of mitral valve prolapse in both sexes. In fact, lean elderly women and lean elderly men have prevalences of prolapse that approach those of young women.

The finding that subjects with prolapse are thinner than those without it corroborates earlier clinical reports. One previous interpretation of this finding is that small body size is associated with small left ventricular chamber size, which is important for echo expression of mitral valve prolapse. However, in the Framingham study, subjects with prolapse were found to have slightly larger internal left ventricular dimensions compared with nonaffected people. Another fascinating facet of the correlation of mitral valve prolapse with lean body habitus is the finding that emaciated subjects with anorexia nervosa had a very high prevalence rate of 40 to 50 percent. The prevalence decreased as they gained weight.[2]

The currently accepted explanation of the high prevalence of prolapse among thin people is that mitral valve prolapse is part of a generalized connective tissue disorder that is linked to certain anthropometric features. Schutte and his colleagues[3] showed that women with mitral valve prolapse are thinner than the US average and have a distinctive habitus, with a long arm span and narrow anteroposterior chest diameter. Mitral valve prolapse has been shown to be a feature of several known connective tissue diseases associated with distinctive habitus including Marfan's and the Ehlers-Danlos syndromes.

DR. FRISOF: Have the recent large-scale studies clarified the familial nature of mitral valve prolapse?

DR. WEINSTEIN: The most comprehensive study of the genetics of prolapse was published by Devereux et al in 1984.[4] After studying 179 first-degree relatives of 45 probands identified at Cornell between 1975 and 1979, Devereux and colleagues concluded that mitral valve prolapse is inherited in an autosomal dominant fashion.

Echocardiographic prolapse was detected in 29 of the 45 families, occurring in 54 (30 percent) of the 179 first-degree relatives. Devereux et al also found age-dependent expression of mitral valve prolapse. Thirty-five percent of the adults were affected but only 8 percent of the children. As in the Framingham study, older women had a decreased frequency of prolapse. In men there was no age trend noted.

Devereux and colleagues inferred that the finding of 30 percent is greater than the 25 percent expected for a recessive trait, but falls short of the 50 percent expected in a fully expressed autosomal dominant disorder. He postulated that incomplete or nonpenetrant gene expression in men and the declining prevalence of prolapse among older women can account for the lower than predicted frequency of this disorder in the first-degree relatives of the probands. In addition, not all the potential first-degree relatives were available for study, which could have affected the final outcome.

Smaller studies—one by Weiss et al[5] in 1975, and another by Fortuin et al[6] in 1977—found prevalence rates of prolapse in first-degree relatives to approach 50 percent. On the other hand, a study by Scheele and colleagues[7] in 1976 and the

Framingham study of 1983 paralleled the results of Devereux et al. In summary, the existing evidence is conflicting. One can say, on balance, that (1) mitral valve prolapse is not recessively inherited, (2) that while its inheritance may well be autosomal dominant, this has not been definitely proven, and (3) mitral valve prolapse may represent a common product of genetic and nongenetic factors.

DR. FOLDY: Conclusions from genetic studies on live individuals tend to be better accepted if there is a recognizable cellular pathology associated with a condition. Have the pathologists been helpful?

DR. VICKY VALVERDE-SALAS *(Third-year Resident in Family Practice)*: Since mitral valve prolapse is a nonlethal disease, especially in the age range where it is most prevalent, pathologists have been unable to mount studies as massive and complex as those undertaken by the epidemiologists and geneticists. Nonetheless, some data are available. The microscopic morphology in mitral valve prolapse shows proliferation of myxomatous tissue in the valve itself. The spongiosa midlayer of the valve, which is composed largely of mucopolysaccharide stroma, is unusually prominent. There appears to be a fundamental, but as yet undefined, abnormality of collagen metabolism, resulting in a grossly abnormal and redundant valve, leading to prolapse. The cusps of the mitral valve, as well as the chordae tendinae and the annulus, may be affected by the myxomatous proliferation.

In most patients this myxomatous proliferation appears to be localized to the mitral valve. However, similar pathologic myxomatous proliferation has also been observed in a variety of connective tissue disorders, including Marfan's syndrome, Ehlers-Danlos syndrome, osteogenesis imperfecta, pseudoxanthoma elasticum, periarteritis nodosa, and lupus erythematosus.

DR. FRISOF: We have been discussing mitral valve prolapse at the cellular, family, and community levels. Let's now focus on recognizing the individual patient with mitral valve prolapse.

DR. VALVERDE-SALAS: Before Barlow and colleagues[8] elucidated the connection between findings and anatomy, midsystolic clicks and the variably accompanying late systolic murmur were widely thought to be extracardiac in origin and benign in nature. Since Barlow et al, these findings have been associated with prolapse of the mitral valve. The midsystolic click is thought to be due to the sudden tensing of the chordae and the prolapsing leaflets as left ventricular pressure rises. The late systolic murmur is due to the failure of apposition of the mitral leaflets, leading to various degrees of regurgitation.

It must be remembered that patients with mitral valve prolapse may have midsystolic clicks, late murmurs, or both. Some have only a click on one examination, only a murmur on another, and no auscultatory findings on a third occasion. In fact, in the Framingham study, where the physicians were not specifically instructed to search for mitral valve prolapse, clicks and murmurs were recognized in only 16 percent of patients with echocardiographically proven prolapse.[9] Despite the variability of auscultatory findings, it is generally accepted that the duration of the murmur is a function of the severity of the regurgitation.

Maneuvers on physical examination can aid the diagnosis. Maneuvers that decrease left ventricular volume by reducing venous return, ie, sitting, standing, the straining phase of the Valsalva maneuver, and amyl nitrite administration, lead to prolapse earlier in systole. The midsystolic click migrates closer to the first heart sound and the onset of the murmur advances toward midsystole. In its extreme, the click will disappear as it merges with the first heart sound, and the murmur may become holosystolic.

Increase of left ventricular volume through isometric exercise or sudden squatting delays the onset of the click and murmur. In auscultatory differential diagnosis, it is worth remembering that the auscultatory effects of these maneuvers are exactly the opposite of that seen in hypertrophic obstructive cardiomyopathy.

Echocardiography is the tool most commonly used to "definitively" make the diagnosis of mitral valve prolapse. Either two-dimensional or M-mode may be used, although two-dimensional is more sensitive. Abnormal posterior motion of either or both valve leaflets is the sole echocardiographic criterion for the diagnosis.

Echocardiography detects many more cases of the valvular abnormality than auscultation. Nonetheless, it is generally accepted that false-negative readings by echocardiography, especially M-mode, are not rare and may be as high as 10 to

20 percent.[10] There is, therefore, no single "gold standard" for the diagnosis of mitral valve prolapse.

DR. WEINSTEIN: My review of the epidemiologic literature and Dr. Valverde-Salas' review of diagnosis emphasize the large percentage of asymptomatic patients. How are we to understand the symptoms of mitral valve prolapse that some patients complain of? I understand that you have a "biopsychopathophysiologic" model to share with us, Dr. Frisof.

DR. FRISOF: Thank you. I do.

Mitral valve prolapse was first recognized in patients with atypical nonanginal chest pain and was used as an explanation of that pain. But, as physicians, we feel uncomfortable ascribing symptoms to a condition unless we can envision some sort of pathogenetic chain, as we do most classically with angina pectoris. How, then, can we credit any symptoms, especially intermittent symptoms, to a hemodynamically insignificant lesion like mitral valve prolapse?

In the mid 1970s, some attempt was made to blame the atypical chest pain on a combination of slight hemodynamic abnormalities and arrhythmia, but this concept was so lacking in substance it was soon dropped. At the same time, propranolol was empirically discovered to relieve the symptoms of mitral valve prolapse. In the late 1970s, then, the symptoms of mitral valve prolapse were reconceptualized as a neuroendocrine disorder of the adrenergic system.

Boudoulas and associates[11] have reported nearly twice as much epinephrine and norepinephrine in 24-hour urine collections from symptomatic mitral valve prolapse patients than from controls. Additionally, the Q-$S_2$ interval, a measure of electromechanical systole known to correlate with adrenergic tone, was significantly shorter in patients with mitral valve prolapse. Finally, isoproterenol infusion recreates the symptomatology of prolapse, causing significantly greater elevations of heart rate in patients with mitral valve prolapse than in controls.[12] Pasternac et al[13] in Montreal have also shown significant elevations in plasma catecholamines among patients with symptomatic mitral valve prolapse.

Is this heightened adrenergic tone really due to mitral valve prolapse, or is it due to some other condition in people who happen to have anatom-

ical mitral valve prolapse? To answer this definitively, we would need studies comparing individuals symptomatic with atypical chest pain with and without mitral valve prolapse, as well as studies in patients with prolapse with and without symptoms. These studies have not been done.

So what can we say about abnormalities of adrenergic tone? In 1969, Frohlich et al[14] reported from the Cleveland Clinic on 14 patients with symptoms of "cardiac awareness" (very similar to what we currently recognize as mitral valve prolapse symptoms), whom he labeled as having a "hyperdynamic $\beta$-adrenergic circulatory state." Unfortunately, this paper predates the mitral valve prolapse era, so Frohlich et al reported on neither their auscultatory nor their echocardiographic findings.

DR. WEINSTEIN: But isn't stress also associated with adrenal discharge?

DR. FRISOF: Yes. Since the mid 1970s it has been known that psychological stress can acutely elevate catecholamine levels. However, it was not until 1981 that chronic anxiety was first demonstrated to be associated with elevated plasma catecholamines. Mathew et al[15] reported on 15 patients with chronic anxiety whose plasma epinephrine and norepinephrine levels were 570 pg/mL before relaxation therapy and 441 pg/mL after it, as compared with nonanxious controls, whose mean level was 256 pg/mL.

It has been more than a century since DaCosta first described the symptom complex we currently associate with mitral valve prolapse—palpitations, sharp precordial pain, breathlessness, anxiety. Over the years, the labels have changed, alternating between physiological and psychological handles—irritable heart, effort syndrome, neurocirculatory asthenia, anxiety neurosis.[16] Both physiological and psychological conditions can be mediated through the autonomic nervous system, a key element of biopsychologic integration. Perhaps the symptoms of mitral valve prolapse result from pathology in the autonomic nervous system itself.

An interesting parallel exists with the entity functional reactive hypoglycemia. This diagnosis became popular in the early 1970s as an explanation for nonspecific postprandial symptoms such as sweating, confusion, headaches, and irritability, found most commonly in middle-class young women. A significant part of the physician community felt this condition to be merely a polite relabeling of psychological problems, and a 1974 *New England Journal of Medicine* editorial entitled "A Non-editorial on Non-hypoglycemia"[17] questioned the legitimacy of the syndrome because many symptomatic people had postprandial blood glucose levels above 50 mg/dL, while others without symptoms had glucose nadirs less than 40 mg/dL.

Recently, however, symptomatic patients have been clearly separated from asymptomatic patients by the level of plasma epinephrine at the time of the glucose nadir.[18] Thus, apparently, patients with functional reactive hypoglycemia, now renamed idiopathic postprandial syndrome, have autonomic nervous systems that are overly sensitive to falling blood glucose concentrations and discharge excessively, thereby producing the typical symptoms.

DR. FOLDY: Studies of adrenergic tone in congestive heart failure have shown that chronically elevated catecholamine excretion impairs sensitivity to catecholamines, so that hyperdynamic symptomatology is not produced.

DR. FRISOF: Good point. We presume, therefore, that in all these conditions we have discussed—anxiety, mitral valve prolapse, idiopathic postprandial syndrome—the adrenergic symptomatology is due to short bursts of catecholamine discharge. Perhaps the genetics of mitral valve prolapse define a group of individuals susceptible to excessive catecholamine stimulation: whether, in the individual instance, the autonomic discharge is due to random physiologic firing or is due to conscious or unconscious anxiety may well be a moot point.

So, although the symptoms of mitral valve prolapse are worrisome, they appear not to be serious. There are, however, serious complications of prolapse. Dr. Bedinghaus, can you elucidate the implications of recent literature on management of the patient with mitral valve prolapse?

DR. JOAN BEDINGHAUS *(Third-year Resident in Family Practice)*: The clinical management of the mitral valve prolapse patient is difficult because the literature raises more questions than it answers. Is mitral valve prolapse a disease or merely a finding? Is it a single disease or the anatomical expression of different diseases? What

is "its" natural history? Which patients are at risk for complications? Are the complications preventable?

None of these questions can be satisfyingly answered at present. As Drs. Weinstein and Valverde-Salas have discussed, echocardiographic evidence of mitral valve prolapse is widely prevalent, and a substantial majority of patients are asymptomatic and lack auscultatory findings. Yet, the presence of a prolapsing mitral valve seems to be associated with a risk of serious complications: bacterial endocarditis, cerebrovascular accidents, progressive mitral regurgitation, ventricular arrhythmias, and sudden death. The dilemma facing the primary care physician is to reduce, where possible, the chances of such catastrophic outcomes without wasting large sums of money or creating many cases of cardiac neurosis.

It is difficult to study rare events prospectively; therefore, the convincing evidence for mitral valve prolapse as a risk factor for rare serious complications has to come from retrospective case-controlled studies.

An increased incidence of endocarditis in mitral valve prolapse was observed in a study comparing the prevalence of echocardiographic prolapse in 51 endocarditis patients with 153 age- and sex-matched controls.[19] The endocarditis group had significantly more patients with prolapse than the controls; the calculated odds ratio indicates that patients with mitral valve prolapse are 8.2 times more likely to develop endocarditis than patients with no recognizable heart disease on echocardiogram.

Stroke in young people is also associated with mitral valve prolapse. Barnett et al[20] studied 60 patients aged under 45 years with stroke. Twenty-four of the patients had mitral valve prolapse. Of the 36 young stroke patients without mitral valve prolapse, 27 (75 percent) had one or more other recognizable stroke risk factors. On the other hand, of the 24 with mitral valve prolapse, only 6 (25 percent) had other risk factors. However, a study by Jones and colleagues[21] puts the role of prolapse as a cause of strokes in broader epidemiologic perspective. Of 401 stroke patients seen by a neurology service, only nine had mitral valve as the sole risk factor (all nine were aged under 60 years). Of 760 patients followed with prolapse in the cardiology clinic of the same center, only one

developed a stroke, and she had other risk factors.

Progressive mitral regurgitation may well be the most common complication of mitral valve prolapse, though most of the follow-up series do not attempt to distinguish between the very common idiopathic mitral valve prolapse and isolated mild mitral regurgitation secondary to rheumatic disease, cardiomyopathy, ischemic heart disease, or connective tissue disease. The series reported by Mills and colleagues[22] is an exception; their population was originally selected by phonocardiograms demonstrating isolated systolic clicks or murmurs. Two thirds of those patients underwent echocardiography, and no cardiac disease other than mitral valve prolapse was found. Five of the 53 patients in this series developed severe mitral regurgitation.

The risk of sudden death in people with mitral valve prolapse has received considerable attention, but the magnitude of the risk is unknown. In the Framingham study,[23] which selected subjects and controls on the basis of echocardiographic findings, the difference in incidence of high-grade ventricular arrhythmias between the two groups did not reach statistical significance. The authors do suggest, however, that prolapse patients with symptoms or auscultatory signs may have a higher incidence of serious arrhythmia.

Campbell et al[24] and Jeresaty[25] identified certain characteristics that they believe indicate increased risk of sudden death: a history of syncope, ST and T changes in the electrocardiogram, atrial fibrillation, and cardiomegaly. Studies of families with frequent mitral valve prolapse indicate that a family history of sudden death is also a significant indicator of risk.

DR. VALVERDE-SALAS: Although, as you have demonstrated, the studies on these rare complications are far from definitive, how would you suggest that we manage the individual patient?

DR. BEDINGHAUS: It may be useful to divide patients into several different categories: The first group are patients with echocardiographic prolapse who have no physical findings. In this setting it may well be reasonable to care for the patients' other clinical problems without specific attention to the prolapse.

The second group consists of patients with the characteristic click or murmur with or without symptoms. Because the diagnosis of mitral valve prolapse affects other therapeutic decisions, an echocardiogram for confirmation of the auscultatory diagnosis is probably justified. These patients seem to be at some risk for endocarditis, stroke, and possibly sudden death, though the magnitude of the risk is unknown. Most authorities agree that antibiotic prophylaxis for dental extraction and genitourinary tract procedures, including childbirth, is indicated in the presence of a murmur. Controversy persists about the need for prophylaxis when only a click is present. Because of the possible risk of stroke, one might hesitate to give oral contraceptives to young women with clicks or murmurs.

In the third group are patients with auscultatory and echocardiographic findings consistent with prolapse and a history of syncope or a family history of sudden death. These patients need a 24-hour Holter monitoring. If high-grade ventricular arrhythmias are found, propranolol seems to be first-line treatment; dilantin may be second choice. In the admittedly small samples of patients with life-threatening arrhythmias, those treated with quinidine did not do well, and this drug should probably be used with caution in the presence of prolapse.

DR. FRISOF: Dr. Foldy, can you make some concluding comments applying the material we have discussed to this patient?

DR. FOLDY: It is frustrating to deal with a concept in evolution, as is mitral valve prolapse. The majority of persons with prolapsing mitral valves will lead perfectly normal lives of normal length and never appreciate the existence of their own echocardiographic oddity. A few, most likely a very few, will experience premature illness or death. Now that increasing recognition of prolapse is outpacing our understanding of the consequences, there is a risk that otherwise completely healthy patients will be turned into psychic cardiac cripples each time a physician reads a new article on arrhythmias in "the" mitral valve prolapse "syndrome."

On the other hand, in the case of the patient presented today, the diagnosis of a recognized syndrome has had a reassuring effect in what to him was a frightening collection of symptoms. For F.M., the threat of sudden death once seemed imminent. It became possible to explain these symptoms, to place them in their proper context of

a syndrome with an excellent prognosis.

Mr. F.M.'s symptoms required careful elucidation by history and examination, with diagnostic studies as indicated. He suffered coexistent gastroesophageal reflux and probable carpal tunnel syndrome. If his evaluation had ended with the finding of a prolapsing mitral valve, readily treatable symptoms might have been forced into a template diagnosis of "mitral valve prolapse syndrome," and the patient would not have benefited from their relief.

His impressive family history still posits a definite risk of coronary artery disease. Efforts at the detection and elimination of cardiovascular risk factors are as important in this patient as in any other. He also demonstrates the perplexing confoundability of ischemic chest pain with that of the mitral valve prolapse syndrome. In this patient, a negative stress test was reassuring; perhaps the systolic hypertension noted during the examination demonstrated the heightened adrenergic tone seen in prolapse. If a stress test is equivocal, stress thallium scintigraphy is a useful tool.

Finally, the case of F.M. demonstrates how the family physician must get involved in family dynamics. By the loss of his career and the family's careful avoidance of involving the patient for fear of precipitating a potentially fatal heart attack, the patient had become highly isolated and impotent. Detailed explanation of the syndrome to the entire family, as well as family therapy aiming at reintegration of the patient into active family life, was essential to the good outcome of this case.

We have reached an awkward stage in our understanding of mitral valve prolapse. It is hoped that future research will be more helpful in splitting, rather than lumping, populations united by this ill-defined common finding. If so, our care of patients can be better tailored to their individual risks. Long-term prospective studies should be of great utility here. Indeed, the continuous study of many patients without any serious illness may be most efficiently undertaken by a large number of family physicians in their offices. Until more is known, we must be careful to avoid making cardiac neurotics out of asymptomatic patients while, as in this case, helping those with frightening symptoms to regain their place in the home and workplace through the explication of a syndrome that is almost always benign.

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