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# A path-analytic strategy to analyze psychoanalytic treatment effects

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This paper introduces a path-analytic strategy to analyze psychoanalytic treatment effects. A simple causal model is used to analyze a well-known case study by Charles Brenner. Application of even this simple model to the case study sharpens causal inferences that may be validly made, highlights important aspects of the psychoanalytic process and builds a foundation for further model development.

**Keywords:** case study analyses, causal analyses, elimination of rival hypotheses, path analysis

Much of our clinical knowledge in psychoanalysis about what helps patients comes from an accumulation of treatment practices that have been selectively retained, often quite informally, from all those tried by psychoanalysts over the past one hundred years. If some approaches have worked better than others, and if these approaches have been retained more persistently and passed on effectively to subsequent generations through case studies, vignettes, supervision and candidates' own psychoanalyses, then the treatment practices that have emerged within our psychoanalytic institutes, and elsewhere, represent a valuable and tested source of knowledge about what works in psychoanalysis.

However, the selection and accumulation of effective psychoanalytic treatment practices may not occur as smoothly and efficiently as we just implied, and additional methods may improve the process. There are biases in memory, imposition of patterns on clinical material where none exist, unconscious coaching of the patient and other phenomena discussed below, all of which can potentially lead to inaccurate beliefs by practicing clinicians.

We introduce the argument in this paper that the current and somewhat informal selective retention of effective clinical practices in psychoanalysis may be improved by application of path-analytic thinking to clinical case material. We will try to show that the path-analytic methods we outline may sharpen the testing, probing and selection of effective practices. The methods are not a source of treatment practices contradictory to what has evolved in and outside of our psychoanalytic institutes. We will try to show that the methods build upon these practices. They are a refining methodology which may improve, in the long run, the selection of effective clinical practices from among those that are tried.

This paper applies a simple path model to a well-known psychoanalytic case study reported by Charles Brenner (1976, 1982). The goal is to illustrate the usefulness of a simple path model to improve our understanding of psychoanalytic treatment effects. One does not absolutely require path-analytic thinking to make many of the points we make below. However, even the very simple model we use here provides a useful representation of the significant cause-and-effect relationships that need to be kept in mind for a thorough accounting of the various causes and effects that might be at work in the psychoanalytic situation. We hope analysis of Brenner's case study will tease the reader into thinking about the potential usefulness of more complex path models of psychoanalytic treatment effects in other case studies.

Path-analytic methods, when fully developed, are quantitative. See Duncan (1975) and Kline (1998) for excellent introductions. They are a subset of methods drawn from econometrics, structural equation modeling, causal modeling and quasi-experimental design, which have been widely used in fields as diverse as psychology, sociology, medicine, history, economics, demography, education, genetics, epidemiology and political science.

We do not try in this paper to illustrate the quantitative potential of these methods for psychoanalytic research and data analysis. Such an endeavor would require data collection and statistical analyses not commonplace in most current psychoanalytic research, and goes far beyond the scope of this paper. We present path-analytic thinking instead as a modest blueprint for application to clinical case study material like that reported by Brenner. Even this limited use, we believe, provides a more optimistic alternative to Grunbaum's (1984, 1993) pessimistic prospects for advances in psychoanalysis from analysis of clinical data. It also follows earlier pioneering approaches to causal understanding of psychoanalytic case-study data, especially by Edelson (1984, 1988), Moran and Fonagy (1987) and Glymour (1982).

Somewhat more generally, path-analytic methods may help address Boesky's formidable requirements for a systematic definition of the clinical psychoanalytic process:

> to describe the most important changes effected by the psychoanalytic treatment … to explain how these changes were effected [and to develop] a methodology for the validation of the claims that certain changes have occurred as a consequence of the interventions stipulated by the definition of the psychoanalytic process (1990, p. 558).

## Guiding background ideas from psychoanalysis

Once a psychoanalyst decides to present clinical work, he/she must decide what clinical data to present, what questions to ask and the methods used to answer them. These criteria are all guided by the background thinking of the investigator, the assumptions he/she makes about how psychoanalysis works and the provisional thinking about what is important. We make no claim that the background ideas we have chosen below to guide our presentation of methods represent the common ground, or even an equal or representative sampling, of the many psychoanalyses (Wallerstein, 1988; 1990). They are, rather, a sampling of psychoanalytic ideas we find useful to develop the methods presented here and in subsequent publications. We feel confident that other samplings could serve just as well, but the reader preferring ideas different from those we present will have to decide this for him/herself.

According to psychoanalytic theory, pathogenic dispositions of some sort cause the patient's dysfunction. It is important to understand how we use the words 'pathogenic dispositions'. We use these words, which we interchange freely below with 'pathogenic mechanisms', much more broadly than their medical meaning of an agent that enters the body like a virus to cause disease. We use them to designate all the features within the patient that are conjectured to be responsible for the patient's dysfunction. They can be mental mechanisms, states or deficits, or physical mechanisms, states or deficits. They can be conscious or unconscious. They can cause dysfunction in some environments and social settings but not in others.

The particular pathogenic dispositions conjectured among analytic viewpoints are numerous and expressed in different terms, and are also thought to have different components and mechanisms. They include drives, affects, fantasies, conflicts, resistances, defenses, transferences, fixations, deficits, self, internal objects, part-objects, selfobjects, grandiose self, depressive and paranoid positions, and interpersonal relations, to give a sampling. But there seems to be almost general agreement that these pathogenic dispositions are often unconscious, and that they often represent features of the person carried over from relationships with early important persons in life. They are ill-working in the patient's current life situation and they result in a broad possible range of negative life outcomes including inhibitions, symptoms, unpleasure, and a general mishandling of people and the patient's environment.

Psychoanalytic techniques aim to help the patient modify the particular pathogenic mechanisms held to cause his/her suffering and dysfunction. The patient provides associations and reflections with a progressive lessening of constraints and increasing degrees of meaningful affect in an analytic setting of neutrality, and an increasing working alliance with the analyst.

As the analysis progresses, the pathogenic dispositions related to interactions with early important persons in the patient's life resurface in the patient's relationship with the analyst. They re-emerge toward the analyst in ways that may be extensively disguised or recognizably similar to how they were expressed toward earlier important persons. The analyst uses these manifestations in the here and now to help the patient achieve a deepening and affectively vivid understanding that the current dispositions are further expressions of the earlier ones which are inappropriate to the patient's current life situation and are causing dysfunction. To do this, the analyst relies on analytic listening and understanding, and uses a full range of psychoanalytic hypotheses to arrive at his/her interventions which focus on the pathogenic mechanisms conjectured to play a role in the patient's problems.

Eventually, in a successful analysis, growing awareness of these mechanisms which contribute to the patient's dysfunction allows the patient to be affected more by rational argument, emotional appeals, satisfaction of needs, success or failure, and rewards and punishments. The patient's ego functions with greater autonomy and flexibility, and is freer to judge and evaluate thoughts. It is assumed that mental mechanisms determined by consciously experienced conflicts are more flexibly adaptable to external signals than are unconsciously operating factors or conflict. The patient perceives, feels, thinks, acts and handles his/her environment with a higher level of ego functioning, and less at the dictates of the pathogenic dispositions of a persisting and troubled unconscious world. These changes are conjectured to result in a durability of symptom relief, and reduction of inhibition and unpleasure.

These psychoanalytic ideas, although very selective and condensed, guide much of the presentation below. We begin with a model of a very simple causal structure in the next section and apply it to Brenner's case study. This initial model represents important aspects of case presentations in psychoanalysis, but is primarily pedagogical and not intended to capture many of the nuances found in case presentations. However, applying even this simple causal model to Brenner's case study sharpens the process of making valid causal inferences, highlights important aspects of the psychoanalytic process and builds the foundation for further model development.

## Applying a simple model of causal structure to case material

The above background ideas from psychoanalysis suggest that durable relief of a patient's dysfunction can be brought about by changes in the pathogenic mental mechanisms that are causally required for its pathogenesis. The changes in these pathogenic mental mechanisms result, in turn, from the application of therapeutic techniques of psychoanalysis. Figure 1 presents the essential ideas of this causal sequence pictorially.

Figure 1 postulates three causal effects: *a*, *b*, and *c*. Effective psychoanalytic technique is assumed to change pathogenic mental mechanisms (effect *a*), and changed pathogenic mental mechanisms are assumed, in turn, to diminish dysfunction (effect *b*). Good psychoanalytic technique may also reduce dysfunction in ways unrelated to changes in conjectured pathogenic mechanisms (effect *c*). The arrows labeled *d* and *e* represent unknown causes. Arrow *d* represents unknown influences on pathogenic mental mechanisms and arrow *e* represents unknown influences on dysfunction.

The placeholders in Figure 1 must be given further definition in any specific application. For example, the psychoanalytic treatment, the specific pathogenic dispositions and the dysfunction must each be specified. Filling out the model in any specific case means saying what technique has been applied, what pathogenic mechanisms are potentially affected and what dysfunction might reasonably change. It also means specifying each of the potential causal effects, *a* through *e*.

Figure 1 represents a very simple causal structure. Its placeholders represent a longitudinal sequence during a treatment. It models only the potential impact of some specified psychoanalytic technique on pathogenic mechanisms (effect *a*), and the effects of these on the patient's dysfunction (effects *b* and *c*). While Figure 1 represents a causal structure that probably applies to many case studies, like all models it represents some features and ignores others, and therefore does not necessarily capture too many aspects of the psychoanalytic process.

For example, Figure 1 represents a longitudinal sequence showing only a potential impact of the analyst's technique on the patient (effects *a*, *b*, and *c*). It does not represent influences in the other direction, from the patient to the analyst. Countertransference reactions of the analyst to the patient are therefore not represented. Nor does the model attempt to understand how any back-and-forth interactions between analyst and patient might create a process which makes analyst-effectiveness and patient-self-understanding possible.

Because Figure 1 is directed only toward a short longitudinal sequence during treatment, it does not directly address other important aspects of psychoanalytic thinking such as the past course of pathogenesis, the goals of treatment, analyzability, the theory of technique, and conceptions of ideal mental health. These are all important, obviously,

because they influence what the analyst does during treatment, and thus what data the analyst and patient generate for analytic data. The model in Figure 1 just assumes the analyst intervenes in some way, and then asks how the intervention affects a patient's dysfunction (effects *a*, *b*, and *c*). Even this limited model, however, asks important questions.

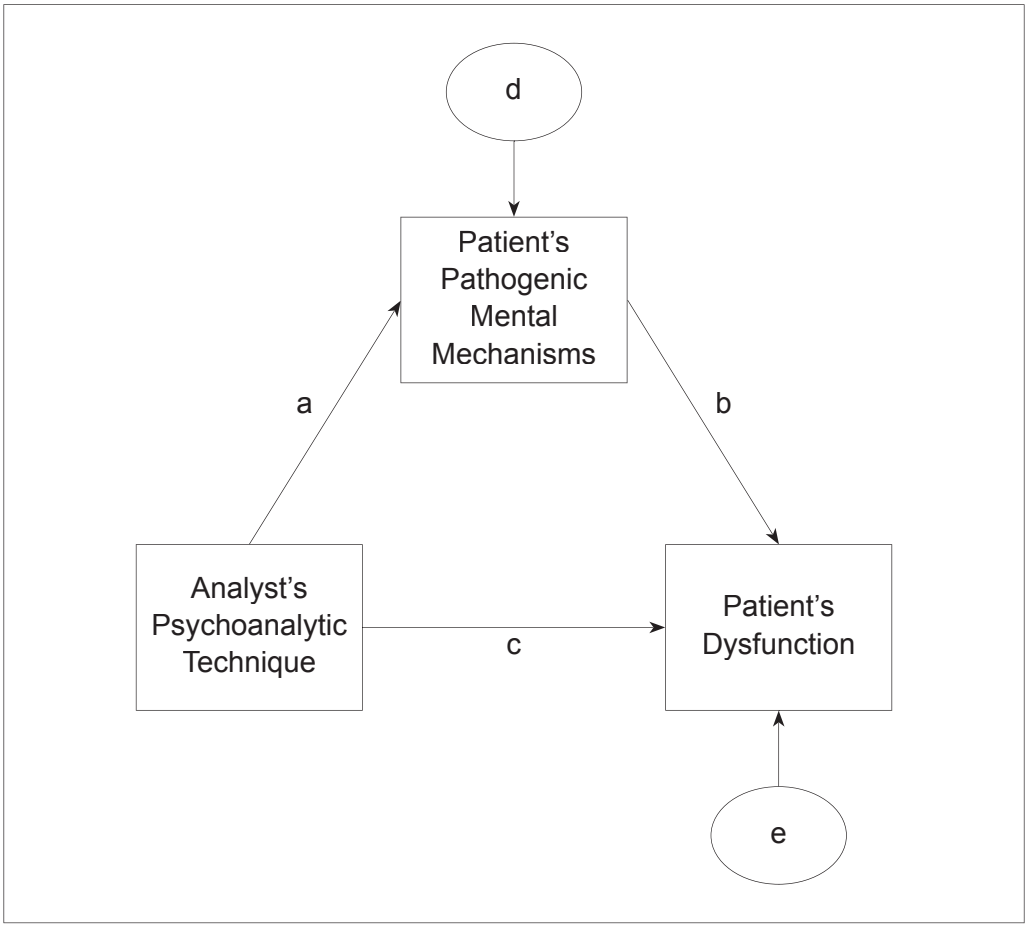


**Figure 1 — Path diagram of the relationships between psychoanalytic technique, patient's pathogenic mental mechanisms and patient's dysfunction**

For example, suppose a patient in analysis improves at the moment, and clear measures of momentary improvement document this reduction in symptoms or various dysfunctions. How much did the reduction in symptoms or dysfunctions result from changes in the pathogenic mental mechanisms conjectured by the analyst (effect *b*) as opposed to other causes (effect *e*)? How much were any changes in the pathogenic mechanisms affected by the specific psychoanalytic techniques (effect *a*) as against other causes (effect *d*)? And did the psychoanalytic intervention bring about reductions in symptoms and unpleasure primarily because it brought about changes in the pathogenic mechanisms (effects *a* and *b*), or because it was a cause unrelated to these changes (effect *c*)?

During the long course of a psychoanalysis, most analysts may answer these questions informally to themselves, and their case reports can sometimes be read partly

as reports of their answers. While case-study reports typically provide minimal data, the model in Figure 1 can still be applied informally to many such studies. The work of Charles Brenner is illustrative in this regard. As Brenner notes in his introduction to *The mind in conflict*, 'An analyst postulates the same cause-and-effect relationships with respect to psychoanalytic data as a physicist' (1982, p. 5).

## Brenner's analysis of causal structure

Brenner illustrates his sensitivity to causal structure throughout his writings. A particularly clear example is his analysis of a 29-year-old woman presented in *Psychoanalytic technique and psychic conflict* (1976, pp. 65–74) and repeated in *The mind in conflict* (1982, pp. 82–8). He describes eight improvements, which are the result of an interpretation he gave this woman, who had been in analysis for six years at that time.

These are the circumstances. The patient had sex only with women prior to her analysis. During the course of the analysis, she began having intercourse with men. These affairs with men were interspersed with ones with women. While the analyst was on vacation, the patient once more began a relationship with a woman. Upon the analyst's return, and apparently during several hours of the analysis, the patient was struggling consciously to give up her relationship with the woman, but was also trying to provoke the analyst with complaints that he behaved unfairly toward her and never gave her her due.

During one session, when she was arguing with herself that she should give up her girlfriend, there were frequent pauses where she was 'obviously waiting for her analyst to speak' (Brenner, 1982, p. 83). At this point, Brenner intervened with an interpretation conveying some of the obscure connections between her efforts to provoke him and elements of conflict that Brenner conjectured were dominating her sexual behavior: 'her unconscious need to deny (1) sexual feelings for her father, (2) jealous and hostile wishes toward her mother and her older, married sister, and (3) her rage and humiliation that she did not herself have a penis' (p. 83).

He interpreted to her that she was trying to provoke him to order her to give up her girlfriend so that she could rebel, as she had so often tried to get her parents to take a position she could use as an excuse to rebel against them. Implicit in this interpretation, and conjectured by Brenner to be well understood by the patient because it had been interpreted to her many times, was that her anger at him was really about her not having a penis, and that her father, mother and analyst didn't love her as she believed they would if she were a man. The interpretation therefore had to do with the patient's wish for love and for a penis in the transference situation, and with the fact that it was the frustration of those wishes that made her feel toward the analyst the anger that she attributed to him, via projection, lest she feel guilty, i.e., to avoid superego condemnation (Brenner, 1976, p. 67).

We turn now to the effects Brenner claims this interpretation had on the patient in terms of the model in Figure 1. We describe how Brenner's thinking fills out the details of the model with his postulation of pathogenic mechanisms.

### The patient improved as a result of the interpretation

Brenner notes that 'this interpretation appeared to have a considerable effect on the patient' (1976, p. 67). During the next week following the interpretation, Brenner says she showed eight improvements:

1) She discontinued her homosexual affair.
2) She was more feminine in dress and manner.
3) She began to date a man.
4) She asked an older, male colleague—an obvious father figure—to accept her as a pupil, even though she said in advance she was sure he would refuse.
5) She was much less angry at her analyst and was aware that she wished to be close to him.
6) She had a frightening dream, the associations to which led to thoughts of being excited while on her analyst's couch. It should be noted that she did not actually feel sexually excited either in connection with these thoughts or at any other time when she was on the couch.
7) She became aware that she was angry at her mother, at her older sister, who was her lifelong rival, and at a married female friend.
8) She recalled longing to be close to her father when she was 5 years old (Brenner, 1982, p. 84).

Brenner calls these eight outcomes 'improvements', as will become clear below, because of their consequences for the patient. Perhaps at the time he also took for granted that his readers would agree that a homosexual relationship is pathological and its abandonment a sign of improvement. This now seems a dated and potentially offensive assumption, and one many people might today reject.

Calling these eight outcomes 'improvements' highlights the issue of what defines a positive outcome in analysis, who defines it and how the definition is reached. In addition to the patient's understanding and analyst's perspective, which is Brenner's approach, a case can be made for considering an outcome's importance to various third parties in society (Strupp, 1996), its non-reactivity (e.g. Webb, Campbell, Schwartz, Sechrest and Grove, 1981), its calibration against real-life events (e.g. Sechrest, McKnight and McKnight, 1996), its clinical significance (e.g. Kazdin, 1999) and its relationship to broad quality-of-life measures (e.g. Gladis, Gosch, Dishuk and Crits-Christoph, 1999). By calling these outcomes 'improvements', we wish to remain faithful to Brenner's presentation. What is most crucial for our purpose is (1) that these are outcomes Brenner attributes to his interpretative work and (2) how he makes these attributions.

Brenner continues, 'Here is an example of analytic progress. The patient improved as a result of a correct interpretation, properly timed and preceded by much interpretative work along the same line' (1982, p. 84). In terms of Figure 1, Brenner's conclusion is that symptomatic improvement occurred as a result of good psychoanalytic technique, and presumably not from other causes (effect *e*).

As yet, we have not seen Brenner's conjectures about how psychoanalytic technique caused these symptomatic improvements, whether by influencing pathogenic mental mechanisms of some sort (sequential effects *a* and *b*) or for reasons unrelated to these mechanisms (effects *c*). We turn to that question now.

### Psychoanalytic work changed the pathogenic dispositions (effect *a*)

Since Brenner's theory is a contemporary conflict theory of psychoanalysis, one rooted historically in the final phase of Freud's theorizing in *The ego and the id* (1923) and *Inhibitions, symptoms and anxiety* (1926), it is not surprising that the pathogenic mental mechanisms for Brenner are the intrapsychic forces in conflict: drive derivatives, unpleasure in the form of anxiety and depressive affect, defenses, and superego

manifestations (Richards and Lynch, 1998). Brenner does not call these components pathogenic dispositions, but it is clear that these components of conflict are the mental mechanisms he postulates to have pathological efficacy:

> Whether the result of conflict over a drive derivative of childhood origin is a somatic symptom, a phobia, an obsessional symptom, or a neurotic character trait, it is a compromise formation in which drive derivative, anxiety or depressive affect, or both, associated with the calamities of childhood, defense, and superego manifestations all play a role that can be recognized and identified when adequate psychoanalytic data are available (1982, p. 143).

These components of conflict, for Brenner, are the pathogenic dispositions that can respond to good psychoanalytic technique: 'what a successful psychoanalysis actually achieves is an alteration of conflict in the direction of normality, an alteration that results in a normal compromise formation in place of the pathological one that was formerly present' (1994, p. 479). Brenner describes the alteration of conflict in his patient as follows.

He notes several oedipal drive derivatives expressed in his patient's transference, which were based on his earlier conjectures about her sexual feelings for her father, her jealous and hostile wishes toward her mother and older married sister, and her rage and humiliation that she did not herself have a penis. These oedipal drive derivatives include affection for her analyst; longing for his love; memories of affection and longing for her father's love when she was a child; the wish to be closely associated with an older, admired man and to be his pupil; the wish to attract men and to have sexual intercourse with them; and anger at rival women (1982, p. 87).

His interpretation in the transference made the patient better able emotionally to understand and tolerate these oedipal derivatives—wishes for her father's love, her angry, defensive reactions to its frustration and her anger at rival women. These changes allowed development of new compromise formations.

Brenner does not call attention explicitly to all the psychological mechanisms that might determine his patient's choice of new compromise formations from among the many possible from the elements of conflict. He would probably include many ego functions which, as he says, 'in their role as executants of the drives and, later, of the superego … will grant to both the fullest expression compatible with a tolerable degree of unpleasure' (p. 116). He would probably say, also, that the new compromise formations could now include some combination of more pleasurable gratification of drive derivatives, less accompanying anxiety and depressive affect, less inhibition of function resulting from defenses, less self-punishment and self-injury, and less need to be in conflict with the patient's environment. But he does not say how these and other mechanisms, individually or in combination, brought about his patient's specific choice of new compromise formations.

Brenner does, however, make it clear that the patient's compromise formations were altered, and that they were altered as a result of his interpretation:

> Before the interpretation the derivatives I have just listed could not be tolerated or gratified. They aroused too much unpleasure. After the interpretation the patient's reaction to her oedipal wishes and jealousy was less defensive in the sense that the resulting compromise between drive derivative and defense allowed more in the way of gratification … The patient's reactions to her positive oedipal wishes after the interpretation were no less a compromise between drive derivatives and defenses than they had been before. Defenses against her oedipal drive derivatives were as easily identifiable after the interpretation as they had been before it. The new compromise formation represented analytic progress, but it was still a compromise formation for all that (pp. 87–8).

In terms of Figure 1, alteration of pathogenic conflict toward less pathological compromises resulted from Brenner's psychoanalytic technique (effect *a*), and presumably not from other causes (effects *d*).

**Changes in the pathogenic mechanisms affected the outcomes (effect *b*)**

Brenner next tells us that alterations in the patient's pathogenic conflict toward less pathological compromises brought about the eight improvements he noted. What happens is that 'the patient's compromise formations change in a progressive way … those compromise formations we call pathological give way to other compromise formations which are properly called normal' (1982, p. 82). He adds, 'By this I mean that, as analysis progresses, the patient's compromise formations change in such a way that the drive derivatives in question are less disguised, less distorted, and can be gratified with pleasure to an increasing degree' (p. 82). He then illustrates beautifully how changes in the patient's compromises resulted in the eight outcomes, which we paraphrase (see Brenner, 1976, pp. 68–70; 1982, pp. 85–7):

1) Her pattern of reaction formations changed. The reaction formation of anger at men to ward off oedipal loving and sexual wishes toward them was diminished. The result was that she started dating a man and gave up her homosexual affair. She was much less angry at her analyst and was aware that she wished to be close to him.

2) Repression of her oedipal wishes for her father was also lifted, but only partly, so that she defended against her sexual wishes for her analyst, which gave rise to anxiety, by experiencing them as ideas as mere inferences from her associations to her dream. She could now recall longing to be close to her father when she was 5 years old. She could also identify with an older, admired colleague—'an obvious father figure'—and ask to be his pupil.

3) Repression of oedipal anger at her mother was also partially lifted and she became aware that she was angry at her mother and sister, which she now displaced partly to her married friend.

4) The defensive identification with men also changed after the interpretation. Before the interpretation, this identification influenced her sexual behavior. She wooed a woman, a sexual relationship in which she sometimes played a male role. After the interpretation, the identification was nonetheless expressed in her vocational behavior as a sublimation.

In terms of Figure 1, the eight improvements resulted from altered expressions of the pathogenic conflicts (effect *b*), and presumably not from other causes (effects *c* or effects *e*).

**Brenner's postulation of pathogenic mechanisms**

Summarizing the causal factors in terms of Figure 1, Brenner says that a good psychoanalytic interpretation altered the patient's expressions of pathogenic conflict toward more normal compromises (effect *a*), which then brought about the eight improvements he observed (effect *b*). This implies that effects *c* and *e* are negligible, though Brenner does not discuss this implication.

His approach to the explanatory problem of how his interpretative work influenced the eight improvements is somewhat as follows. His thinking is along the following lines: I will postulate as mental-mechanisms conflicts and their expression in compromise formations. I will further postulate how my interpretative work changed these conflicts

and compromises (effect *a*), and how the shifting compromise formations caused the eight improvements I observed (effect *b*).

By postulating the changing compromise formations, Brenner brings his interpretative work and eight outcome observations under a single set of explanatory ideas. These postulated mechanisms are widely applicable and can also explain outcomes in the analysis of other patients. Thus, Brenner's postulated mechanisms—which change with psychoanalytic work and affect results when they change—adhere to the basic idea in explanation of accounting for the greatest number of observations with the fewest assumptions. He brings a number of independent interpretative- and results observations under a minimum number of postulated causal processes.

## Improving the analysis of causal structure

Brenner's work is a masterful use of the case study in psychoanalysis to analyze causal structure, to postulate shifting pathogenic mechanisms to explain the outcomes he observes, and to say how changes in these pathogenic mechanisms result from a 'correct interpretation, properly timed and preceded by much interpretative work along the same line' (Brenner, 1982, p. 84). As useful as his case study is, however, further application of Figure 1 and attention to plausible rival hypotheses reveal a number of places where validity could be improved.

### The importance of plausible rival hypotheses

A danger exists in analysts assuming an effective impact of their interpretative work on outcomes when they do not know the results in the absence of their conjecture. Knowing the outcome in the absence of the conjectured cause is not always necessary, of course. If you see a lightning flash outside your window and a bolt hit a healthy tree, followed immediately by the tree falling down, you can probably conclude validly that the lightning strike was the cause. The tree could have been blown down or could have fallen from natural dying, but you rule out these plausible rival causes when you do not see evidence supporting them.

Unfortunately, causal attribution of psychoanalytic outcomes to a 'correct interpretation, properly timed and preceded by much interpretative work along the same line', or to any aspect of psychoanalytic work, is not the same as attributing a falling tree to a lightning strike. The causal inferences in psychoanalysis are weak or invalid to the extent that plausible rival hypotheses to the conjectured causes are not entertained and not ruled out in the minds of critical observers. While analysts often find the rival causes posed by non-analysts implausible, many non-analysts remain convinced that case reports too often fail to assess the validity of the causal inferences they make. It is useful to examine the causal inferences Brenner makes or implies with the standard of successful elimination of plausible rival hypotheses in mind.

### Did the interpretative work cause the outcome changes?

Brenner concluded that the interpretative work was responsible for the eight improvements he observed. In doing so, he in effect says that any possible other rival causes of these improvements (causes *e*) are implausible. Years of analytic work may have convinced Brenner, and perhaps other analysts who read his report, that effects *e* are implausible or of only minor importance. Brenner and others might argue that the history of such

a patient usually shows that changes in interpersonal relationships, adaptational needs, time passing for natural change or fluctuations, and sometimes previous treatments, have typically not yielded the kinds of changes Brenner described. But his conclusion can still be questioned.

Busch notes how Brenner reports these changes 'as if they were self-evident examples of the result from well-timed interpretations … and couldn't be due to numerous factors' (1998, p. 45). A glance back at the eight outcomes suggests that other causes (effects *e*) seem potentially as plausible as Brenner's interpretative work. For example, how can we be sure these outcomes could not have occurred as a result of extraclinical causes in the patient's life, such as concurrent changes in peer influences or her wishing to fit into socially acceptable patterns? Do we really know that the patient's mother and father could not have changed their attitudes and ways they acted, and that the eight outcomes did not directly reflect these changes instead? Could not the outcomes have resulted from the continuing influences of spontaneous remission? How can we rule out the possibility that the outcomes represent the more or less random fluctuations in attitudes, emotions and choices that occur in most people's lives over a period of time? The inference that effects *e* are implausible as rival explanations to Brenner's interpretative work seems at least contestable.

Brenner's causal inference that the interpretative work was responsible for the eight improvements is based on the eight carefully noted outcomes, which he then compares with other outcomes. The causal inference is based upon a general expectation of what the outcomes would have been had the interpretative work not occurred. There are no formal means to certify that the observed outcomes would not have occurred from other causes (*e*) in the absence of the interpretative work. As a result, the causal inference lacks validity to the extent that the other causes (*e*) can plausibly rival the interpretative work as an explanation of the outcomes. For an excellent discussion of these views and related issues, see Rubin (1974), Holland (1986), and Glymour (1986, 1999).

**Did the interpretative work affect the compromise formations?**

Brenner concluded not only that the interpretative work was responsible for the eight outcomes he observed, but that the interpretative work resulted in a restructuring of compromise formations (effect *a*). In doing so, he said in effect that the conflictual dispositions that initially led to pathological compromise formations were changed by his interpretative work, which brought them into conscious awareness (effect *a*), and not some other cause (effect *d*).

Brenner does not discuss any reason to believe that other causes (effects *d*) were unimportant. Can we rule out that the patient's wishes to achieve more pleasure in her life or to adapt better to her social environment—the very things that led her to analysis in the first place—also led to self-analysis that affected her compromises between drive derivatives and defenses, superego demands and prohibitions? Can we say that talking with everyone else she knows and who wished to help her had no impact on her compromises? These are potential causes to rival Brenner's interpretative work as the cause of her shifting patterns of reaction formations, repressions and identifications that expressed her compromises between drive derivatives and defenses. Any efforts that eliminated some of these rival hypotheses (effects *d*) would strengthen the validity of the desired causal inference that the interpretative work was the effective cause (effect a).

**Did compromise-formation changes cause the improvements?**

Brenner seems committed to intrapsychic supremacy when he says that restructured compromise formations that became less pathological and more normal resulted in the eight outcomes he observed (effect *b*), but he does not supply reasons to rule out rival hypotheses that the eight outcomes resulted from other kinds of concurrent causes (effects *e*). For Brenner, no need exists to discuss the possibility that the eight outcomes resulted from other concurrent causes because 'everything in psychic life with which one has to deal as an analyst is a compromise formation' (Brenner, 1986, p. 39). He elaborates, 'To say everything is a compromise formation, means *everything*. Not just symptoms, not just neurotic character traits, not just the slips and errors of daily life, but everything, the normal as well as the pathological' (p. 41). So, for Brenner, any outcome is a compromise formation by definition.

It seems useful, however, to distinguish between compromise formations as conjectured mental mechanisms, expressing compromise among elements of postulated conflict, and observable outcomes. Boesky makes this point well:

> The notion of compromise formation is a theoretical construct which is intended to give a lawful explanation of human behavior. Thus we should distinguish observable clinical phenomena from the term *compromise formation* as a theoretic term introduced to organize our understanding of these observable data (1991, p. 19).

We therefore allow for the possibility that observable outcomes can be influenced by causes other than compromise formations (effects *e*) and that technique can affect outcomes for reasons other than its influence on compromise formations (effect *c*).

**Did psychoanalytic work affect the outcomes for reasons unrelated to Brenner's conjectured pathogenic mechanisms?**

The key question here is how much the outcomes that may have ensued from Brenner's interpretative work were due to the particular compromise-formation changes his analytic listening and theory picked out as deserving credit for the outcomes (sequential effects *a* and *b*), as against the outcomes resulting from other unrelated factors (effects *c*). We can think of effects *c* as condensing the impact of psychoanalytic treatment on all factors other than those designated as the ones influencing the outcomes by the analyst's listening and interpretation—in this case Brenner's conjectured conflicts and compromise formations, and their changes (sequential effects *a* and *b*).

Brenner does not discuss the possibility that his interpretative work, or psychoanalytic analysis more generally, could influence outcomes via effects *c*. Consequently, he does not discuss the plausibility that his interpretative work affected any other factors or mental mechanisms than the ones he singles out, and that these factors could plausibly rival the ones he selects as the eight outcome explanations.

For example, Brenner does not discuss potential rival pathogenic mechanisms drawn from related areas of psychoanalysis. From self-psychology (e.g. Leider, 1996): Could Brenner have been experienced as a soothing selfobject that stabilized the patient? From object-relations theory (e.g. Grotstein, 1996): Could Brenner's remarks have encouraged some split-off part of the patient's self to reunite with the rest? From interpersonal psychoanalysis (e.g. Mitchell, 2000): Could Brenner's remarks have shown the patient that she had a specific pattern of relatedness to him and to others, causing her to make the changes?

There is also considerable work, much of it more recent than Brenner's analysis, that could be examined for mechanisms potentially to rival those selected by Brenner: research on person schemas and role relationship models (Horowitz, 1991, 1998); aspects of mental control and self-regulation (Wegner and Pennebaker, 1993); cognitive behavioral mechanisms (Beck and Freeman & Associates, 1990); modern learning-theory concepts (Bouton, Mineka and Barlow, 2001); core conflictual relationship themes (Luborsky and Crits-Christoph, 1998); fundamental repetitive and maladaptive emotional structures (Dahl, 1991, 1998; Holzer and Dahl, 1996); storying and restorying (White and Epston, 1990); and mechanisms of automaticity and apparent mental causation (Kirsch and Lynn, 1999; Wegner and Wheatley, 1999; Bargh and Ferguson, 2000). Recent research in neuroscience suggests that cortical and sub-cortical brain activity may further illuminate pathogenic changes that respond to psychoanalysis and talk therapy, and thus provide explanatory rivals to current pathogenic mechanisms (Schwartz, Stoessel, Baxter, Martin and Phelps, 1996; Damasio et al., 2000; Mayberg et al., 2000). Brenner's analysis does not lend itself to addressing how much any of the outcomes he observed resulted from changes in compromises among the elements of pathogenic conflict he conjectured (sequential effects *a* and *b*), as against pathogenic changes drawn from any of these other areas of research (via effects *c*).

Effects *c* not only capture the influence pathogenic mechanisms have to rival those selected by the analyst, they also capture incidental effects that accompany the analytic treatment. Psychoanalytic technique includes a spectrum of identifiable components. Some of these components are picked out by the orientation of the analyst as more or less central to his/her definition of analysis, and ones thought to change (via effect *a*) the conjectured pathogenic mental mechanisms postulated by the analyst and, thus, reduce (via effect *b*) some sort of dysfunction. These are characteristic treatment components (Grunbaum, 1993). The other treatment factors not picked out in the spectrum of identifiable components of psychoanalytic treatment are incidental treatment components (Grunbaum, 1993). Additional hypotheses might hold that incidental treatment components could affect outcomes (via effect *c*) and, thus, plausibly rival more favored explanations that the characteristic components affected the outcomes (via sequential effects *a* and *b*).

For example, the culturally accepted trappings of the healing role and analytic environment provide numerous incidental treatment components: well-practiced rituals; a specialized vocabulary; a knowledgeable and systematic manner; a long-term and intimate relationship; a more or less strict frame defining payment and scheduling; and an often charismatic personality of the analyst. In these circumstances, it is easy to imagine that Brenner's patient wished to please him by changing in ways she sensed would earn his approval (i.e. some form of transference cure). It is easy to imagine that these aspects of the healing role could arouse hope, mobilize a sense of mastery, or allow the patient to follow advice given or inferred from the analyst, and thus reduce dysfunction. They could, therefore, (via effects *c*) plausibly rival analysts' more favored explanations that the characteristic components affected the outcomes (via sequential effects *a* and *b*).

**A summary and generalization: Enumerative inductivism and pseudodiagnosticity**

Four points summarize much of the previous discussion:

1) To conclude validly that some psychoanalytic intervention results in an outcome change, all other potential causes of this change (effects *e*) must be uncorrelated with the given use of technique.

2) To conclude validly that some psychoanalytic intervention results in a change of pathogenic mental mechanisms, all other potential causes of these changes (effects *d*) must be uncorrelated with the occurrence of the intervention.
3) To conclude validly that some change in pathogenic mental mechanism results in an outcome change, all other potential causes of the outcome change (effects *e*) must be uncorrelated with the changes in pathogenic mental mechanisms.
4) To conclude validly that some psychoanalytic intervention affects outcomes because it affects the conjectured pathogenic mechanisms via sequential effects *a* and *b*, these sequential effects must make a contribution that is independent of other rival effects of the intervention on the outcomes (effects *c*).
We doubt whether most causal inferences made and implied in case studies of how analytic treatment influences patients' pathogenic dispositions, and how changes in these dispositions influence analytic outcomes, satisfy these requirements for valid inference. Just as Brenner has done, analysts typically listen to their patient's material and then formulate hypotheses about the correctness of their interpretations, the effectiveness of their interventions, their pathogenic conjectures, the correctness of their reconstructions and the validity of their theoretical understandings. Support for their hypotheses comes from the accumulation of observations consistent with their premises. Insufficient attention seems paid to the issues we have raised. Arlow says, for example, 'Most of the time the intuitive work has been so efficient that a sense of conviction is immediate, gratifying, and accompanied instantly by recollection of the supporting evidence from the patient's productions' (1979, p. 203).

The problems of making valid inferences that we have pointed out in Brenner's report are generic to all hypothesis-testing strategies in clinical psychoanalysis that accumulate instances consistent with the analyst's hypotheses. These strategies have been called enumerative inductivism (Compton, 1990; Rubovits-Seitz, 1998). They are characterized by a relative emphasis by an analyst on searching for additional evidence, given that his/her hypothesis is assumed to be true, instead of looking for the same evidence given the possibility of alternative hypotheses.

The flaw with enumerative inductivism applies to any analytic hypothesis, regardless of whether that hypothesis pertains to effectiveness of some analytic technique, conjecture about a pathogenic mechanism, or something else, and where evidence of some sort is cited to support it: evidence of some effect (alleged) is observed. What caused it? Can you think of more than one thing that could have and that you don't have a principled way to exclude? If you can, then you do not have enough evidence to draw a causal conclusion.

Enumerative inductivism rarely has any formal assessment of the likelihood that the evidence observed could have resulted when the analyst's hypothesis was incorrect. This error from looking only at the likelihood of the observed effect, given that the hypothesis under consideration is true and neglecting the likelihood of an alternative hypothesis, is called 'pseudodiagnosticity' and is prevalent in everyday thinking as well as among psychoanalysts (see, for example, Kahneman, Slovic and Tversky, 1982; Dawes, 2001).

Solving the problems of enumerative inductivism and pseudodiagnosticity to improve case studies will not be simple. The solution must avoid these problems by always comparing the likelihood of the observed evidence given that the hypothesis under consideration is true against the likelihood of the same evidence given that rival

hypotheses are true. Another way to say this is that the methods must allow investigation of whether the observed evidence resulted not from the investigator's hypothesis being true, but from some equally plausible rival hypothesis.

Clearly there exists a tension between good clinical practice and research practice that squarely faces the problems of enumerative inductivism and pseudodiagnosticity. For example, an analyst who tries to do everything we suggest above, and at every pass in a treatment, would be utterly stymied. Not only would the task be mentally impossible, but it would be an inappropriate reaction leading to the analyst missing important clinical material.

However, thinking about some of the things we suggest might be quite appropriate for the analyst to do outside the analytic hour, when making case presentations or formulations of psychoanalytic treatment effects, and describing whether and why treatment affects the patient. These practices could usefully militate against over-drawing causal conclusions, over-reliance on one's favorite pathogenic mechanisms, and drawing invalid conclusions not supportable by empirical evidence that rules out plausible rival hypotheses. To do these things is, of course, the main recommendation of this paper.

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## Translations of summary

**Eine pfad-analytische Strategie zur Analyse der Ergebnisse psychoanalytischer Behandlungen.** Dieser Beitrag stellt eine pfad-analytische Strategie zur Analyse der Auswirkungen psychoanalytischer Behandlungen vor. Ein einfaches kausales Modell dient zur Analyse einer bekannten Fallstudie von Charles Brenner. Die Anwendung selbst dieses einfachen Modells hilft, valide Kausalfolgerungen zu präziseren, beleuchtet wichtige Aspekte des psychoanalytischen Prozesses und schafft eine Grundlage für die Weiterentwicklung des Modells.

**Una estrategia de análisis de trayectorias ["path analysis"] para analizar los efectos del tratamiento psicoanalítico.** Este artículo introduce una estrategia de análisis de trayectorias para analizar los efectos del tratamiento psicoanalítico. Se emplea un modelo causal simple para analizar un célebre caso de Charles Brenner. Incluso la aplicación de este modelo simple al caso agudiza las inferencias causales que podrían hacerse de manera válida, da relieve a importantes aspectos del proceso psicoanalítico, y construye una base para el desarrollo de otros modelos.

**Une stratégie utilisant des voies analytiques pour analyser les résultats du traitement psychanalytique.** L'article présente une stratégie utilisant des voies analytiques pour analyser les résultats du traitement psychanalytique. Un modèle causal simple est utilisé pour analyser un cas bien connu étudié par Charles Brenner. Même l'application de ce modèle simple à l'étude du cas affine la validité des inférences causales qui peuvent être déduites, souligne des aspects importants du processus psychanalytique, et établit les fondements pour le développement de modèles ultérieurs.

**Una descrizione procedurale analitica per l'analisi degli effetti di un trattamento psicoanalitico.** Quest'articolo presenta una descrizione procedurale analitica per l'analisi degli effetti di un trattamento psicoanalitico. Tale strategia impiega un semplice modello causale per analizzare un noto caso studiato da Charles Brenner. Proprio l'applicazione di questo semplice modello allo studio del caso accentua le inferenze causali che si possono validamente fare, evidenzia degli aspetti importanti del processo psicoanalitico e getta le basi per un ulteriore sviluppo del modello.

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