
Family Practice Grand Rounds

A Patient Homebound by Panic: Understanding and Treating Agoraphobia

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BARBARA DICICCO-BLOOM (*Nurse, Department of Community Medicine*): A patient made homebound by panic is the subject of today's conference. Our nurse-physician-social worker teams have worked with more than 1,000 individuals chronically ill at home in the past decade¹ through the hospital's Chelsea-Village Program (CVP), but the need to deal with major phobias is unusual in our experience.

We will present the pertinent social and health issues of our patient, followed by comments from Dr. Harold Taylor, a member of the Department of Psychiatry, who is our guest consultant today. We hope to better understand the theoretical basis of agoraphobia and panic attacks, leading to plans that will allow us to help our patient more effectively.

Mr. V.O. is a 57-year-old homebound man, known to the CVP for the past three months. He was referred to us by a private physician who was

concerned that the patient was unable to get to his office. Mr. O. had told the physician that he was afraid to leave his apartment.

On our first home visit, we noted that Mr. O. lived in one room, six by twelve feet in size, on the third floor of a walk-up brownstone building. Cooking facilities and sink were included. The bathroom was in the hallway.

The patient was found to be lying in an unmade bed. The shade was drawn on the single window. The room was cluttered and unclean. He told us that he wanted to get up and go out, but knew that he would be unable to breathe if he left his room. He was in a state of terror. He wished us to know that he was an artist and poet. The walls of his room were covered with his watercolor paintings.

Mr. O's father, an alcoholic, abandoned his family when the patient was 6 years old. Prior to this, the father had beaten his wife regularly in the patient's presence. There are two younger siblings. The patient feels that they received fairer treatment than did he. Mr. O. now has no relationship with his siblings, but has occasional contact with his mother by telephone. She is 86 years old and lives in Queens. Mr. O. has never married and is a self-proclaimed homosexual.

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The patient lived with his mother until he was 44 years old, when she turned him from her home. He spent the next seven years living intermittently on New York City's skid row, the Bowery, in a state of severe alcoholism. This led to an admission to the alcohol detoxification unit of our hospital in 1976. The patient states that he has not drunk alcohol since that time. He has been living in his single room, alone, since then. Two years ago he applied for an elevator apartment in a New York City housing project. He is deeply anxious that when this is finally approved, he will be afraid to move.

DR. PHILIP W. BRICKNER (*Physician, Department of Community Medicine*): In brief, Mr. O. has chronic obstructive pulmonary disease of moderate severity. His alcoholic history has been noted. This required a hospital admission in 1976. He has had a major depressive episode, with a second hospitalization for one month in 1979.

On our initial examination in April 1982, the vital signs were normal, with a resting respiratory rate of 10 per minute. The patient's attitude was open, courteous, cooperative, and humorous. He did not cough during the examination and was able to walk freely in his room without dyspnea. In general appearance he was thin and healthy. Skin and mucous membranes were of normal color. The spine was slightly kyphotic and he had a moderate barrel chest. The lungs were normal to inspection and percussion. Breath sounds were coarse throughout, without wheezes, rales, or rhonchi. There was no clubbing of the nails and no other positive physical findings. Laboratory data, including complete blood count, urinalysis, and SMA-12, were within normal limits. Electrocardiogram showed normal sinus rhythm, complete right bundle branch block, and right axis deviation. Chest roentgenograms taken during an emergency room visit in January 1982, the last time the patient was out of his apartment, were consistent with moderately severe, chronic obstructive pulmonary disease. Present medical regimen includes 200 mg of theophylline taken orally three times a day and albuterol spray.

In summary, the patient has chronic obstructive pulmonary disease of a severity insufficient to make him homebound.

MARY SUGRUE (*Social Worker, Department of Community Medicine*): In the first three months

of my relationship with Mr. O., I made five home visits and had several telephone contacts.

At first our team effort centered around improving the patient's physical illness. He claimed that his emphysema was the reason that he was not able to go outside. He felt certain that he would stop breathing on the street.

He requested that we obtain portable oxygen for him, and insisted that this "crutch," his own term, would be the answer to his feelings of insecurity about going outside. Even though we did not encourage this step, the patient was so determined to proceed that he obtained the money from his mother and purchased the equipment. When he got dressed and put it on, however, he found once again that he was unable to leave his room.

Since then my efforts have centered on the fear and anxiety that make him homebound. I found it difficult to get the patient to focus on his feelings. He preferred to talk about his physical symptoms in great detail or blamed his difficulties on negative past personal experiences. He explained to me that he spends hours thinking about his phobia. He chides and blames himself.

At times he exhibits infantile confidence in our ability to alleviate his distress but has gradually realized that he must assume more responsibility. He is highly dependent and continually seeks reassurance and nurturing.

Recently the patient started to do more art work and writing of poetry. His mood became brighter; his thinking was somewhat grandiose: "I'll arrange an art exhibit on Madison Avenue. I'm at the highest peak of my creativity." He was enthusiastic about life, but still unable to go outside.

In the most recent three weeks, however, his mood has again changed. He is now once more depressed. He seems to realize that his magical thinking is not working. He denies spontaneously and repeatedly being suicidal. This concerns me.

DR. C. HAROLD TAYLOR, JR (*Physician, Department of Psychiatry*): This patient presents an interesting and challenging clinical problem. It appears that he has developed fear of leaving his home, associated with obsessive ruminations that some type of medical catastrophe might occur if he did. The date of onset of these symptoms is not clear. They may have emerged as soon as he stopped abusing alcohol six years ago. They were certainly present during his psychiatric hospitalization three years ago. At that time, it was thought

that his phobias were more prominent than his depression. There was some improvement in these phobic symptoms during his hospitalization, but he was lost to psychiatric follow-up after discharge.

There has been a great deal of interest in recent years in the agoraphobic syndrome,^{2,3} of which the patient's problem is a variant. The term *agoraphobia* is currently used to refer to a constellation of fears and avoidance behaviors that seems to be a valid, discrete clinical entity. Fears of leaving the home are combined with anxiety about being in crowds, traveling on public conveyances, or standing in line. The patient is usually much less anxious if accompanied.

The agoraphobic cluster may or may not be accompanied by full-blown panic attacks, characterized by dyspnea, palpitations, chest pain, dizziness, and so forth. Even if the agoraphobic person does not suffer from frank panic attacks, the intense anxiety experienced in trying to enter the phobic situation may induce secondary fears—the patient may be afraid of fainting, suffering a cardiac arrest, or going insane. A variant I have seen on several occasions is the patient who suffers from “irritable bowel syndrome” and who has the typical constellation of agoraphobic fears. For these patients, going away from home, traveling, or keeping appointments produces feelings of tenesmus, associated with anxiety that they might lose control of their bowels. Their thoughts center on whether they will be able to find a toilet in time to avoid an embarrassing episode.

The majority of agoraphobic patients are women, and the typical age of onset is between 18 and 35.² In this sense, the patient we are discussing today is atypical, although the age at which his symptoms began is obscured by his history of alcoholism. It is clear that he has suffered from this problem with varying degrees of intensity for several years, since it was present prior to his hospitalization in 1979. In general, it has been found that once the syndrome has been present for at least a year, it will persist unless treated.⁴

MS. DICICCO-BLOOM: Is there any value in reassuring these patients that the catastrophe they fear will not occur? Might his portable oxygen machine have helped in this regard?

DR. TAYLOR: In mild cases, where full-blown attacks are not experienced, using the health care provider-patient relationship as a source of encouragement and reassurance can make it

possible for the patient to enter the phobic situation. If this is done in a systematic manner, the patient may learn to overcome his fears.

There is another approach. A well-known psychoanalyst used to give his agoraphobic patients a letter, which they could carry as a kind of magical talisman. It read something like “I, Dr. X., personally guarantee that Mrs. Y. can go anywhere she chooses, and absolutely no harm will befall her.”⁵ Some form of reassurance and education in regard to the somatic manifestations of anxiety, which the patient often interprets as symptoms of a dire illness, is important. However, this is by no means sufficient in most cases.

SR. SUGRUE: Are these patients typically very dependent individuals?

DR. TAYLOR: In cases of psychiatric disorder, it is important to ask whether a given set of behaviors and attitudes represents enduring personality traits, or whether they are the product of the psychiatric illness or state. For example, certain people who act in dependent ways when they are clinically depressed do not act in those ways when their depression is treated. Certainly, people who suffer from an agoraphobic syndrome are likely to come across as quite dependent; they may insist on a companion in order to get to the places they have to go. The constriction of their activities may mean that others have to be active on their behalf. A spouse may have to do the shopping, a friend may have to take the children to school.

The question remains, Do these patients tend to be more dependent than average prior to the onset of the illness? Most clinicians, including myself, believe they do. However, this has not been validated by systematic studies. Today's patient certainly seems to be a highly dependent person. He was quite indignant when his mother insisted that he move out of the family home and get an apartment of his own, although he was by then in his forties. It was after this that he became a Bowery alcoholic.

This raises another issue. Whatever the cause of the behavior patterns associated with this syndrome, it is important to evaluate the role of other people who are significant to the patient. For example, are family members fostering or rewarding specific behaviors? For some patients, the family's response to their disability can be so gratifying that they are reluctant to give it up. This sec-

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

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

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

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

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

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

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

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ondary gain can be a major obstacle to treatment. In the case of Mr. O., the vicissitudes of his relationship with his mother may be relevant to the severity of his symptoms. It turns out that after his discharge from the psychiatric inpatient service in 1979, his mother allowed him to return home for a while. Perhaps he hopes that if he becomes disabled enough again, she will let him return once more.

DR. ANTHONY LECHICH (*Physician, Department of Community Medicine*): What are the current therapies for this syndrome?

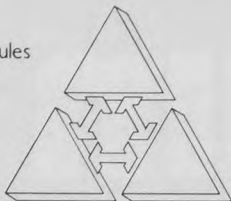
DR. TAYLOR: Over the past 20 years or so, the major developments in treatment strategy have been psychopharmacologic therapy and approaches based on learning or behavioral theory.

In regard to pharmacologic treatment, some interesting developments have taken place. A growing number of psychiatrists believe that medications ordinarily used in the treatment of depression, such as tricyclic antidepressants or monoamine oxidase (MAO) inhibitors, are of value in the treatment of agoraphobia.⁶⁻⁸ A model for the development of agoraphobia, expounded by Klein, has gained wide acceptance in this country.^{7,9} According to Klein, the agoraphobic syndrome is regularly fueled by panic attacks. He speculates that there is an element in the neurophysiology of these patients that makes them especially vulnerable to panic attacks. After the occurrence of one or more especially severe attacks, patients may develop secondary fears of going outside lest they have one of these terrifying attacks when alone and unprotected. According to this theory, then, the core of the disorder is usually the tendency to develop panic attacks; as a secondary phenomenon, the patient develops anticipatory anxiety about going outside. It is claimed that tricyclic antidepressants or MAO inhibitors can be effective in reducing or eliminating the panic attacks.

Even after this is accomplished, the patient will still have to "unlearn" the anxiety he anticipates in going outside. This may require behavioral treatment. It is an interesting effect of these medications that their usefulness does not seem to correlate with the degree of the patient's depression. In other words, their therapeutic action seems to be distinct from their antidepressant effects. The tricyclics can sometimes be effective in doses

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CONTRAINDICATIONS

IMODIUM is contraindicated in patients with known hypersensitivity to the drug and in those in whom constipation must be avoided.

WARNINGS

Antiperistaltic agents should not be used in acute diarrhea associated with organisms that penetrate the intestinal mucosa, e.g., enteroinvasive *E. coli*, *Salmonella*, *Shigella*, and in pseudomembranous colitis associated with broad-spectrum antibiotics.

Fluid and electrolyte depletion may occur in patients who have diarrhea. The use of IMODIUM does not preclude the administration of appropriate fluid and electrolyte therapy. In some patients with acute ulcerative colitis, agents which inhibit intestinal motility or delay intestinal transit time have been reported to induce toxic megacolon. IMODIUM therapy should be discontinued promptly if abdominal distention occurs or if other untoward symptoms develop in patients with acute ulcerative colitis.

PRECAUTIONS

In acute diarrhea, if clinical improvement is not observed in 48 hours, the administration of IMODIUM should be discontinued.

Abuse and Dependence: Physical dependence to IMODIUM in humans has not been observed. However, studies in monkeys demonstrated that loperamide hydrochloride at high doses produced symptoms of physical dependence of the morphine type.

Carcinogenesis: In an 18-month rat study with doses up to 133 times the maximum human dose (on a mg/kg basis) there was no evidence of carcinogenesis.

Pregnancy: Safe use of IMODIUM during pregnancy has not been established. Reproduction studies performed in rats and rabbits with dosage levels up to 30 times the human therapeutic dose did not demonstrate evidence of impaired fertility or harm to the offspring due to IMODIUM. Higher doses impaired maternal and neonate survival, but no dose level up to 30 times the human dose demonstrated teratogenicity. Such experience cannot exclude the possibility of damage to the fetus. IMODIUM should be used in pregnant women only when clearly needed.

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Pediatric Use: Safety and effectiveness in children have not been established. Therefore, use of IMODIUM is not recommended in the pediatric age group (under the age of 12). In case of accidental ingestion of IMODIUM by children, see Overdosage Section for suggested treatment.

ADVERSE REACTIONS

The adverse effects reported during clinical investigations of IMODIUM are difficult to distinguish from symptoms associated with the diarrheal syndrome. Adverse experiences recorded during clinical studies with IMODIUM were generally of a minor and self-limiting nature. They were more commonly observed during the treatment of chronic diarrhea.

The following patient complaints have been reported: Abdominal pain, distention or discomfort; Constipation; Drowsiness or dizziness; Dry mouth; Nausea and vomiting; Tiredness.

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OVERDOSAGE

Animal pharmacological and toxicological data indicate that overdosage in man may result in constipation, CNS depression, and gastrointestinal irritation. Clinical trials have demonstrated that a slurry of activated charcoal administered promptly after ingestion of loperamide hydrochloride can reduce the amount of drug which is absorbed into the systemic circulation by as much as ninefold. If vomiting occurs spontaneously upon ingestion, a slurry of 100 gms of activated charcoal should be administered orally as soon as fluids can be retained.

If vomiting has not occurred, gastric lavage should be performed followed by administration of 100 gms of the activated charcoal slurry through the gastric tube. In the event of overdosage, patients should be monitored for signs of CNS depression for at least 24 hours. If CNS depression is observed, naloxone may be administered. If responsive to naloxone, vital signs must be monitored carefully for recurrence of symptoms of drug overdose for at least 24 hours after the last dose of naloxone.

In view of the prolonged action of loperamide and the short duration (one to three hours) of naloxone, the patient must be monitored closely and treated repeatedly with naloxone as indicated. Based on the fact that relatively little drug is excreted in urine, forced diuresis is not expected to be effective for IMODIUM overdosage.

In clinical trials an adult who took three 20 mg doses within a 24-hour period was nauseated after the second dose and vomited after the third dose. In studies designed to examine the potential for side effects, intentional ingestion of up to 60 mg of loperamide hydrochloride in a single dose to healthy subjects resulted in no significant adverse effects.

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AGORAPHOBIA

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much smaller than those which would be used to treat a depressive syndrome.⁷ In the case of Mr. O., since he was hospitalized in 1979 he has carried the dual diagnosis of phobic disorder and depressive disorder. Amitriptyline could be useful for both. Even if he is not currently clinically depressed, the intensity of his panic reactions is such that there is a rationale for resuming treatment with this medication.

DR. BRICKNER: What is the role of tranquilizers in the treatment of this syndrome?

DR. TAYLOR: It is generally accepted that phenothiazines are not helpful in treating this disorder. The benzodiazepines may be of value in some cases as an adjunct to other treatments, but they are not effective alone. The latter medications also involve the risk of drug dependence.

SR. SUGRUE: What are the behavioral therapies that are used?

DR. TAYLOR: There are several.^{2,8} The common element is that they involve exposing the patient to the feared situation, either through imagination or real life, until the patient's anxiety in that situation begins to diminish. Thus, the patient is gradually desensitized to the feared experience, and patients "unlearn" their conditioned anxiety responses to going outside, traveling, and so forth. There is reason to believe that systematic exposure in real life ("in vivo") is more effective than exposure using a hierarchy of scenes that the patient imagines in the clinician's office.² Desensitization in imagination probably is effective only if the patient can eventually regularly practice entering the phobic situation in real life between treatment sessions.² It is crucial that the patient be able to stay in the phobic situation until anxiety begins to diminish. Otherwise, his fears of the situation will merely be intensified by the experience, and he will become demoralized about treatment as well. An example of an in vivo treatment program for Mr. O. might be as follows: construct a hierarchy or a list of feared situations, ranging from those that cause mild anticipatory anxiety to those that create tremendous apprehension. For Mr. O. an example of an item low on the scale might be to walk down to the end of the corridor on his floor. A mid-range item might be to walk from his apartment to the hospital. A very difficult item might be to take the subway unaccompanied uptown to an

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art gallery. Since he lives by himself and his mother's influence is possibly not constructive, his attachment to the treatment team would provide reinforcement for trying these activities. He appears to be quite sensitive to the team members' approval or disapproval. He might be encouraged to try the easiest item on the hierarchy. After receiving much praise from the team if he accomplished it, he could repeat this item until he was comfortable. Then he could move on to a more difficult item.

SR. SUGRUE: Since he is homebound, won't this require a great deal of staff time and energy?

DR. TAYLOR: Yes. For severe cases, this can be a very labor-intensive kind of treatment at the beginning. For this reason, a group in Oxford, England, has made a practice of teaching the patient's family members how to help the patient to implement the treatment program.² It is the family members, with their systematic encouragement and praise, who help the patient work his way up the hierarchy. In essence, they are enlisted as assistant therapists. Alternatively, groups of agoraphobic patients have been used for in vivo desensitization.¹⁰ They may meet at an assigned rendezvous, and with the help of a therapist, all attempt tasks from their respective hierarchies. Afterwards, they meet to discuss their experiences. Again, group praise and encouragement can serve as a significant reinforcer. The emphasis is on positive accomplishments rather than on distressing symptoms.

MS. DICICCO-BLOOM: What if Mr. O. cannot be persuaded even to attempt the simplest task, such as coming out of his room and walking the length of the hall?

DR. TAYLOR: It may be necessary to hospitalize him so that he can be stabilized on a medication such as amitriptyline and some efforts at a behavioral therapy can be made. Fortunately, this kind of situation is not common.

DR. LECHICH: It seems that the patient may have used alcohol to medicate himself, that is, to suppress his anxiety. Do agoraphobics do this often?

DR. TAYLOR: It has been observed that some agoraphobics do indeed medicate themselves with alcohol or minor tranquilizers and thereby become substance dependent. It has been estimated that 5 to 10 percent of agoraphobics will develop this kind of coping mechanism.^{2,11} Sometimes they

present first for treatment of substance abuse, and only after psychiatric evaluation are their agoraphobic symptoms recognized.

MARY KOHN (*Social Worker, Department of Community Medicine*): Is there a characteristic family background of these patients?

DR. TAYLOR: There is some evidence that a tendency to develop anxiety disorders can be inherited, and that this may play a role in the development of the agoraphobic syndrome.^{2,12} The syndrome itself has not been shown to run in families.² From the point of view of family environment, it is said that the mothers of these patients have frequently been quite fearful and overprotective,^{13,14} although this has not been well documented. In the case of Mr. O., it seems that his early family environment was quite frightening and that his mother may, indeed, have been overly protective.

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