we find a very different pattern. Here what we are interested in, though, is not whether a woman developed symptoms but whether she was able to get medical assistance before her symptoms overwhelmed her. And here the order of reporting to the doctor is quite significant. We find, for instance, that of the 25 affecteds who had ties to earlier cases, only 5 (20.0 percent) fainted. In contrast, of those 15 who had ties only to later cases, 8 (or 53.3 percent) fainted. This is almost exactly the proportion (50.0 percent) who fainted among the 16 who had no ties to any other affecteds. Thus, again we find that the difference between using one or the other of the possible dependent attributes discussed in Chapter 3 is considerable. If we use the emotional experience as our dependent attribute, the order of affected cases is not important, but if we use the behavior which follows from that experience (fainting or going to the doctor) the order affected is quite important.

A MULTIDIMENSIONAL MODEL

Throughout our analysis it has been apparent that no one of our variables is a completely successful predictor of a woman's becoming affected, even though the three different types of predictors (strain, personal characteristics, and social relations) have all proved to be effective. In several instances we have seen that combinations of these variables have proved more effective than the individual measures. The implication throughout has been that different kinds of people, under different kinds of strain, and with different patterns of social relations, respond differently. We should thus expect that some kind of multidimensional model would be most effective in organizing our findings.

If we are to attempt an overall statement of the contribution our analysis makes to an understanding of the dynamics of the epidemic, however, we must again recognize that we have to this point been dealing with only a part of the population of plant workers who were exposed to the experience of the epidemic. Although there seems to be good reason to leave the Negroes and those outside the dressmaking departments out of our analysis, it is also true that thus far we have not even dealt with all of those white women in the dressmaking departments. The fact that we have a sample of those

who did not report to the doctor plus the total population of those who received medical aid complicates the situation. This is particularly true since the self-defined affecteds have played such an important part in our analysis. Ideally, a summary statement should reflect the process occurring throughout the relevant population (i.e., white women in the dressmaking department). We must therefore make some assumptions about those women in the population whom we did not interview. The most reasonable assumption appears to be that our sample of nonaffected cases (controls plus self-defined affecteds) is representative of the population of nonaffected cases, and that the distribution of all characteristics within that sample is the same as the distribution within the population. We must also assume that we actually achieved a one-fourth sample of those nonaffected cases. Thus, whenever we examine the distribution of a particular characteristic, we need to multiply the data we have on the nonaffected cases by four and then add it to the data we have on the affected cases. This should then allow us to make the best estimate of the distribution of these characteristics within the total white female population in the dressmaking departments. Given such assumptions, we can analyze the distribution of the important characteristics within what can most legitimately be called the "population at risk."

In order to present in summary fashion the effect of the kinds of factors we have discussed earlier within the total population at risk, we selected the three most important measures from the earlier analysis and combined them. First there was the summary measure of strain which we viewed as an index of the situations in which our subjects found themselves. It will be recalled that this consists of four measures of strain: working a great deal of overtime, failing to mention the supervisor as one to go to with a complaint, seeing variation in output among the members of one's section, and providing half or more of the family income. This was then combined with the measure of the woman's position in the interpersonal influence process during the epidemic. Since we were going to use as our dependent measure the fact that a woman was or was not an affected case, we counted intimate ties with *any* other affected woman as an indication of a woman's social position. Such ties ranged in number from zero to three. Finally, within the categories defined by these

situational and sociometric measures, we differentiated between two kinds of persons, those who were and were not deniers by one or both of our measures. Since there were 5 possible levels of strain, 4 degrees of social linkage with affecteds, and 2 types of persons, this led to 40 possible combinations. Since some of the combinations did not appear and others were very infrequent, we reduced the number of categories by collapsing the strain and social position measures into 3 levels each. This provided the basis for an 18-cell table.

The data are reported in Table 7.5. The first number in each cell is the number of affecteds of that type, the second is the number of self-defined affecteds, and the third is the number of controls. Both of these latter two numbers were arrived at by multiplying the number of self-defined affecteds and controls of each type in our sample by four, thereby providing an estimate of the number of such women within the population at risk. The total number of cases in the table is 408.[7]

TABLE 7.5

Distribution of the Population at Risk Classified by Level of Strain, Ties with Affected Cases, and Denial

		Level of Strain		
		0–1	2	3–4
No Affected Intimates	No Denial	4-16-96	2-4-36	2-0-0**
	Denial	3-0-56	2-4-4*	3-0-0**
One Affected Intimate	No Denial	4-20-52	2-8-12*	4-0-4**
	Denial	3-0-4*	7-0-4**	4-0-4**
Two or More Affected Intimates	No Denial	3-4-0*	3-4-8*	2-8-0**
	Denial	3-0-0**	2-0-4**	3-0-0**

NOTE: The first number in each cell is the number of affecteds, the second is the number of self-defined affecteds, and the third is the number of controls. An "intimate" is a friend, one to whom one would go for advice or would expect to come to her for advice, a carpool or lunch partner, or one defined as the best worker in the section. "Denial" includes both the denial of symptoms and role-conflict denial. Cells with double asterisks designate those categories in which more affected cases are found. Those with single asterisks, together with those with double asterisks, designate those categories in which more affected and/or self-defined affected cases are found (see text).

If we wish to "predict" which cases were affected, using the three factors of strain, social influence, and denial, we might pick out cells containing 56 cases in such a way as to maximize the accuracy of prediction.[8] The cells with the double asterisks contain a total of 54 cases. To fill out the required number we might draw randomly 2 more cases from some adjacent cell. Since no such cell has half or more affecteds, we may assume that both cases so drawn would be nonaffecteds. How well does such a table order the cases? Perhaps most important is the fact that there is a simple pattern to be seen. First, there is a general tendency for the proportion of affecteds in the cells to increase as we move from upper left to lower right. All but one of the cells with double asterisks are in the last column or last row of the table. Second, either an extreme level of strain or close association with two or more other affecteds tends to lead a woman to be affected, irrespective of the other factors. Third, denial, when added to either social influence or strain, greatly increases the probability of being affected.

Also, it is striking that so many of the 56 cases we have just singled out are affecteds. Not only are 30 (or 53.6 percent) of them

[7] This number is much smaller than the 490 women in these departments listed in Table 1.1. Although some of the difference may be due to errors in sampling, most of it is due to three other facts: (1) the Negroes in the dressmaking departments are excluded in this analysis; (2) all supervisory personnel were excluded before the sample was taken; and (3) some of the women who were at the plant at the time of the epidemic were not there when the interviews were carried out (and their replacements were excluded from our analysis).

[8] This is not, of course, an actual case of prediction. In effect we are asking the following question: Given the fact that we know that 56 out of an estimated total of 408 women were affecteds, which combination of our three predictors permits us most accurately to identify the affected cases? To answer the question, we must find those cells (cells defined in terms of the predictors) which contain 56 cases and which include the maximum possible proportion of the affecteds. We are thus using the predictors to pick out 56 cases which have the highest probability of being affecteds.

affecteds, but of the remaining 352 cases, 326 (or 92.6 percent) are *not* affecteds. There is thus an overall accuracy of designation of 87.3 percent. This level of accuracy, of course, is far greater than could be accomplished using any of the three kinds of measures individually. Although it is clear that the strain and social relations measures predict well in the more extreme cases, the *combination* of variables is clearly important at the less extreme levels.

We may also ask whether such a table helps to single out the self-defined affecteds as well. Since there were 17 such cases in our sample, we can assume that there were 68 in the population at risk. These, plus the 56 affecteds, would give a total of 124 women who experienced symptoms during the epidemic. The cells with the single and double asterisks contain 115 cases. To fill out the required number, we could draw 9 cases randomly from the cell in the first row and second column, and we could assume that 1 of these 9 cases would be either affected or self-defined affected. Of these 124 cases, 72 (or 58.1 percent) are either affecteds or self-defined affecteds, and of the remaining 284 cases 232 (or 81.7 percent) are controls. There is thus an overall accuracy of designation of 74.5 percent.

The important part of these results, however, is the high proportion of affected (or self-defined affected) cases in the critical cells, since only a very small percentage of the total of 408 were either affected (13.8 percent) or self-defined affected (16.7 percent). Perhaps the best way to illustrate the effectiveness of the three independent attributes is to note that the cells in Table 7.5 which are marked with asterisks represent less than three-tenths (28.1 percent) of the population at risk, but they contain more than three-fourths (76.8 percent) of those who received medical treatment and more than half (57.3 percent) of all those who experienced symptoms. The three independent measures are thus much more effective in discriminating between those who did and did not go to the doctor than they are in discriminating between those who did and did not experience symptoms, although they do both to a significant extent. We will return to this matter in the next section.[9]

[9] The apparent effectiveness of our independent attributes makes it tempting to compute some kind of summary statistic to measure their effectiveness. We have not presented such a statistic because none appeared appropriate for our data. Multiple correlation assumes that the same equation applies (with minor deviations) to all cases, whereas we have found that different contributions are made by the critical characteristics under different conditions. Of the other possible techniques, those suggested by Coleman (1964, Ch. 6) appeared most promising. However, the cell frequencies in Table 7.5 are sometimes so small that some kind of reduction would be necessary. Whatever reduction is made tends to combine cells of the nondeniers which are quite different in their proportions affected, and this leads to a summary measure which suggests that only deniers contributed significantly to the epidemic. The fact that nondeniers tend to become affected only when strain and/or social influence is great (and also the cell frequencies are small) thus means that the various summary statistics we have tested seem to distort the results too much to be very useful.

THE PREDICTABLES AND THE DEVIANTS

Although the degree of accuracy of discrimination achieved is very striking, it is still true that a number of cases we might have expected to be affected were not, and other cases that were affected are found in cells that are not generally predictive of being affected. The most easily predicted affected cases (those who got medical treatment) are the deniers with at least one affected close associate. Of the 38 such cases in Table 7.5, 22 (57.9 percent) were affected. Those with high levels of strain (irrespective of other characteristics) are also very likely to have been affected: 18 of 34 such cases (52.9 percent) had been seen by the doctor.

When we turn to the misplaced cases we find, not surprisingly, that those nonaffected cases which are included in the cells having a high proportion of affecteds (those cells with the asterisks) are very likely to be self-defined affecteds. There are 72 nonaffected cases in the cells with the asterisks, and 28 of these are self-defined affecteds, the rest being controls. This represents 41.2 percent of the self-defined affecteds and only 19.7 percent of the controls. It is also important to see, however, that the rest of the self-defined affecteds are in the cells without asterisks. Also all but 4 of the 68 self-defined affecteds appear in the "no denial" rows. In fact, we have reported in Chapter 5 that none of the 17 self-defined affecteds in our sample was a symptom denier, and 10 of them were in the high symptom category. Because of the small number of such cases in our sample, we have until now tended to de-emphasize this fact. Now that we are examining the total population at risk, however, it assumes

greater importance. It gives further meaning to the pattern of deviant cases.

Since in our sample there were 17 affecteds, 15 controls, and no self-defined affecteds who denied all symptoms, we may assume that there were 17 out of a total of 77 symptom deniers in the total population at risk who experienced symptoms. In the high symptom category (those who exceeded the sample mean of one or more of the symptom scales by at least one standard deviation) there were 16 affecteds, 10 self-defined affecteds, and 26 controls in our sample. On the assumption that both affecteds and self-defined affecteds experienced symptoms, we may also assume that there were thus 56 out of 160 high scoring women who had symptoms during the epidemic. If this was actually the case, then a greater proportion of those with high symptom scores (35.0 percent) experienced symptoms than did those who were deniers (22.1 percent). Evidently, therefore, those with high symptom scores were more likely to "feel affected," but they were less likely to need or seek assistance, while deniers were less likely to experience symptoms but were more likely to require or seek medical aid if they did.[10]

This suggests with respect to Table 7.5 that many of the self-defined affecteds who are found in the cells without asterisks should be women who were in the high symptom category. This is actually

[10] The problems of interpretation here are considerable, however. Since the two definitions of being affected are so different, they bring into play to very different degrees the same characteristics we are attempting to interpret. Those who deny symptoms, for instance, presumably would be less likely to acknowledge during the interview that they were affected by the epidemic. In fact, four women who were known to the medical authorities as having been affected denied their involvement in the epidemic during the interview, and three of these are in our category of symptom deniers. We cannot say with any real confidence, therefore, that more of those with high scores *actually* felt affected during the epidemic. However, it seems highly likely that many more women than we know "felt affected," and presumably many more than those we have called affecteds actually "acted affected." This suggestion is further supported by the women's responses to the question: Did this happen to anyone (else) in the plant whom you know well? Although none of our controls was named in response to this question, 4 of the self-defined affecteds were named as well as 14 women in the dressmaking departments who were not in our sample. Since only 79 percent of the affected women were named, it seems likely that this represents a less than full accounting of those who in some way "acted affected," but it strongly suggests that others besides the affecteds were actively involved in the development and spread of the epidemic.

the case. Of the 40 self-defined affecteds in these cells, 32 had high symptom scores (on the assumption that the population at risk had the same proportions of such scores as the sample). It may well be, therefore, that the self-defined affected category is a mixed one, being composed in part of women with few predictive characteristics but a readiness to admit to symptoms and in part of women who had the predictive characteristics but who also had means of coping with the strain they experienced.

When we turn to the affecteds who seem to be deviants, we find 13 of them in the cells with no asterisks. In 9 of these cases the woman has one of the predictive attributes. Four have one affected intimate, 2 have two of the four sources of strain, and 3 are deniers. There are 4 cases, however, who were affected in spite of having none of the predictive characteristics. Although these must be viewed as evidence of the limitations of our predictors, it may be well worth mentioning that 3 of the 4 cases were affected quite late in the epidemic. They are cases numbered 40, 43, and 54. If we accept the notion that some kind of "crowd effect" was involved in the epidemic, it would certainly have been stronger if more cases had been already affected. We find, in fact, that of the 13 affected cases in these four cells, 7 were numbered 40 or higher. Seven of the last 17 and 4 of the last 11 cases are found in these cells.

The cases which seem to deviate from the pattern predicted by our three independent attributes, therefore, tend to be largely of three types. Those which are incorrectly predicted as affected cases are more frequently self-defined affecteds than one would expect if this were just random error. Those which are predicted as nonaffected but are self-defined affected more frequently have high symptom scores than one would expect if this were just random error. And those which are predicted as nonaffected but are actually affected are more frequently affected late in the epidemic than would be expected if this were random error. We may thus at least speculate that factors which are not included in the construction of Table 7.5 are involved here. For those cases which were predicted to be affected but were not, it may be that the self-defined affecteds' greater facilities for coping with strain are at least partly involved. For those cases which were predicted to be nonaffected but were self-defined affecteds, it may be that their readiness to acknowledge symptoms

was involved. And for those which were predicted to be nonaffected but were actually affected, it may be that a crowd effect late in the epidemic was operating. Although this can be nothing more than speculation, such an explanation fits well with both our other data and the conceptualization we have evolved of the process of hysterical contagion.

A GENERAL OVERVIEW

In this chapter we have followed several of the cues suggested in the earlier analysis, in each case attempting to bring together some of the parts of that earlier analysis into a more comprehensive and cohesive view of the epidemic. We have examined a number of connections among the findings and the presumed processes involved in the analysis of the earlier chapters.

Evidence of the distribution of the "two-edged" measures of strain among the affecteds tends to support the idea that such sources of strain both added to the woman's burden and deterred her from giving expression to the tension she experienced. Affecteds who experienced such sources of strain were more likely to be affected late in the epidemic. The effects of social ties with affecteds have been presumed to be in the direction of impelling a woman to believe in the threat of the insect and thus increasing the probability that she will also be affected. If we ask why self-defined affecteds and controls who had such social ties were not affected, the best answer offered by the data seems to be that these women generally had fewer sources of strain, were better able to cope with strain, and were less sensitive to interpersonal influence. Although the pattern of belief in the threat of the mysterious insect which our data provides cannot be used in our explanation of the epidemic, the data suggest that one's role in the epidemic is an important determinant of such beliefs afterwards. Those who became affected or were closely associated with someone who did were most likely to express complete belief in the bug. Those who were deniers (by either definition) were most likely to doubt the significance of the bug, unless they had themselves been affected. Among the affecteds, the most complete belief in the bug was expressed by those who fainted.

All of these findings add to the picture gained from the earlier analysis. But the most fruitful parts of the investigations reported here were those which examined the combined effects of two or more of the independent attributes on the probability that a woman would become affected or that, if affected, she would exhibit the most extreme form of response by fainting. With regard to the latter issue, we found that having social ties to others who were affected earlier decreased the probability that a woman would faint. Even more striking was the tendency for women with such ties not to faint if they were either nondeniers or had a high inclination to adopt the sick role.

When we attempt to identify the affected cases through the use of the several independent attributes, we are able to do so with a considerable degree of accuracy. Using three levels of strain, three levels of social relations with other affecteds, and the difference between deniers and nondeniers, it is possible to identify categories representing less than three-tenths of the total population at risk but which contain more than three-fourths of the cases treated by the doctor and well over half of those who experienced symptoms during the epidemic. Those cases which are misclassified in this procedure are largely self-defined affecteds who had the predictive attributes and affecteds who were affected late in the epidemic who did not have the predictive attributes. Also, those self-defined affecteds who did not have the predictive attributes tended to be women who very readily admitted to having symptoms. All of this analysis adds force to the argument that multiple factors must be considered if we are to account for the women's behavior during the epidemic and that different combinations of the same factors lead to very different outcomes.

Even though our data fail to provide a completely adequate explanation of the facts of the epidemic, the analysis presented in this and the previous chapters has indicated that there is more order in this seemingly chaotic situation than one might at first have expected. Before turning to the matter of a general conceptualization of hysterical contagion, therefore, it may be well to state in a more succinct summary form what we believe this analysis has done to clarify the order in the chaos. We will do this through a set of statements, each

of which will be followed by a brief review of the relevant data. (In reporting the data, all frequencies and percentages are based on our estimate of the assumed population at risk.)

Most affecteds were exposed to a great deal of strain, or were deniers or both. Although we have suggested that various combinations of these factors, plus social relations with other affecteds, are predictive of an affected status, this is the simplest general statement that can be made about the affecteds. Almost two-thirds (66 percent) of the affected cases have these characteristics. In contrast, only slightly more than one-fourth (27 percent) of the nonaffecteds have the same characteristics. Only 18 percent of the self-defined affecteds had these characteristics, compared with 30 percent of the controls. Thus, those qualities which most clearly distinguish the affecteds from the nonaffecteds are particularly successful in distinguishing them from the self-defined affecteds. When social relations with affecteds are used as a predictive attribute, greater distinction is seen between the controls and the affecteds, but the line between affecteds and self-defined affecteds becomes less clear.

The difference between affecteds and self-defined affecteds was largely due to differences in personal qualities and in sources of strain. In general, the affecteds acknowledged symptoms much less readily and more frequently denied the importance of role conflict. The self-defined affecteds also were exposed to less strain than the affecteds, and the strain they did experience was more likely to be of the "two-edged" type which generally tended to delay the woman's becoming affected.

The difference between self-defined affecteds and controls was largely a difference in social relations and personal qualities. The self-defined affecteds were more likely to have close social ties with affecteds, but they much more easily acknowledged symptoms. The controls were more like the affecteds in their denial of symptoms, but they less frequently were socially linked with affecteds. If we view social ties, strain, and denial as forces leading a woman to become affected, it is apparent that the self-defined affecteds had more of the first, the controls had more of the last, and neither of them had a great deal of the second.

If the strain experienced was sufficiently great, it alone was likely to lead a woman to become affected during the epidemic. Not only were several of our measures of strain (such as working a great deal of overtime) clearly related to the tendency to be affected, but our summary index using four such measures showed that more than one-half of those in the population at risk who were under heavy strain became affected compared with less than one-tenth of those under little or no strain. There is a general pattern of increasing probability of being affected as the level of strain increases.

If social influence was strong enough, it alone was likely to lead a woman to become affected. There were 44 women who had two or more social ties with affecteds and 16 (36 percent) of them were affected cases. In contrast, only 7 percent of the 232 women who had no ties with affecteds became affected. More generally, the more ties a woman had with affecteds the more likely she was to become affected herself.

Among those affecteds with social ties with other affecteds, the ones who became affected first exhibited the most extreme symptoms. Although social ties with any other affected increased the probability that a woman would become affected, if she became affected before her associate, she was much more likely to faint. Of linked pairs of affecteds, over one-half of the earlier affected members of the dyads fainted, but only one-fifth of the later affected members fainted.

The combination of denial and a moderate level of strain or social influence greatly increased the tendency to become affected. Those who were deniers but experienced low levels of strain and no social influence were not very likely to become affected—only 5 percent did so. But deniers who had two sources of strain and/or one affected associate were very likely to become affected themselves—43 percent did so. This is in contrast to those nondeniers who had two sources of strain and/or one affected associate, only 5 percent of whom were affected. There is thus a kind of interactive effect of social or situational influence with the personal characteristic of denial to produce a high probability of becoming affected.

Personal and social characteristics interact to affect the tendency to exhibit the most extreme symptoms. Those affecteds who were nondeniers and who had ties with someone affected before them, were very unlikely to faint, less than one-tenth doing so. In contrast, over

one-half of nondeniers without such ties fainted. Among deniers, the presence of ties with earlier affecteds did not influence the tendency to faint.

There were many persons who played an important part in the development and spread of the contagion who were not known by the authorities to have been affected. It is difficult to demonstrate this statement as convincingly as the others, but it seems very likely that within the population of women in the dressmaking departments there were at least four times the number of women as in our sample who would have been self-defined affecteds if we had been able to interview them. If so, there would have been more of them (68) than affecteds (56). Since they were likely to acknowledge symptoms rather easily, it seems almost certain that they were influential in convincing many other women that there was a reason to be fearful, and thus they contributed significantly to the epidemic.

8

THEME AND VARIATIONS

In this chapter we will return to a more abstract level of theoretical statement in an effort to present in general terms those propositions which our analysis seems to justify. In doing this, we will attempt both to remain consistent with the data from this single study and to conceptualize this case in sufficiently general terms to make our discussion potentially applicable beyond the limits of the single case. Since the data we have are limited to a single case, and since all such cases are certain to have their own idiosyncrasies, some of our data are judged to be less relevant to the more general discussion, and some of that discussion will necessarily go beyond the available data.

THE GENERAL PERSPECTIVE

In Chapter 2 we discussed a number of earlier conceptualizations of hysterical contagion and closely related types of collective behavior. We approached this particular case with a number of general expectations. The epidemic was viewed as an expression of frustration and a reflection of the inadequate structural characteristics of the work situation. It was argued that in such a situation as existed

in Montana Mills prior to the epidemic there were a number of people subjected to many sources of strain which they found frustrating and from which there was no simple, socially legitimized means of escape. In such situations, individuals become tense and, presumably, they tend to reinforce each other's sense of frustration and tension.

There is undoubtedly a wide range of possible outcomes from such tension-filled collective situations. In some cases, there might simply be a selective elimination from the situation of those who are least able to cope with the tension. This could be on a permanent basis through resignation or being fired or on a temporary basis through absenteeism and "illness." In other cases, the source(s) of the strain might be temporarily limited and, after a period of extreme tension, a period of relaxation of strain might occur which would permit a reduction of the tension. In still others, the individuals in the situation might band together in some way to combat the strain and/or to release the resulting tension in socially approved or socially disapproved ways. Thus, the outcome will depend on a number of things including the structure of the situation within which the strain is experienced, the kinds of individuals experiencing the strain, the potential for collective as opposed to individual reactions, and the possible occurrence of a number of random events.

In order for the kind of epidemic we have studied to occur, there must be provided, from some origin, a suggestion that the situation possesses a threatening element whose potency is high and whose "behavior" is mysterious and unpredictable. That is, the threat must be seen as one which is genuinely frightening (to the extent it is believed), and the means of avoiding the threat must be seen as very limited and/or undefinable. If the threat is not seen as very serious, the added tension due to its presence will not be appreciable; and if there are easy ways of avoiding the threat, it thereby becomes much less threatening.

The particular kind of threat which will meet these criteria in any given situation can undoubtedly be highly variable. The most effective kind, however, will be one which is easily accepted as genuine. Although almost *any* kind of threat *might* have provided the basis for such an epidemic of symptoms (e.g., a vengeful spirit

whose "home" was disturbed by the building of the plant), the credibility of some kinds of threat will be greater than others. This credibility will be a function of general cultural norms and the specifics of the situation in which the collectivity is located. In our case, the threat of a poisonous insect was rather easily accepted since such insects are known in the area (e.g., the black widow spider). Also, the foreign origin of some of the machinery and materials used in the plant made it possible that some similar but unfamiliar insect might have been shipped in (like the tarantula spider in the bananas) and thus might pose a threat which was located only in the plant. We cannot say that belief in a threat of this kind *had* to develop, of course, but only that it was easier for it than for other possible beliefs to develop in this situation.

It may well be that the threat is originally completely "invented" by one or more persons in the situation as a need-fulfilling element. That is, there may be no manifest evidence of the threatening element at all from the point of view of the outside observer. It may simply be a need-fulfilling hallucination. On the other hand, the element may be manifestly present, even though its potency may be less than fully evident. That was apparently the case with the Montana Mills epidemic. Certainly there were insects in the plant. Even after several sprayings some were found, and there must have been more before the sprayings. The significant "invention" here was the effect of the insect, its potency. In such a case, it is much easier to evolve a belief in the threat since it is both common in form and possible in its effect. Belief in this threat seemed reasonable not only to the women who worked in the plant but also to management and a number of outside specialists who found it sufficiently credible to warrant serious investigation. This type of response on the part of outsiders tends to reinforce the belief and to make the threat appear even more genuine.

However, a connection between the stressful situation and the newly invented external source of potent threat is required. Smelser (1963) suggests that the *function* of belief in such a threat is to give substance to the feeling of frustration and anxiety. Instead of simply having a feeling that something is awry, the belief in a tangible threat makes it possible to *explain* and *justify* one's sense of discom-

fort—instead of anxiety, one experiences fear, and it is then possible to act in some meaningful way with respect to this tangible threat rather than just feeling frustrated and anxious. The belief in the threat, then, solves the problem of objectifying the source of the discomfort.

The crucial point here, however, is that, for some reason the *actual* source of discomfort is not (or cannot be) clearly recognized by at least some of those in the situation. In our case, the principal presumed source of the frustration and tension was the strain on the job and the conflict between home and job responsibilities. The women were tired and torn between conflicting demands. But, why did they not simply face their problems and cope with them directly? What possible connection could there be between working overtime and being bitten by a poisonous insect? Basically, this is a question of identifying the alternative modes of behavior in the situation. Given the stress, what could the women do about it which would have constituted coping directly with the problem? They might have simply stayed home and rested up. They might have refused to work overtime. They might have complained to their supervisor and asked her to relieve them of this burden. They might have used the union as a basis for bargaining with management for a better work load. We have found that, for at least some of the women, each of these various modes of direct coping posed serious difficulties. They were either ineffectual or they were unavailable to some of the women for various reasons.

In the face of such difficulties, the original strain remained, and the fact of impotence in coping with it provided an additional source of stress. Also, the difficulties involved in coping create one or more additional sources of strain. If the supervisor is not trusted, the most "rational" thing to do is to treat her as if she is not trustworthy. Yet a worker would find such a reaction very threatening, and the necessity of controlling one's distrustful tendencies becomes an additional burden. Likewise, if having a small child makes the life of a working woman more stressful, it might be seen as "natural" or "reasonable" to reject either the job or the child. But if both of these are important to the woman, the very thought of either type of rejection is threatening. One dare not even *think* about being unhappy

with the job or the child. In this way, each of the sources of strain and each of the barriers to coping with the strain places the woman in a position of compounded stress.

But, why does getting bitten by a poisonous insect enter the picture at all? At the most simple level of functional analysis, we have said that it objectifies the source of discomfort and helps solve the problem by providing the woman with a legitimate and credible reason for leaving the situation. For some women, the simple existence of this threat provided an acceptable reason for "clocking out of there." For others, it was necessary to get sick before leaving could be justified, but once they got sick even management condoned their leaving, and the insect made it "easier" to get sick. Not only did the insect provide a legitimate reason for leaving the plant and getting the needed rest, it also provided a means of "striking back" at management. From this point of view, the epidemic could be seen as a kind of "psychological strike." It is not likely, of course, that it was consciously viewed in this way by any of the women, but the insect did legitimize leaving the plant, and it did cause management a great deal of difficulty.

This functional view of the epidemic is only one side of the picture. It is equally important to see the close relationship between the symptoms one might expect to develop under the frustrating conditions and those symptoms which were attributed to the toxic insect. As stated by Engel, when a person is faced with psychological stress with which he cannot adequately cope, he develops both a cognitive sense of distress and "the awareness of physiological changes such as palpitation, sweating, flushing, muscle tension or 'butterflies in the stomach'" (Engel, 1962a, p. 384). Such symptoms are general emotional responses, and it is but one small step from the experience of such symptoms to the definition of them as due to a much-discussed toxic insect. In fact, it is reasonable to postulate a need to identify and label (to understand) such symptoms, and we know that similar physiological experiences are capable of very different interpretations. This is where the "generalized belief" becomes significant. It is at this point that those who, for whatever reason, do not accurately attribute their symptoms to the stress they are experiencing accept the newly evolved explanation. In our particular case,

the newly evolved explanation was ideal in that such an insect could easily be accepted as the cause of the kinds of symptoms the women experienced.

Our general view of such cases as we have investigated here, therefore, may be summarized briefly as follows: there are a number of people exposed to common sources of strain from which there are available but not easily acceptable avenues of escape. The combination of the original strain and the added stress experienced because of available but forbidden solutions brings about a general state of tension whose intensity is probably increased by interstimulation among the people so situated. The state of unresolved tension leads to the experience of physiological symptoms. These symptoms become associated in the minds of the distressed people with some credible (but factually incorrect) "cause" in the situation, the connection deriving from what are probably random events that become interpreted in the context of the experienced discomfort. This external cause both objectifies the source of the discomfort and adds a further source of strain to the situation—fear of a threatening force. Given this evolved credible explanation of symptoms, various means of coping with the (newly found) threatening force become legitimate, and those who were unable to cope with the original sources of strain are provided with acceptable forms of response not originally available to them (e.g., staying away from the plant). Another acceptable form of response, under the new definition of the situation, may be to acknowledge one's inability to handle the problem and to seek assistance from outside experts (usually, as in our case, medical experts) who are capable of dealing with the symptoms. Whatever means of coping are deemed legitimate (and legitimacy is undoubtedly a function of both the participants' and outsiders' definitions) will become socially facilitated, and the probability of any given participant's use of these means will tend to increase as the epidemic runs its course. The manifest phenomena of the epidemic are thus the reports of the external threat, the physiological symptoms (which purport to be the result of the external threat), and the coping behaviors of the participants (and various relevant outsiders).

The central problem faced in this study, however, goes beyond this general conceptualization. In fact, it actually takes this conceptualization for granted and asks the further question: Given such a development, *which* persons in the situation will be most active in the epidemic? We have operationally put this question in the following form: Which persons will seek the aid or behave in such a way as to come to the attention of outside (medical) experts? To deal with this question, we have had to go beyond the general conceptualization just presented, but we have also had to remain within that general framework. We have consequently raised more refined questions about variations in the experiences and characteristics of the persons found in the stressful situation. The basic logic involved has been that, if a particular combination of circumstances is likely to lead to a case of hysterical contagion, those who become involved in such a collective behavioral expression should be more likely to exhibit this combination of circumstances than should those who are not affected. We have defined the conditions which are likely to form the basis of hysterical contagion as being the collective experience of strain which cannot be adequately coped with occurring in a situation where interpersonal influence is possible. We have thus sought evidence of variations in strain, variations in personal qualities associated with effective coping with strain, and variations in social position.

VARIATIONS IN SITUATIONAL STRAIN

Many discussions of collective behavior approach the subject as if all of the participants (or potential participants) were responding to the same situational factors. There is considerable gain in such an assumption from the point of view of conceptual parsimony. In most cases, it is possible to point up salient features of the action setting which are presumably relevant to the behavior of all persons in the situation and which are related to the kind of collective phenomenon being studied. Such general factors were undoubtedly important in the development of the case we studied. But, being general factors, they cannot be used to explain why some people were affected by the epidemic and others were not.

When one looks for situational factors which might differentiate between those affected and those not affected, several possibilities

come to mind. One of these, and the most obvious one in our particular case, is the fact that all of the potential participants were not and could not be physically located in exactly the same place. Studies of crowd behavior have made a point of differentiating between those in the center and those on the periphery of the crowd and have noted that the experience of the situation is quite different depending on such differences of placement. In a more structured situation, such as the one we studied, there are other structural bases of differentiation, such as the departmental organization of the plant and the fact that the dressmaking departments were in a separate area marked off by walls. This factor was so clearly relevant that we did not even include in this analysis (except in the Appendix) any of those persons located outside the dressmaking departments. The fact that working conditions, and thus the level of strain, were so different in these departments made such exclusion very appropriate.

In addition to this kind of spatial and organizational differentiation, prior knowledge of the distribution of cases and of cultural factors made further limitation of our analysis possible. We knew that very few men were involved in the epidemic. Both the absolute number of male affected cases (three) and the percentage of the male work force affected (1.0 percent) were so small, we immediately ruled out their inclusion in the study.

The other categorical difference we have used in this report represents a type that is not easily generalized to other similar situations. This is the elimination in the present discussion of the Negro women in the dressmaking departments. These women were in the same big room with the majority of the affected cases, and two of them in fact were affected. We have separated them from the others in those departments in this report as a matter of analytic convenience as well as for conceptual reasons. However, the data support the view that they were sufficiently different and so clearly socially segregated from the others that, although they were spatially involved in the activity of the epidemic, they were much less socially involved. To the extent that social relations and processes of identification are significant in the dissemination of hysterical symptoms, such people are much less "in the situation" than are others in the same room.

Whatever the justification of the separation in this case, however, the more general point is that cultural factors may differentiate between kinds of persons all of whom are physically in the same situation. To the extent that this is true, some structural features of the situation which are stressful for some persons may not be for others. Although there may be good reason to expect such cultural differences in the significance of situational strain, however, our ability to predict their significance in any given situation is probably so limited that it is safer to make the analytic differentiation only after the data are in hand. This, of course, is what we did with respect to the Negro women in the dressmaking departments.

Such categorical bases of differentiation are important in understanding the reasons for different levels of participation in such events, but we also know that other, more individual, factors are similarly relevant. Not only are the situational sources of strain unevenly distributed among those in the situation, but each of the actors finds himself in a total action context, only one part of which is represented by the situation in which the collective behavior occurs. The situational context of collective behavior is therefore a point of intersection of the total behavioral contexts of all of the actors. It is thus only one part of the total relevant context for any given actor. Because of the immediacy of that setting, the force of the collectively evolved definition of the situation, and the pressure of the feared threat, it is an immensely important part of the total context, but all of these characteristics of the immediate situation will be responded to in light of the total situation of the actor. And, both as a function of these different bases of response and as a result of varying locations in the action setting, each actor's perspective is likely to be somewhat unique.

It is not possible in such a study as this to take into account all of the potential sources of variation resulting from these kinds of differences. What we have done here rather crudely is to focus on two sources of variation in levels of strain. One of these was variation in sources of strain on the job, the other was variation in sources of strain off the job that might have carry-over relevance to the work situation. More generally, this would call for an examination of variation in sources of strain in the immediate action situation and

variation in sources of strain outside that situation which would presumably be relevant to the individual's behavior in that situation. Since the latter kinds of sources of strain are theoretically unlimited, we have chosen to focus on those which are both structurally common in the population of actors and known to have direct significance in the action setting (i.e., family relations and role-conflict problems). Of course, the sources of strain in the action situation are also potentially very numerous. We have distinguished among those sources which are interpersonal (relations with peers and with superiors), those which are personal (ability to do the work tasks), and those which are structural (requirement to work overtime). However, we have done a better job of dealing with the first and third than with the second of these.

The general point to be made in this section, then, is that even those persons who are in the same action situation are differentially subject to sources of strain. The variations involved come both from their varying positions (spatially and socially) in the action setting and from their varying involvements in other action settings outside the immediate one. The general hypothesis relevant to these variations, and one which proved fruitful in the present study, is that those who face a greater number and more intense sources of strain have a greater probability of being affected by the hysterical contagion. This is to be expected because the greater the strain, the greater the probability that the individual will be unable to cope satisfactorily with it, and thus the greater the probability that physiological symptoms will occur which may be interpreted in terms of the belief in an external source of attack. Even though our catalogue of sources of strain was certainly far from complete, one of the clearest patterns to emerge from the analysis was the increasing probability of becoming affected by the epidemic as the number of sources and the intensity of strain increased.

THE PERSON AND COPING MECHANISMS

If the general conceptualization we have followed is to be accepted, it is obvious that there should be other factors involved in determining the distribution of affected cases besides the level of strain experienced by the actors. One of these factors should be the

characteristics of the actor and the resources he has to cope with the strain he experiences. Situational strain is not as likely to bring about psychological stress and the disturbed affective states and somatic symptoms associated with such stress if the problem posed by the strain is adequately dealt with by the actor. Thus, the characteristics of the actor and the coping mechanisms he has at his disposal are certainly important in determining the ultimate psychological (and physiological) effect of any given level of strain he experiences.

For the women at Montana Mills, most of the kinds of strain experienced on the job were such that direct "rational" coping was limited to a few mechanisms. The women could leave work and pretend to be sick, they could complain to their supervisor, they could refuse to work overtime, and so on. In general, these mechanisms were either morally unacceptable to many of the women (if we are to believe their verbal reports) or they were threatening to them because of the importance of the job and the fear of losing it. Those who were most clearly affected by the epidemic were more likely to reject such direct coping mechanisms. They were thus not only left with the original strain but they presumably had the added strain of knowing that these mechanisms could be (and probably were) used by others.

Although such norms and attitudes are certainly qualities of the individual actors, they are more specific to the situation than others which we have considered to be relevant. We assumed that the women would vary in personality structure and that such variation would be significant in understanding the distribution of affected cases. Our original expectation was that those women who were generally more anxious and more concerned about physiological disturbances would be more likely to be affected (particularly since "being affected" meant going to the doctor), and we expected this concern to be reflected in their admission that they experienced a number of physical symptoms. We found, on the contrary, that those who were affected were much more likely to *deny* having any of the symptoms we asked them about than they were to admit to a large number of symptoms. In fact, there were so many affected women who denied having *any* of these symptoms that this extreme denial was viewed as potentially significant in the dynamics of the epidemic.

We interpreted the fact that symptom deniers were likely to be affected as being due to the greater significance for such women of symptoms experienced during the epidemic. That is, it seems reasonable, with the wisdom of hindsight, that if a woman takes some pride in not being subject to physical disturbances (or is for some reason threatened by a conscious admission of such disturbances), she will respond in a more exaggerated manner to an undeniable experience of physical symptoms (e.g., by fainting) than will a woman who easily accepts such symptoms as part of her normal life. Also, one who denies the reality of symptoms is not likely to cope with them very effectively. By the same token, we would expect that such women, who normally deny symptoms, would more easily accept some external explanation for these symptoms.

Here again we must keep in mind that our original definition of "being affected" was based on a woman's being known to the medical authorities. Given that definition, it is true that those who normally denied symptoms were more likely to be affected than were those who readily admitted to such symptoms. However, if we base our definition of "being affected" on a woman's admission in the interview that she had been affected by the epidemic ("Did anything like this happen to you?"), we get a picture much more like our original expectation. Many more women admitted being affected than ever became known to the medical authorities, and most of those who said they were affected but who were not known to the medical authorities also readily admitted to having symptoms. Thus, it is not unreasonable to assume that actually there were more who experienced symptoms during the epidemic among the high symptom women than among the symptom deniers. It also seems very reasonable to assume that they played a very important part in the development and spread of the epidemic.

Relevant here also is the contrast between what some of the affected women actually experienced and what they said about their experience. In some cases there seemed to be evidence of denial of the significance of external sources of strain. We followed this lead most systematically with respect to the denial of the significance of role conflict, and again such denial was associated with becoming an affected case. In fact, such role-conflict deniers were more likely than any others to faint during the epidemic. In some respects, this kind of denial may be viewed as the opposite of symptom denial. In

the one case the respondent denies the significance of external forces; in the other she denies the existence of internal experiences.[1]

Although our measure of role-conflict denial is far from a convincing measure of a "tendency to deny the significance of external forces," the data from our study do support this view. Those who denied the significance of role conflict were very likely to become affected, and they were also very likely to faint if they did become affected. At the same time, both symptom deniers and role-conflict deniers who were not affected were more likely than any others to question the exclusive role of an insect in causing the epidemic. This corresponds to our interpretation of these women as resistant to admitting that they had problems and thus as being incapable of coping with them.

Our analysis of the personal characteristics of the subjects suggests, then, that such characteristics will "make a difference" in the probability of a person's becoming an active participant in such an epidemic, but that the characteristics which will increase the probability will depend upon the nature of the epidemic and what "part" in it we study. Since all epidemics of hysterical contagion involve the experience of physiological symptoms which are interpreted as being the result of an external threat, both symptom deniers and "external force deniers" should have a high probability of becoming active participants and of being seriously affected by the epidemic. Those

[1] In both cases, of course, we have had to *assume* that these factors were "really" more important than the women were willing to admit. We might therefore expect that a woman who denies the significance of external forces would find it difficult to accept the explanation that the physiological symptoms which developed during the epidemic were caused by an insect or any other external source. Thus, when she experienced such symptoms, she would be more likely than the other women to feel internally threatened, to think that something was drastically wrong with *her* rather than with her environment. Or she would at least be forced to admit that there really was something wrong with her environment. In either event, the experience would undoubtedly be a very upsetting one. Another interpretation of the role-conflict deniers is also possible. It may be that these women actually *did* prefer to avoid their responsibilities at home, that they welcomed the chance to escape their children and other familial obligations. If this were the case, one would expect that their real problem lay in the strain between their personal preferences and cultural expectations of devoted motherhood. It is not possible, of course, with the data in hand to determine the validity of either of these interpretations. We can only point out that the verbal responses and the actual situation of the role-conflict deniers are inconsistent in the context of our culture's role definitions.

who easily acknowledge symptoms, on the other hand, should be readily affected, but they should not be so seriously upset by the experience. To the extent that the accessibility of coping mechanisms (such as being willing to leave the scene of the threat) is a function of personal characteristics, such characteristics of the individuals should also influence who becomes affected. Thus, both the ability to recognize a problem and the ability to cope with it enter the picture. Finally, since such events are examples of *collective* behavior, the sensitivity of the person to suggestion from others should contribute to the probability of his being affected by the contagion.

SOCIAL RELATIONS AND CONTAGION

The term "contagion" clearly implies that the hysteria moves from one individual to another, and all discussions of this phenomenon emphasize the importance of interpersonal influence and communication. Our investigation indicates that the role of social relations in such epidemics is much more complex than the previous literature would seem to suggest. We have found that in the initial stages of the epidemic social isolates were most active and seemed to supply the basic new idea that an external threat was causing physiological symptoms. Only after the impetus was supplied by these isolates did the role of social relations and interpersonal influence become manifest. During the major portion of the epidemic, the period in which the symptoms spread most rapidly, intimate relations among the participants appeared to play an important role. Finally, there is the suggestion that if such an epidemic continues long enough interpersonal ties are no longer as significant, presumably because the reality of the threat is validated by such a large number of cases and the method(s) of responding to the threat becomes legitimized through widespread use.

In our case, we found that five out of six of the earliest victims were social isolates. Several of them had a history of "nervousness" and fainting. They were generally dissatisfied with their jobs and their role as workers. By comparison, those affected during the major portion of the epidemic were both socially and personally more secure. The epidemic spread through networks of friends and close associates, the networks in many cases consisting of women in the same

work groups. Beginning sometime during the second big day of the epidemic, however, and continuing through the rest of the period, such social ties appear to be much less significant. There are fewer who have ties with other affected cases during this period in spite of the fact that after so many cases had appeared there was a higher probability that any case would be linked with another one.

We also found that when we were considering whether a woman became affected or not the order of the cases made little difference. Being linked with *any* affected case was as predictive of becoming affected as being linked with an *earlier* case. We interpreted this to mean that the process of becoming affected was not identical with the process of reporting to the doctor. Evidently two or more women could influence each other in the direction of believing in the insect threat and interpreting symptoms as due to the insect without necessarily becoming "stricken" at the same time. Influence of this kind did not necessarily depend on one of the women's going to the doctor. However, we did find that the seriousness of the symptoms exhibited varied with the order of the cases. If a woman were the first one of a network of close associates to become stricken, she was more likely to faint, whereas later cases in the network reported to the doctor before fainting. Evidently the experience of the first case in the network was enough to convince the women that the threat was really serious and that medical aid was necessary.

We have interpreted our findings as indicating that the social context of contagion is significant in at least two different ways. First, it operates as the medium of influence with respect to establishing the veridicality of the external threat. Those who know someone who has gotten sick (or even who believes in the bug) are likely to believe that a real threat exists. Second, it acts as the basis for defining the appropriate response for those who feel the influence of the symptoms. It also seems true, although it is more difficult to demonstrate, that these contributions of the social context occur in two parallel ways. They occur as a result of direct interpersonal influence, and they occur as a result of the force of a more general social validation. In the first case, it is the fact that one or more of a woman's close associates is affected that leads her to believe in the reality of the threat and to interpret her symptoms accordingly. In the second case, it is the fact that so many others have been affected

that leads to this effect. The distinction here is thus between intensity and extensity of social influence. This is not an either/or distinction, of course, but it helps to emphasize the fact that both dimensions seem to be significant.

The effect of social influence on the response made by the individual who experiences symptoms in this setting can presumably be one of many different kinds. We have emphasized the effect in terms of increasing the probability of the woman's feeling sufficiently upset to faint or to seek medical attention, which has been our basic definition of being affected. Affecteds with ties to earlier cases were much less likely to faint, for instance. Their ties with other affecteds presumably both increased their chances of becoming affected and increased their tendency to seek help before it was too late. However, it is equally likely that social influence operated in the opposite manner in many cases. If one were almost wholly in contact with those who had *not* become sick and those who did *not* believe in the validity of the external threat, presumably this association would reduce the probability of one's becoming sick. Also, if one's associates had evolved a different definition of the proper response to the experience of symptoms (such as to "clock out of there"), even if a woman got sick she would be less likely to seek medical help or to stay in the situation until she fainted. It seems likely that this was the case with many of the self-defined affecteds. Although our focus of attention has been on processes leading to a medical report, clearly there were other processes leading to other outcomes also occurring at the same time.

Perhaps the most important generalization to suggest on the basis of this analysis is that social relations supply the medium through which the processual aspects of such an epidemic operate. Social relations act not only as channels of communication and interpersonal influence but also as mechanisms of collective definition and decision making. It is, of course, this dynamic aspect of the epidemic which is most difficult to illuminate directly. We cannot unequivocally demonstrate that the social relations actually functioned in the way we have suggested, but it is impressive that so much of the evidence points in this direction. Not only is the *fact* of being affected related to the pattern of social ties, but the *response* to this fact (fainting or not) and the *interpretation* of the experience (belief in the causal significance of the insect) are also related to this pattern. The orderliness of this pattern of findings is probably the most significant part of the study.

PROBLEMS AND PROSPECTS

Some of the limitations of the present study result from the fact that it occurred so long after the event in question. The delay was in part a function of the peculiar characteristics of the situation we were investigating. Since we had to carry out our interviews in the plant for the most part, we needed the cooperation of management, and this cooperation was granted with the proviso that a time lag occur. However, some of this delay would have been necessary in any case. We had to prepare a research instrument and get it printed; we had to draw a sample and set up interviewing procedures; we had to obtain trained interviewers. Even without management's restriction, therefore, some lag would have occurred.

We may assume, therefore, that no matter how well-equipped or well-financed we might have been there would have been a sizeable time lag between the time the epidemic came to our attention and the completion of our data collection. The alternative would have been to send in a limited number of investigators immediately without a uniform interview schedule or a well-defined sample so that they could gain some general impressions. Even then, the process *during* the epidemic could have been viewed only in retrospect. It may well be, though, that a combination of these two approaches would have been the best arrangement if it had been possible.[2]

When one considers these alternatives, or the combination of both approaches, however, another factor must be taken into account: the effect that the data collection itself has on the situation being investigated. We were required by management to limit our

[2] It is our impression that the alternatives noted here are the only ones available and that, of the two, the one we chose is to be preferred if only one can be used. One of the basic inadequacies of the considerable work that has been done in the area of disaster research is the necessarily hurried and nonsystematic way in which the data have been collected. It has led to some interesting insights into human behavior during such disruptive events, but it has not contributed very much systematic information upon which generalizations may be based.

questions about the epidemic to an absolute minimum, and we even presented these questions as if they were an aside rather than a central concern of the interview. Though we had to do this under the circumstances, it is likely that we would have done something similar to this in any event. One of the problems faced by anyone investigating such an event is the fact that it is a highly significant event in the lives of the participants. They are very much involved with the experience and its aftermath. Any suggestion that the interview was intended to find out why some became affected and others did not would have been threatening to many of the women.

We cannot be sure, of course, that we did not give this impression, but there was little evidence that we did. In fact, most of the evidence points the other way. There were only 4 women who had reported sick during the epidemic who did not acknowledge this fact in the interview. Of the remainder, two-thirds acknowledged their own participation when they were asked for only a general description of the incident, and the remainder acknowledged after a simple probe. On the other hand, 17 women, about whom we had no record, mentioned their connection (the self-defined affecteds), about one-third of them without even being asked directly. This would indicate, if anything, an attempt in the interview of trying to be included but not of being defensive.

We had one other check on the accuracy of the reports. We asked the respondents whether they knew of anybody else who had been affected. Within the population at risk, all persons who were so identified, and who fell into our sample, mentioned themselves as affected.

Thus, our evidence would show that the women did not feel ashamed of their participation in the epidemic, but perhaps acknowledged it too freely. We can learn from our experience that it is possible to overcome resentment and suspicions but also that one should not assume how labeling an epidemic as hysteria might affect answers. Leaving a time lapse after the event and imbedding the questions in a general context helped to dissipate suspicion; further, it was important that we had some cross-checks to evaluate their answers. It was not obvious, however, how defensiveness would affect the answers. Respondents could deny the event, but this did not seem to have happened. They could also insist on the reality of the

epidemic in spite of the labeling of the epidemic as "hysterical" by the mass media. There is a possibility of this having happened as indicated by the self-defined affecteds and the pattern of lasting belief. However, both these phenomena assume relevance in relation to other data. The epidemic seen from a vantage point of two months later, even with possible attending distortions, can be explained within the framework which we have studied.

We would conclude that events of this kind can be studied in a retrospective survey technique, with proper cautions in organization of the study and interviewing as well as in providing possibilities of cross-checks and care in data analysis.

Closely related to these issues is the effect of the experience during the epidemic on the measures made. To what extent does the fact of that experience change the conditions which the analysis assumes to be the "independent attributes"? For instance, is it more reasonable to say that women who were socially related to each other were more likely to become affected, or that women who were affected *became* socially related to each other? Short of omniscience, there seems no way to collect the relevant data before the occurrence of the hysterical contagion, and thus this problem appears to be inherent in such research. One can only attempt to insure against obtaining measures that are contaminated in this way. The most vulnerable of our major independent attributes were probably the sociometric and personal attribute measures. The latter are probably necessarily vulnerable if we assume that such an experience can appreciably alter the personalities of the victims. We rather doubt that this would be a common outcome, however, and would suggest that measures which deal with the more stable continuing qualities of the person should be subject to little criticism. Undoubtedly our own measures could have been improved in this regard. With respect to sociometric measures, the crucial question is whether the relationships existed before the incident. Although we did not have such a safeguard, it would be possible to include questions about how long one had had such a relationship with persons named. Such safeguards would increase one's faith in the findings, but it is important to acknowledge that this temporal problem is a necessary part of such studies.

There were other aspects of the research which we recognize as

less than ideal and which could be done more effectively in future studies. Basically, these aspects are all a function of the fact that we collected our data in the manner of the survey researcher. Both the strengths and the weaknesses of this approach are reflected in our results.

The fact that we have comparable data from all of our subjects, that we can speak of the distribution of responses and the position of any individual or subgroup of our sample with respect to this distribution, is a strength which we value highly. The fact that the vast majority of our questions had precoded responses and did not permit any great degree of internal analysis is a weakness which we would hope could be counteracted in the future. The major difficulties we have experienced because of this structured approach have come with respect to the characteristics of the individuals, in part because of the inadequacy of our prior conceptualization. The several scales from the Cornell and MMPI inventories were originally included to help us tap such dimensions as anxiety, tension, and so on. We found, however, that the subjects' responses seemed to reflect another dimension which we had not originally considered but which appears to be highly relevant: the dimension of denial. Similarly, some of our straightforward attitude questions were answered in such a way as to indicate some denial of the significance of external forces. In both cases we have interpreted the findings to our own satisfaction as relevant to the dimension of denial, but we would feel more secure in this interpretation if we had a greater diversity of sources of insight into this dimension.

The solution to this problem is not to negate the strengths of the survey approach, of course, but to combine these with the strengths of more intensive, less structured methods. To argue on the basis of our experience that this would be desirable in future studies of this kind, however, is to assume that the kinds of effects of personal characteristics which we have noted would be expected in other studies. We think that this is a reasonable assumption. In such cases as the one we have investigated, one of the salient characteristics is the combination of a level of strain and difficulty in coping with it by direct means. Since there will evidently always be something blocking effective coping, we would expect that various psychological defense mechanisms would often be involved, and one

important mechanism which should be expected to appear is denial.[3]

In any event, whether or not denial as a specific defense mechanism is to be found universally in such cases, we should expect to find some kinds of defense mechanisms playing an important role in the responses made by the individuals involved. This would suggest that the investigator should be sensitive to *some* kind of latent content in the subjects' responses, and it would be well for him to have included in his research instruments a method of investigating this possibility.

One other factor which is related to our survey research approach is the question of sampling. There are two issues to be noted here. First, the fact that we originally drew a sample and then added all of the remaining affected cases is in many respects one of the strengths of the study. It presented some problems of analysis, however. We found ourselves faced with a population (the affected cases) and a not very easily defined sample (the original sample with the sampled affected cases removed). We now think that it would have been better to take all of the affected cases out of the population before sampling and then draw a sample of the population of nonaffected cases. This would have strengthened our discussion of the characteristics of "the population at risk." We do not think that this has been a serious flaw in the current study, but it is an issue worth considering in future investigations of this kind.

Second, we found that in the analysis of the sociometric data we were hampered by the fact that we had only a sample of the population rather than the entire population. We thus found a sizeable number of our subjects' choices were individuals not in our sample, and therefore, we could not make statements about either the personal characteristics or the choice patterns of such individuals. Again, the fact that we had a sample of nonaffected cases and the

[3] Miller and Swanson (1960) have discussed defense mechanisms as falling into two "families," and they say (p. 200) of the first of these: "Members of the first family share the characteristics of simplicity, maximal distortion, generality, and the creation of social difficulties. Denial is representative of such mechanisms. Almost anything can be denied, be it observable fact or motivational state, since the mechanism results in a blotting out or reinterpreting of the event." (See also, Swanson, 1961.) It is also interesting that they suggest that this family of defenses should be more frequently found in lower class people in our society, people such as our subjects.

population of affected cases raised problems. Although we feel that our statements about the relative tendencies to choose within or between any of our three major subject categories are reasonably well-founded, the kind of sample we had made it impossible to be very confident with respect to statements about the absolute numbers of choices received by any of our subjects. We cannot say with any certainty, for instance, that those whom we have called "isolates" did not actually have many good friends in the plant. We only know that we did not find any and that it is probable that more absolute isolates are among those who received no choices than are among those who received some choices from our subjects. There would be real advantages, therefore, in having information about the total population at risk if this were possible.

Given the difficulties of research in this kind of situation, is there good reason to persist in attempting to investigate such epidemics? We think there is, because we view such situations as prime examples of dynamic human events. We have referred to such epidemics earlier as "pure behavioral events" because we believe that in these situations one can observe social and psychological forces at work under conditions in which other kinds of factors play as limited a role as possible. The discomfort that is experienced is a function of situational factors that have little or no direct physical or biological relevance, and the definition of the meaning of this discomfort as well as the process through which one decides what to do about it are almost purely social and psychological in nature.

However, the fact remains that the experience is a physical one; actual physiological disturbance does occur. Of course, we have simply assumed this for our purposes and moved on from there. And yet this is a big assumption, and at best it is a crude one. There is good reason to view such epidemics as *both* medically and behaviorally relevant even if we are correct in assuming that the physiological upset is "only" a manifestation of situational pressures and psychological characteristics related to one's ability to cope with stress. There seems to be little doubt that the physical characteristics of the individuals involved are important components of the ultimate action taken, just as the cultural context is an important factor in determining what kinds of definitions of their experiences will be seen as credible.

We did not have any data on the psychophysiological characteristics of the women, but it seems reasonable to assume that individuals vary both in their abilities to keep stress "psychologically compensated" and in the kinds of physiological responses they exhibit when they fail to do so. Differences of this kind may be highly related to such factors as the use of certain kinds of psychological defense mechanisms such as denial, but it seems just as likely that different defenses could be used by persons with similar levels of ability in psychological coping.[4] Important aspects of this kind of collective event, therefore, which we have been unable to deal with at all, are the processes through which situational strain is transformed into psychological stress and then into physiological arousal. Such processes are also highly significant ones and should receive careful study.

All of this discussion would indicate that we need more and better investigations of the type we have carried out, and we hope that the success of the present study has been sufficient to motivate others to move in that direction. However, it is equally clear that further clarification at the conceptual level is a necessary part of such future work. In the next chapter we add as a kind of postscript a discussion of one type of clarification we think desirable. The concepts of epidemic, contagion, and diffusion are all somehow related to each other, but the relationship is not very clear. It is toward a clarification of that relationship that our last chapter is directed.

[4] Such studies as that of Funkenstein, King, and Drolette (1957) have made a contribution here. They make an interesting distinction between the immediate reaction to the initial experience of stress and the ability to "master" the stressful experience over time. They found these two characteristics to be almost completely independent of each other.

9

DIFFUSION, CONTAGION, AND EPIDEMICS

Throughout this report we have referred to the event we were studying as an epidemic. This term is generally used in medical settings and usually refers to the spread of a disease. As a result, the term may be seen as not really applicable to our case since no evidence of a toxic element in the environment could be found. Although such a conclusion does not seem justified to us, it can be defended. In fact, those experts whose business it was to deal with epidemics ultimately defined this event as outside their realm of expertise. We have also emphasized throughout this volume that this was close to a "pure behavioral event," and we have called it a case of hysterical contagion. The term "epidemic" is not usually used by the behavioral scientist to refer to events within his area of inquiry; he is likely to use the term "contagion." But evidently the contagion normally dealt with by the medical epidemiologist is different from the contagion of the behavioral scientist. We also know that the behavioral scientist uses another term, "diffusion," which is evidently closely related to contagion. We have suggested elsewhere (Kerckhoff, Back, & Miller, 1965) that these two terms may refer to phenomena which are

quite similar, but we have not yet attempted to describe the similarities and differences. The purpose of this chapter will thus be to clarify the distinction between diffusion and contagion and to locate hysterical contagion and epidemic in this conceptualization.[1]

The term "contagion," as used by the behavioral scientist, generally suggests a more rapid dissemination of the pattern than does the term "diffusion." There is also often the suggestion that diffusion is a process which occurs through the free choice of the participants, whereas contagion occurs against the participants' wishes. Both of these characteristics of contagion are reflections of its traditional use by the medical profession in reference to the spread of communicable diseases. We will begin, therefore, with an examination of a set of postulates which reflect the usual meaning of contagion in the medical setting and contrast them with the characteristics of diffusion as viewed by the behavioral scientist. We will then turn to a consideration of the distinction between contagion and diffusion in behavioral science, and, finally, we will discuss the difference between hysterical contagion and other forms of contagion and diffusion and relate these terms to the concept of epidemic.

MEDICAL CONTAGION AND BEHAVIORAL DIFFUSION

Most discussions of medical epidemics in which the toxic element is known to be involved postulate either or both of two sources of dissemination of the malady in question. These may be referred to as the "constant source" and the "interpersonal" postulates. The former would be likely if some single source of pollution (e.g., a garbage dump) were viewed as the source of the spreading sickness in a community. The second would be the approach taken in studies of communicable diseases. It is possible, of course, for both of these processes to operate simultaneously. But whatever process is postulated, the logic calls for some form of contact between the source (the garbage dump or an infected person) and a new victim.

Such contact between source and new victim may be postulated

[1] The discussion in this chapter has profited from a number of earlier considerations of some of the issues involved. Of particular importance are those of Wheeler (1966) and Katz, Levin, and Hamilton (1963).

to occur in one of two general ways. It may occur wholly at random, in which case each uninfected person in a population has an equal chance of making such a contact, or there may be some assumed structure which increases the probability that some persons will make contact. This structure may be a spatial one (who lives closer to the garbage dump) or a social one (who is likely to interact with an infected case).

These several postulates appear to be applicable in differing degrees to cases of the spread of some behavior within a population (e.g., using a technological innovation, adopting a new fad, reporting an unusual experience, etc.). For instance, an innovation that is being advertised widely by a manufacturer may be viewed as emanating from a constant source, whereas the dissemination of a new fad that begins in a college dormitory and spreads over a campus may be viewed as largely a function of interpersonal channels. In some cases the random exposure postulate may seem appropriate for both of these examples, for others a more structured postulate may seem appropriate.

Although we could undoubtedly construct examples in which any combination of these postulates is applicable, it seems safe to say that in most examples that are of interest to the behavioral scientist it would be unwise to assume that only a constant source is involved,[2] and it would be equally questionable to assume that all persons have an equal opportunity to be exposed to the sources of influence. In general, we must assume that diffusion occurs in structured contexts in which preestablished social and spatial patterns are highly relevant to the rate and pattern of dissemination.[3]

Several other characteristics of the kinds of diffusion which interest the behavioral scientist must be considered and contrasted with the traditional view of the contagion of disease. In the pure case of a medical epidemic, the important aspect in the spread of a disease

[2] The media of mass communications represent the most likely "constant source" in the social setting, but there is considerable evidence that even these media function through a network of interpersonal relations. See Katz and Lazarsfeld (1955).

[3] For a thoughtful discussion of the implications of such structured situations for the development of mathematical models of diffusion, see Coleman (1964, Ch. 17). Coleman acknowledges the complexities involved by referring to such situations as "incomplete social structures."

is the contact between the infectious element (e.g., a germ) and the victim. Although there are complications introduced by the fact that all sorts of things may transport such an element (the air, food, another person), the crucial "cause" of a new infection is the contact between that element and the victim, and this is a physical contact between a person and a physical element. The behavioral scientist is also concerned with the contact between an element and a potential new case, but both the element and the form of the contact are different. In these cases the most important element is an idea, a belief, or an attitude; it is basically a cognitive rather than a physical element. The contact in question is likely to be a visual or an auditory one which may occur over a widely varying set of spatial relationships; it does not require physical contact or even a close spatial association between the "carrier" and the "victim."

With good reason, most behavioral scientists expect that there will be more contacts, and more potential influence per contact, between friends, relatives, or others who have a continuing relationship involving positive affect. A more subtle and perplexing issue is involved here, however. It is quite possible for transmission of a behavioral innovation to occur between persons who normally do not interact at all. The most obvious example of this is one in which a very prestigious person adopts an innovation and a less prestigious person observes this and models his behavior after the first. This is still not a case of random contact in the population, but it involves a different kind of relationship than that implied by the examples of friend or relative. Discussions of reference group behavior illustrate the importance of such relationships, but, unfortunately, the situation is still more complex. Since the core element being transmitted is a cognitive one, it is quite possible for someone to "receive" that element from a person with whom he normally has no relationship at all, simply by observing such a person's behavior. This is an underlying assumption in much of the literature on crowd behavior.

This is not to say that spatial or social relationships are irrelevant, but it does mean that different kinds and degrees of relationship will be important in different cases. In order to suggest the conditions under which they will be more or less relevant, we must discuss two other differences between the process of spread of a physically transmitted disease and the process of dissemination of

a behavioral pattern. First, there is the difference between what constitutes "susceptibility" in the two cases. Second, there is the nature of the link between the "causal" element and the manifest behavior (or symptoms) in the two cases.

It is commonly recognized that some people are more susceptible to certain diseases than others, and the concept of immunity suggests that some persons may be fully exposed to the infectious element without becoming infected. Our discussion thus far has been limited to factors influencing the probability of exposure, but it is equally obvious that susceptibility is an important factor in the spread of disease. The same is true with respect to the spread of a behavior pattern. We would suggest that there are at least two kinds of determinants of a person's susceptibility to behavioral innovation: the qualities of the person and the characteristics of the situation he is in. There seems little doubt that any given innovation may have special appeal to those with particular personal characteristics because it meets some need (to be expressive, successful, popular, different, and so on). But it is equally obvious that being in a given situation may enhance particular kinds of needs in any person and thus increase the attractiveness of an innovation. Though it is not always possible to keep these "internal" and "external" sources of needs fully distinct, they are both important. And as the need increases, it is likely that less significant sources of social influence which offer a means of serving that need will become effective. Closely associated with the need element in the individual is the nature of the innovation—the degree to which it is acceptable in terms of personal and cultural values.

Turning to the second point, we note that in the pure case of a medical epidemic it is not the overt symptoms which are directly transmitted, but some infectious element. One gets sick (exhibits symptoms) because he is infected by a germ. At the risk of seeming to overintellectualize the process, we would argue that a similar process is involved in the diffusion of behavioral innovations. Behind each such innovation lies an idea or belief which gives meaning to the behavior. It is not the behavior as such that is disseminated, but the idea or belief. Just as the sick person exhibits symptoms because he is infected with the germ, the adopter of an innovation

is likely to behave as he does because he has accepted the idea. However, whereas there is a rather direct link between the germ and the sickness, there is another step involved between the idea and the behavior. According to the traditional view of epidemics, if a person is infected, he gets sick; but if a person accepts the idea, he may or may not behave in the innovative manner. The new idea may "make a difference" in his behavior, but that "difference" may be something other than a direct adoption of the innovation. One may believe that a new type of fertilizer will grow better plants but actively urge its rejection because its use violates community norms. One may believe that the theater is on fire but stay in his seat to avoid being trampled. We shall suggest later that there may be a similar relationship between the physical cause and the disease.[4]

It seems likely that many of the same factors which determine whether a person will accept a new idea or belief also determine whether he will take the kind of action, based on that belief, which forms the manifest evidence of dissemination. The nature of the actions taken by those around him, his own needs, and the cultural definition of the act should be equally relevant here. But, as we have suggested, in the case of the spread of a behavioral innovation there are actually two processes occurring at the same time. One is the process of dissemination of a belief, the other is the process of putting that belief into action through carrying out a new form of behavior. Although the behavior is the manifest element involved, we cannot understand its dissemination without reference to the belief. Yet the *same* relationship or need pattern may encourage a person both to believe and *not* to adopt the innovation. One may be told by a good friend (or the leader of his religious sect) that a new kind of fertilizer does grow bigger corn *but* that its use would ultimately ruin the soil (or violate a commandment from God). One may be told by a loved one that a disaster is approaching *but* that the best chance of survival is to stay put since attempting to escape would only increase the danger. One may need to believe that there are

[4] There may well be a parallel between this distinction in behavioral examples and "how sick" a person gets once his body "accepts" (i.e., does not fully repel) the germ. Since we are not attempting a full review of all the factors involved, however, we will not follow up this possibility.

great dangers in the world, but he may also need to believe in his own ability to face danger fearlessly.[5]

Thus, we have suggested that the dissemination of a behavioral innovation differs from the spread of a physically transmitted sickness in at least the following ways: (1) the element being disseminated is cognitive rather than physical; (2) the element may be transmitted through space and by means of a variety of social relationships; (3) "susceptibility" is a function of the need structures and cultural definitions of those "exposed" to the element, although situational factors may contribute significantly to these needs and serve to alter these definitions; (4) there is a less direct link between the element (the idea or belief) and the critical outcome (manifest behavior in keeping with the innovation); (5) the same kinds of factors operate to influence the acceptance of the belief and the adoption of the innovative behavior, but a given source of influence may operate in different ways with reference to these two outcomes.

SOCIAL DIFFUSION AND CONTAGION

We are now in a position to attempt a summary of the previous discussion in a series of postulates about the diffusion of a behavioral innovation similar to those with which we began. To simplify the statement of these postulates, we will refer to one who has accepted the underlying idea or belief as a "believer" and to one who actually carries out the behavioral innovation as an "adopter."

[5] The opposite may also be true. One may be influenced to carry out the behavior in question without actually accepting the belief. The farmer may begin to use the new fertilizer, or the doctor to prescribe the new drug, because all of the prestigious practitioners are doing so. Studies of crowd behavior have described cases of people who rush along with the crowd without knowing the cause of the excitement. One may engage in a public demonstration because of a need to be rebellious without accepting the beliefs of those who are leading the demonstration. Although it is important to emphasize this side of the picture, there is the danger of viewing the spread of a behavior as wholly unrelated to cognitive or motivational elements. We would submit that, although there may well be those who adopt the innovation without accepting the belief, an underlying belief is always involved, and the core of the diffusion is the result of the acceptance of that belief. We must recognize that there may be *other* reasons for some to adopt the behavior, but this should not lead us to deny the central role of a cognitive element in the process of dissemination.

Postulate 1. The probability of acceptance of the belief increases as the intimacy of a person's social relationships with those who believe increases. (If such relationships are segmental, the probability of acceptance will vary with the relevance of the belief to the life segment encompassed by the relationship.)

Postulate 2. The probability of acceptance of the belief increases as the number of persons known to have accepted it increases. (There are, however, counter forces in any situation which make universal acceptance unlikely.)

Postulate 3. The probability of acceptance of the belief increases as the level of prestige of persons known to have accepted it increases. (If a believer's level of prestige is higher in some specialized problem areas than in others, his prestige level in the area most directly related to the content of the belief will dominate.)

Postulate 4. The probability of acceptance of the belief increases with the degree of consistency between that belief and the established cultural definitions of the population of which the individual is a member.

Postulate 5. The probability of acceptance of the belief increases to the extent that the belief serves the needs of the person (whether these needs are a function of stable personality characteristics or situational determinants or both).

Postulate 6. The probability of adoption of the innovation increases to the extent the person becomes a believer. There will not be a perfect relationship here, however, because:

Postulate 7. The probability of adoption of the innovation increases as the intimacy of the social relationships between the person and adopters increases.

Postulate 8. The probability of adoption of the innovation increases as the number of persons known to have adopted it increases.

Postulate 9. The probability of adoption of the innovation increases as the level of prestige of persons known to have adopted it increases.

Postulate 10. The probability of adoption of the innovation increases with the degree of consistency between the action involved and other culturally approved forms of behavior in the population.

Postulate 11. The probability of adoption of the innovation in-

creases to the extent that the behavior in question serves the needs of the person.[6]

These 11 postulates appear to apply best to cases of diffusion of a behavior pattern such as a technological innovation. When we consider the concept of contagion in relation to these postulates, they appear at first to be too orderly and systematic. The difficulty is evidently that the term "contagion," as distinct from "diffusion," normally refers to a more rapid dissemination of the pattern. This greater rapidity implies a less rational type of process, and it has been usual in discussions of contagion to suggest that the distinctions among sources of transmission included in these postulates are less relevant to contagion than to diffusion. Within the framework of these postulates, contagion is characterized by the greater significance of the needs of those persons involved. Contagion occurs in situations where the need is immediate and great. There is a strong need, shared by a number of people, to take action, but there is no clear definition of an appropriate act. When a seemingly appropriate act is suggested (and probably carried out by one or more of those experiencing the need), the blocked impulses to act are released and adoption of the suggested solution is rapid and widespread.

Redl (1949) has suggested that the original restraint in such cases is internally imposed by the actors due to their fear of the consequences of action in keeping with their needs, consequences which themselves may be internal (guilt) or external (punishment). The implication in his discussion is that the contagious act is already known and viewed as attractive by the actors, and knowledge (sight) of someone carrying out such an act serves to reduce the self-imposed restraints by making the act seem more acceptable. This certainly seems true in many cases, but it will generally be difficult to know the extent to which the restraint should be viewed as self-imposed. Thus, a more general statement of this position might be that contagion occurs in situations in which the need for action is great, where some barrier exists to prevent action in accord with the need, and where a seemingly suitable means of action is proposed

[6] Although all of these postulates are put in a single positive form, they should be understood to include the obverse of the statements made. Also, the parenthetical statements after Postulates 1, 2, 3, and 5 should be understood to be included in Postulates 7, 8, 9, and 11.

(usually through the example of one or more persons) which lowers that barrier.

Such a position predicts that rapid dissemination will occur where the need element is highly salient and the behavior of an adopter serves to lower the barrier which has prevented action to satisfy the need. Rapid dissemination might also be expected under other circumstances. For instance, even though the need element were not particularly high, one might expect rapid dissemination of a pattern once it had been adopted by a prestigious person (e.g., the admired Army sergeant whose manner of wearing his cap is copied by his men). Similarly, patterns which serve to symbolize the membership of individuals in various groups or categories which they define as significant are likely to be adopted rapidly (e.g., the "odd" hair and clothing styles adopted by adolescents). In the same way, the relationship between established cultural definitions and an innovation will influence the rapidity of dissemination (e.g., the difficulties faced in introducing technological innovations in traditional societies). All of the factors listed in the above 11 postulates, therefore, should be seen as relevant to the rapidity of dissemination of an innovation.

There is another dimension which should be considered, however, when we attempt to explain the differences in rates of diffusion, a dimension which is not reflected in our 11 postulates. Basically, the factors reflected in the postulates are all relevant to the ease with which an innovation (or the idea behind it) will be accepted once an individual is made aware of it. Situations vary, however, in the rapidity with which knowledge about an innovation (and its effects) may be disseminated. A major determinant of rapidity of dissemination of information is the distance between members of the collectivity. Perhaps the best way to denote such a variation would be to refer to "diffuse" and "compact" collectivities (Turner & Killian, 1957). In general, the more diffuse the collectivity, the more slowly will information travel and the more slowly will an innovation be disseminated. Obviously, the media of mass communications make this kind of statement debatable, but although these media are capable of transmitting information and are thus capable of reducing the "distance" between members of a collectivity and a central source of information, they do not reduce the distance be-

tween a given number and all other members of the collectivity. The immediacy of a crowd experience, for instance, cannot be duplicated by the mass media. This is in part a function of the rapidity of information flow among members in a crowd and in part a function of the flow of different kinds of knowledge than are communicable through the mass media—knowledge of other individuals' emotional arousal, their immediate response to shared experiences, and so on. Therefore, other things being equal, the compact collectivity is likely to facilitate the dissemination of intimate knowledge of members more rapidly and fully than the diffuse collectivity.

It has also been implicit in the preceding discussion that the effect of the action of innovators may have either or both of two different kinds of effects on those who know of their innovative behavior. The observation of such action may serve to increase the attractiveness of the behavior in question (by demonstrating that it is really possible, that it is enjoyable, that it is effective, that it is prestigious, and so on). On the other hand, as the discussion of Redl's analysis indicated, it may also serve to lower the barrier between the desire and the act. In the first case the innovator serves to increase the attractiveness of an act; in the second case he provides a means for carrying out an already attractive act. In the first case the observers become *motivated* to act by observing the innovator; in the second case they are already motivated and become *able* to act by observing the innovator.

We may now express these ideas in two further postulates which together with the greater relevance of needs in contagion help us to differentiate between diffusion and contagion.

Postulate 12. Rapidity of dissemination of the belief and/or the act will tend to be greater in a compact than in a diffuse collectivity.

Postulate 13. Knowledge of the behavior of adopter(s) (and its outcome) may serve either to heighten the attractiveness of the act or to lower the restraints from performing the act, or both.

In the pure case of diffusion, dissemination occurs in a diffuse collectivity. It depends more on social and cultural factors, and knowledge of the behavior of adopters serves to heighten the attractiveness of the act. In the pure case of contagion, dissemination occurs in a compact collectivity. It depends more on personal (need) factors, and knowledge of the behavior of adopters serves to lower preexisting

restraints to performing the act. It is obvious, however, that these considerations do not permit a clear-cut differentiation between diffusion and contagion. Various combinations of these factors are possible. Rapid dissemination, based on heightened needs of the adopters, may occur in a diffuse collectivity. The spread of a pattern in a compact collectivity may be the result of the adoption of an innovator's act by a prestigious figure whose adoption of it makes it more attractive. Because of such complexities, we can only describe the characteristics of pure cases of contagion and diffusion while at the same time we must acknowledge the existence of various kinds of mixed cases.

HYSTERICAL CONTAGION

How does our view of hysterical contagion fit into this discussion? We have, of course, indicated throughout the previous chapters that the kinds of variables referred to in our discussion so far were operative in the example we studied. We have presented evidence of the significance of intimate associates (Postulates 1 and 7), of a "crowd effect" (Postulates 2 and 8) and of the importance of the personal characteristics of the women (Postulates 5 and 11). We also found that those who were most severely affected were the strongest believers (Postulate 6). We have suggested that the initial opinions of experts (Postulate 3) and the behavior of union stewards (Postulate 9) may have had an effect on the contagion. We have also suggested that the belief and the behavior in question were consistent with our cultural definitions of toxic insects and illness behavior (Postulates 4 and 10). Our data suggest that the compact nature of the collectivity in the dressmaking departments contributed to the rapidity of the spread of the symptoms (Postulate 12).

It seems reasonable, therefore, to view hysterical contagion as amenable to the same general kind of conceptualization as the diffusion and contagion of many other types of behavior in a population. Yet there are differences, and these need to be emphasized along with the similarities. The differences are most apparent, perhaps, with reference to Postulate 13. Implicit in Postulate 13 is the assumption that the behavior pattern which is disseminated through a population in both diffusion and contagion is one which is positively valued