

Denial Predicts Favorable Outcome in Unstable Angina Pectoris

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Denial may be prognostically favorable in patients with acute myocardial infarction. We analyzed the significance of denial in 26 patients referred to a tertiary care center for advanced therapy of unstable angina. Group A comprised 14 patients characterized as deniers on the Hackett-Cassem Denial Scale. Group B comprised 12 nondeniers.

There were no differences between groups in multiple baseline social and demographic characteristics, cardiac history, or risk factors. Similarly, there were no differences in the number of diseased vessels or left ventricular function in those patients catheterized (11 Group A patients, 9 Group B patients). Group B, however, had a longer hospitalization until medically stabilized (pain-free for 36 hr) than Group A (5.9 ± 3.6 days vs. 3.0 ± 1.6 days; $p=0.02$) despite similar treatment regimens. There were no significant differences in incidence of myocardial infarction or need for surgery. There were two deaths—both in Group B patients.

We conclude that denial independently predicts rapid medical stabilization in unstable angina patients. Whether it predicts better longterm outcome requires further study.

INTRODUCTION

Physicians have been interested in psychological factors influencing ischemic heart disease since 1818, when Heberden observed that angina pectoris "is increased by disturbances of the mind," (1). It has long been observed that many patients with coronary disease deny their illness (2). Hackett and Cassem further explored the idea that denial may be an adaptive psychological response to acute coronary disease (3-7). They studied 50 patients with myocardial infarction in a coronary care unit (CCU) and divided

them into major, partial, and minimal deniers (3). Since none of the four deaths in the sample occurred in major deniers, they suggested that denial might have value for immediate survival. Doehrman (8) noted that although the denial-survival hypothesis was frequently cited, the data of Hackett et al. (3) and others (9) were at best only suggestive, and not statistically significant. Furthermore, recent work has not shown significant differences between deniers and nondeniers in CCU mortality (10, 11), infarct size (10), readmission rates (11), exercise tolerance (12), or rehabilitation status (11).

Despite the paucity of supporting evidence, clinicians widely believe that deniers have less morbidity and mortality in the CCU (4, 12, 13). This belief can affect the doctor-patient relationship. Many physicians support the denial of illness, rather than encouraging their patients to immediately face the realities of being critically ill (14). Although the denial hypothesis continues to receive wide attention

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(e.g., 15, 16), its broad acceptance is not sufficiently supported by empirical data.

Most studies have focused on patients with acute myocardial infarction in the CCU, with much less attention to patients hospitalized for unstable angina (12, 16, 17). Unstable angina patients can usually be medically stabilized in the CCU, but a number go on to have myocardial infarctions or require urgent revascularization and a few die. It has been difficult in the studies of myocardial infarction (cited above) to significantly correlate denial and survival because there were few or no deaths in the CCU. The present investigation was designed to determine whether denial is associated with better outcome measures in patients admitted to a CCU with unstable angina.

METHODS

Initially eligible patients for this study included all those admitted to the coronary care unit at Westchester County Medical Center with the diagnosis of unstable angina between 12/1/81 and 4/1/82. Unstable angina was defined as the presence of typical rest pain associated with transient ST-T wave changes on the electrocardiogram. Patients were excluded if a) there was retrospective ECG or cardiac enzyme evidence that an acute myocardial infarction had occurred on or immediately prior to admission, b) there was a documented myocardial infarction in the two weeks prior to admission, c) the patient did not give informed consent (one patient excluded), d) the patient's medical condition precluded a 20-min interview (none excluded).

All study subjects underwent a semistructured interview within 48 hours of admission and were categorized as deniers or nondeniers based on the Hackett-Cassem Denial Scale (4). This is a 31-item objective rating scale comprised of questions, asked by an interviewer, assessing coping behavior used by patients before and during CCU stay. Reliability and validation studies for the scale have been conducted (5). For the purposes of this study, a score of 16 or more signified deniers (Group A) and a score of 15 or less signified nondeniers (Group B).

Physicians who had no knowledge of the patients' psychologic categorization collected demographic

and social data through interviews (Table 1) and obtained data on coronary risk factors, cardiac history, N.Y.H.A. Class (Table 2), cardiac catheterization (Table 3), and hospital course (Table 4). Mean daily blood pressures and heart rates were taken as the average of 6–12 readings as recorded by the nursing staff on the vital signs sheets. Medical stabilization was defined as absence of chest pain for at least 36 hr, as documented in the patients' charts by their physician. Acute myocardial infarction was defined by standard criteria (18, 19). Coronary artery lesions in catheterized patients were graded according to the Ad Hoc Committee on Grading of CAD of the American Heart Association (20). Left ventricular function was assessed by the ejection fraction from the RAO cineventriculogram (21), and by the end-diastolic pressure measured through fluid filled catheters (22).

Therapy was at the discretion of each patient's personal physician, although most physicians prescribed bed rest, β -blockers and sublingual isosorbide dinitrate as initial therapy. Intravenous nitroglycerin and oral calcium blockers were instituted when patients continued to experience episodes of pain on initial therapy. Intraaortic balloon counterpulsation and/or urgent catheterization and surgery were reserved for the occasional patients who were refractory to all the above measures. Many patients, however, underwent elective catheterization on the same admission after a period of stabilization.

At the conclusion of the study period, the code was broken and the baseline characteristics and hospital courses were compared between groups. Group means were compared using Student's t-test for unequal groups, and group characteristics using Fisher's exact test.

RESULTS

The study group was comprised of 26 patients. Group A consisted of fourteen patients categorized as deniers with a mean Hackett-Cassem score of 25.6. Group B consisted of 12 patients categorized as nondeniers with a mean score of 8.1. The mean age of the entire group was 60 years, without a significant age difference between Group A and Group B. Twelve Group A patients and eight Group B patients were male. All patients were Caucasian. There were no differing trends

TABLE 1. Demographic and Social Data

	All patients (n = 26)	Group A (Deniers, n = 14)	Group B (Nondeniers, n = 12)	Statistical Significance ^a
Mean age	59.5	58.4	60.8	(NS)
Sex				
Male	20	12	8	(NS)
Female	6	2	4	
Vocation				
Blue collar	14	9	5	(NS)
White collar	12	5	7	
Education				
12th Grade or less	15	8	7	(NS)
Beyond high school	11	6	5	
Vocational status				
Working	16	12	4	(NS)
Retired	10	2	8	
Religion				
Catholic	11	7	4	
Protestant	5	3	2	
Jewish	8	3	5	
Other	2	1	1	
Living situation				
With spouse	20	11	9	
With spouse and family	4	3	1	
Alone	2	0	2	
Social supports during hospitalization				
Spouse	22	12	10	
Family	24	12	12	
Friends	13	6	7	
None	2	2	0	

^aFisher's exact test, two-tailed.

TABLE 2. Clinical Baseline

	All patients (n = 26)	Group A (Deniers, n = 14)	Group B (Nondeniers, n = 12)
Coronary risk factors			
Hypertension	12	6	6
Smoking history	19	11	8
Hypercholesterolemia	4	2	2
Diabetes	5	3	2
Family history	21	11	10
Cardiac history			
Angina	20	11	9
Rest angina	6	3	3
Myocardial infarction	14	8	6
Congestive heart failure	5	3	2
Arrhythmias	9	5	4
N.Y.H.A. class			
Class 3	3	2	1
Class 2	12	6	6
Class 1	3	1	2
No antecedent angina	8	5	3

TABLE 3. Cardiac Catheterization Data

	Group A (n = 11)	Group B (n = 9)	Statistical Significance ^a
Number of vessels with significant (≥70%) lesions			
Three	5	4	
Two	3	3	
One	3	2	
Mean LV ejection fraction	56%	54%	(NS)
Mean LVEDP	12.5	13.0	(NS)

^aStudent's *t*-test for unequal groups, two-tailed.

in any of the other baseline demographic and social data (Table 1) except for vocational status. Twelve Group A patients were still working, but this was true of only four from Group B.

The baseline clinical data from Table 2 were also similar for Group A and Group B. Six Group A and six Group B patients had a history of hypertension. Eleven Group A and eight Group B patients had smoking histories. Two patients from each group had documented hypercholesterolemia. Three Group A and two Group B patients were diabetics. Eleven Group A and ten Group B patients gave positive family his-

ories for coronary disease. Eleven Group A patients and nine Group B patients had histories of prior angina pectoris, and three patients from each group had prior rest angina. Eight Group A patients and six Group B patients had prior myocardial infarctions. Three Group A and two Group B patients had prior symptoms of congestive heart failure, and five Group A and four Group B patients were being treated with chronic antiarrhythmics. The two groups were very similar in their division into N.Y.H.A. classes.

Although the number of different B blocker preparations and nitrate preparations precluded statistical analysis, there were no obvious trends toward greater baseline therapy with these agents in either group. Three Group A and two Group B patients were on calcium channel blocker therapy (nifedipine) upon admission.

Eleven Group A patients and nine Group B patients underwent cardiac catheterization. Five Group A patients had three vessel involvement, three had two vessel involvement, and three had single vessel disease. Four Group B patients had three

TABLE 4. Hospital Course

	Group A (n = 14)	Group B (n = 12)	Statistical Significance ^a
Anginal episodes per day ^b	2.8	3.0	(NS)
Sublingual nitroglycerin, per day ^b	2.0	1.9	(NS)
β-blocker dose (propranolol) per day ^b	125 mg	121 mg	(NS)
Nitrate dose (S.I. isosorbide dinitrate), per day ^b	15.5 mg	16.0 mg	(NS)
Blood pressure (mean) ^b	128/83	130/85	(NS)
Heart rate (mean) ^b	76 bpm	73 bpm	(NS)
Time to medical stabilization, mean	3.0 days	5.9 days	<i>p</i> = 0.02
IV nitrates and/or calcium channel blocker therapy	9 patients	8 patients	(NS)
Intraaortic balloon pump use	0	0	
Documented myocardial infarction	3	2	
Emergency revascularization	0	0	
Death	0	2	

^aStudent's *t*-test for unequal groups, two-tailed.

^bMean, hospital day 1.

DENIAL: A FAVORABLE OUTCOME IN ANGINA

vessel disease, three had two vessel disease, and two had single vessel disease (NS). Mean ejection fraction was 56% in Group A patients and 54% in Group B patients (NS). Mean left ventricular end-diastolic pressure was 12.5 in Group A patients and 13.0 in Group B patients (NS).

Analysis of the hospital course parameters for day 1 showed no difference in the number of anginal attacks (2.8 vs. 3.0), mean β -blocker dose on day 1 (125 mg vs. 121 mg of propranolol), mean nitrate dose (15.5 mg vs. 16.0 mg sublingual isosorbide dinitrate), mean heart rate (76 bpm vs. 73) or mean blood pressure (128/83 vs. 130/85). However, Group A patients were medically stabilized (absence of chest pain for 36 hours) much more rapidly than Group B patients. The mean time to medical stabilization in Group A was 3.0 days, and in Group B, 5.9 days ($p=0.02$). The need for intravenous nitroglycerin or addition of oral calcium blockers did not differ between the groups, with nine group A patients and eight Group B patients requiring either or both of these agents. No patient in the study required emergency revascularization or intraaortic balloon counterpulsation for symptom control. Three Group A patients and two Group B patients experienced myocardial infarctions. There were two deaths, both in Group B patients.

DISCUSSION

Weisman and Hackett defined denial as the "conscious or unconscious repudiation of part of all of the total available meaning of an event to allay fear, anxiety, or other unpleasant affects" (23). Thus, denial is viewed as a psychologic defense mechanism, whereby the individual avoids awareness of a threatening reality. Many physicians believe that denial is

associated with an improved prognosis in acute ischemic heart disease syndromes. The studies on which these impressions are based have operationally defined denial in a variety of ways, making comparison between studies difficult. In earlier research (3, 9, 10, 24) denial was assessed qualitatively, with each research group utilizing a different method. Hackett and Cassem developed and validated a quantitative rating scale (4-6), which has been used inconsistently by others. In some instances, the scores and cutoffs were not described (11, 15), while in others (12) the cutoff score for separating groups has not agreed with that used by Hackett and Cassem. There have also been investigators who used other instruments to measure denial (16, 25). In the present study, the cutoff value of 15 on the rating scale of Hackett and Cassem was used, following their example (6).

The outcome of any medical illness usually depends on a multiplicity of factors. In coronary artery disease, prognostic factors can be as diverse as left ventricular function (26) and educational level (27). When attempting to determine if any one factor predicts outcome independently, it is important to be sure that this factor is not merely an indicator for another determinant of outcome. In our patients a number of social, demographic, clinical, hemodynamic, and anatomic characteristics showed no statistically significant disparity between deniers and nondeniers. Previous studies have found no correlation with age, sex, social class, previous coronary events, and clinical state (3, 4, 6, 10, 11, 15, 24, 28) but none appear to have assessed comparability of groups along *all* of these dimensions. These studies have also not adequately demonstrated that deniers and nondeniers had the same degree of baseline cardiac disease. They

are, therefore, unable to evaluate the possibility that deniers had a more favorable outcome because they were initially less sick. The two groups in our study showed no differences in measures of baseline coronary disease.

Vocational status was the only baseline parameter that was significantly different between the two groups, with a much higher proportion of the deniers working at the time of hospitalization. This is consistent with the denier's repudiation of his or her illness and its limitations.

We did find that deniers were much more rapidly stabilized than nondeniers, with cessation of angina significantly sooner. There are several possible explanations for this finding. The first is that although the two groups had similar extents of disease, the deniers stopped reporting symptoms much sooner in their hospitalization. It is well known that the reporting of pain is not necessarily a simple function of transient ischemia. Several studies have shown a marked discrepancy between ST segment shifts and the reporting of pain (29, 30). However, we noted no difference in the need for advanced therapy such as intravenous nitroglycerin between Group A and Group B patients. This implies that at least in the early stages of CCU therapy there was no difference in the communication of the need for more therapy between the groups. In order to fully answer the question of whether perception or reporting of symptoms changed more rapidly in the deniers, we would have to use a separate marker of transient ischemia, such as continuous ECG monitoring with ST segment analysis (31). This was not done in the present study, but is an important issue to pursue. If deniers fail to report ischemic symptoms, then long-term negative consequences may outweigh short-term gains.

A second explanation for the seemingly favorable effect of denial on medical stabilization would be a salutary affect of denial on the patient's adrenergic state. That is, deniers might have lower levels of circulating catecholamines, require less beta blockers and have lower blood pressures and heart rates. Although we did not measure catecholamines directly, our indirect clinical data on heart rate, blood pressure, and beta blocker dose did not suggest such a difference.

Lastly, denial may actually be an adaptive mechanism that favorably affects the course of illness through other mechanisms not detected by the simple parameters measured here.

This study was limited to the hospital phase of unstable angina, and the small size of our sample makes our conclusions cautious ones. We did not expect to detect differences in mortality rates with the small number of patients involved in a syndrome where in-hospital death is relatively infrequent. However, both deaths in our study occurred in nondeniers. While not statistically significant, this finding is consistent with two previous studies (3, 9). It is noteworthy that one of our patients who died had the lowest denial score recorded (Hackett-Cassem Rating Scale value of 1).

In conclusion, we found that denial was an independent predictor of rapid medical stabilization in unstable angina patients. While prior studies failed to exclude the possibility of marked clinical differences between deniers and nondeniers, our analysis of multiple social, demographic, clinical, hemodynamic, and anatomic variables in the study group strongly suggests that the highly significant association between denial with time to stabilization was an independent one. The mechanism for this association will require further study.

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J. L. LEVENSON et al.

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