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Souvenir de la Salpêtrière: M. le Dr. Freud à Paris, 1885*

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1985 is the one hundreth anniversary of Freud's visit to Paris to study at the Salpetriere with Charcot. The impact of that visit on Freud's understanding of the mechanisms responsible for the formation and removal of neurotic symptoms is assessed. An evaluation of the progress made in understanding the basis of therapeutic effects since that time is attempted. It is argued that our understanding has remained defective because we have continued to look at the problem from the perspective inherited from Freud, a perspective that identifies specific causes and specific remedies incorrectly and fails to give placebo effects their proper recognition. The obverse of this perspective has led us to pay insufficient attention to the ways patients seek help or experience the processes by which they resolve their problems.

> If the Honourable College of Professors will award me the travelling grant, it is my intention to spend 3-4 months with Professor Charcot in Paris studying the wealth of material provided by the Salpêtrière Clinic, such a favourable opportunity not being available to me in the Departments of the General Hospital.

So in April 1885 did the 29-year-old Sigmund Freud begin his successful application for a Travelling Bursary from the University of Vienna (Leupold-Lowenthal, 1972, p. 18). Freud proposed continuing his histological work on the brain, but what came to interest him most at the Salpêtrière were Charcot's studies of hypnosis and hysteria. As we know, much of psycho-analysis originates in the French school of psychopathology to which these investigations of Charcot made such a great contribution. The journey which Freud's successful application allowed exactly 100 years ago was to prove as decisive in the development of our science as in his own life.

Of course, it is not possible to set out all the ideas Freud acquired at the Salpêtrière. I shall therefore confine myself to evaluating the contribution which Freud's stay in Paris made to his ideas about symptom formation and symptom removal, and I shall attempt to draw out the implications of what we have subsequently learned.

The mechanism of symptom formation

The most central things that Freud learned from the Salpêtrière concerned the mechanism of hysterical symptom formation. As important as these ideas were in themselves, their real significance came when he later placed them in a more general causal framework. I shall take up in turn the topics of the mechanism and the causal framework.

Work at the Salpêtrière established three things about the formation of hysterical symptoms: symptoms resulted from an unconscious transformation of ideas, symptoms retained the sensory content of the ideas, and the main characteristics of symptoms

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were determined by ideas. Charcot's own work led to the first formulation of a mechanism that explained how hysterical symptoms were created. Prior to him there had been nothing that could be called a coherent view. His studies of hysterical patients and his experiments with hypnosis established that hysterical symptoms resulted from ideas that had been transformed or realized by an unconscious mental process that had escaped control of the ego (Charcot, 1887/1889).

Charcot deduced that symptoms resulted from ideas from the similarity between hysterical symptoms and the alterations in function produced by suggestion under hypnosis. Ideas altered function directly when limbs became paralysed or anaesthetized as a result of explicit hypnotic suggestion. Sensory loss produced by unexpectedly striking an hypnotized patient had the same determinants. Here Charcot believed that the ideas conjured up in these simulated traumas acted as indirect suggestions. Thus, it was the sensation of momentary anaesthesia accompanying the idea of loss of movement caused by a blow that was transformed into a permanent loss of sensation. Indirectly suggested ideas were realized or transformed just as certainly as those suggested directly. Whether they resulted from direct or indirect suggestion the anaesthesias were often very delimited, separate suggestions causing sensation to be lost in such localised parts of the body as a hand, an arm or a part of an arm, a thigh, or a shoulder, but in no other. Examination of Charcot's illustrations (Charcot, 1887/1889, Lecture 22 and Appendix 1) shows how close the experimentally-produced symptoms were to the real thing. For example, the basic features of one of his directly suggested leg anaesthesias are practically identical with the anaesthesia of a patient who had been knocked over by a horse-drawn van, but had been otherwise uninjured.

Because neither hypnotic subjects nor hysterical patients knew how the alterations in function had been produced Charcot inferred the presence of unconscious processes. He believed that during "the intense cerebral commotion" of a trauma normal ego functions were in abeyance. Ideas arising at that time spread without hindrance thereby producing symptoms. He assumed that hypnosis induced an analagous cerebral state and that ideas suggested during it similarly spread without check.

The suggested ideas, said Charcot, lodged in the mind "like parasites" and, to account for the permanence of the symptoms to which this metaphor referred, he drew on what was probably the most important precursor to his work — the concept of dissociation. Dissociation notions had developed from the study of the many cases of alternating states of consciousness and multiple personality reported between 1816, when the first case to be fully described appeared (Mitchill, 1816; Carlson, 1984), and 1858 when Azam began his observations on Félida X. (Azam, 1876; Taylor & Martin, 1944). What makes Félida X. important to our story is not merely that in her primary state of consciousness she had no knowledge of what transpired in the secondary state: she was the first case in which it was noted that symptoms present in one state were absent in the other. In her primary state, Félida X. was morose and afflicted with hysterical deliria, convulsions, paralyses, and contractures, but bright, affectionate, and symptom-free in the second. Later workers like Mesnet (1874, cited by Taine, 1878), Dufay (1876), Camuset (1882), and Bourru and Burot (1885) showed that the different states could be brought about in lawful ways and that the presence of any symptoms was dependent upon which of the states was manifest. Beginning in 1882 Pierre Janet (1889) had made similar observations and went on to develop a therapy that used direct hypnotic suggestion to remove the symptoms after the patient had been placed in the appropriate state.

Charcot took it that it was always the case that the symptoms of hysteria were maintained within an embryonic *condition seconde*. Because it was the only view that accounted for the isolation of the symptom, Freud also accepted it completely, at least initially. We can even be reasonably sure that it was this concept that enabled Breuer to develop his hypnoid explanation of Anna O.'s symptoms, and that it was *via* Freud that this and related concepts reached Breuer from Paris. Breuer's original case notes (Hirsch-

müller, 1978) contain no hint of such notions, and not even Azam's earlier terms of "état prime" and "état seconde" are used. In the version published much later, however, we find such terms from the French school as Charcot's *condition seconde* (Breuer & Freud, 1893, 1895). Traumatic experiences occuring in Anna O.'s secondary or hypnoid state were realized as symptoms, either because they intruded into the primary state or were manifested during the revival of the second state. Freud himself went so far as to make the potential for forming a secondary state an essential precondition for the development of hysteria (Breuer & Freud, 1893; Freud, 1893a). Later he gave this role to the proclivity for repression and conversion.

If dissociation was the most important precursor to Charcot's thinking, Janet's thesis about symptoms was the absolutely critical successor. We can see that it follows from Charcot's conclusions that the sensations called up in the experimental and natural traumas had to be reflected in the symptom. In fact, the sensory content of the symptom had to be the *same* as the sensory content of the traumatic experience or the suggested idea, and Freud retained this notion in his concept of "determining quality" (Freud, 1896, pp.192-195). Well before this, Janet (1892) had drawn out a further implication of the point in his thesis that the characteristic details of hysterical symptoms were also determined by ideas. He noted that it was not the hand or arm in the anatomical sense which was paralysed or anaesthetic. Comparision of organically based anaesthesias with their hysterical counter-parts showed the characteristics of the altered function in hysteria to be at variance with what was known about the innervation of the anaesthetic organ. The distribution of the anaesthias was, Janet observed, physiological or functional rather than anatomical. But it was a physiology of a peculiar sort — an everyday physiology based on the ordinary *concept* or *idea* of the hand or arm rather than as those organs were known to science. In hysteria, the affected hand was the organ between the wrist and the finger tips and the anaesthetic arm was that which existed between the wrist and the shoulder and back.

Freud developed Janet's notion by arguing that patients could not form associations between their ideas of their hands or arms and their other ideas. He proposed that the former were cut off *because* of their large quota of affect[1] and immediately emphasized the therapeutic consequences:

> *The arm will be paralysed in proportion to the persistence of this quota of affect or to its diminution by appropriate psychical means . . . it can be shown that the arm is liberated as soon as this quota is wiped out.* (Freud, 1893b, p.171)

It was in this way that Freud first introduced emotion, more correctly "the quota of affect", into his theorizing. Janet had presented his ideas to a clinical meeting at the Salpêtrière on 11 March 1892, and they were published in May of that year. Freud's first reference to emotion in hysteria, his first use of the term "abreaction", and his first mention of working and publishing with Breuer occurs in his 28 May letter to Fliess, that is, within days of Janet's paper appearing (Cf. Freud, 1892).

Janet's thesis was therefore critically important. It provided the impetus to the first step in Freud's independent causal theorizing about hysteria. Janet's thesis, and only that thesis, allowed what could be said about the mechanism of hysterical symptom formation to be lifted from what was virtually the descriptive level of Charcot's theory and placed within a more sophisticated theoretical framework.

Psycho-analytic mythology has it that when Freud left Paris in February 1886 he returned to Vienna afire with enthusiasm for the treatment of hysteria and soon proved that Charcot's "traumatic hysteria" was the model for all hysteria. He revived "Breuer's method" for treating hysteria and by strictly scientific methods of enquiry, free of any prejudice or preconception, soon established that traumatic sexual experience was the cause. Three points show that this myth, like all myths, bears very little relation to the facts.

First, it was 1889 before Freud first adopted any version of Breuer's method, and then only after Janet and Delbouef had independently described similar treaments (Macmillan,

[1]This blocking role of affect appears to derive from Jackson (1879-1880), whose work Freud had given close attention in preparing his own *On Aphasia* the year before (Freud, 1891/1953).

1979). Second, although Freud had formed the belief that the "talking cure" that Anna O. had developed for Breuer was a sovereign remedy for hysteria it was nothing of the kind. Anna O., as Ellenberger (1970, 1972) has stressed, was as typical a case of somnambulism in which the *somnambule* dictated the treatment as can be found in the nineteenth century literature. Even if she had been cured (which she was not) the "talking cure" was by no means the specific remedy for hysteria that Freud took it to be (Macmillan, 1977a). Third, well before going to Paris, Freud had already formed some very dubious ideas about the determinants of psychological phenomena that made his method of investigation rather less than objective. During his time as Meynert's *Secundärarzt* Freud seems to have accepted as an implication of his chief's view of associations the conclusion that inadvertent suggestion could not divert a train of associations. For Meynert, associations had a physiological basis. If a train of thoughts starting at A ended at Z it was because the physiological processes underlying the intermediate elements had once occurred together and not because the train was under control of an indirect suggestion (Macmillan, 1977b).

This last point has an especial importance because it meshed so well with Charcot's view that unconscious suggestion was not a determinant of hysterical and hypnotic phenomena. According to Charcot, the very uniformity of hysterical and hypnotic phenomena, at varying times and in varying places, meant that they could only have a physiological basis. Freud used exactly this argument to defend Charcot against the charge that the phenomena demonstrated at the Salpêtrière were due to suggestion:

> If the supporters of the suggestion theory are right, all the observations made at the Salpêtrière are worthless . . . every physician would be free to produce any symptomatology that he liked . . . We should not learn . . . what alterations in excitability succeed one another . . . we should merely learn what intentions Charcot suggested (in a manner of which he himself was unconscious) . . . a thing entirely irrelevant to our understanding alike of hypnosis and hysteria. (Freud, 1888, pp.77-78)

Freud's later defence of his own methods for enquiring into the origins of symptoms against the charge that they influenced patients unduly was essentially an extension of these ideas of Charcot and Meynert (Breuer & Freud, 1895; Freud, 1896).

I have foreshadowed that Freud's souvenirs of the Salpêtrière had their greatest significance as components of a more general causal framework that Freud began developing for exploring the specific causes and remedies of the neuroses. It is to that framework that I now turn.

Germ theory and causes

Freud's more general causal framework was an adaptation of the germ theory of disease proposed by Pasteur and Koch. Most of those working in the area of therapy, whether as therapists or research workers, have adopted a formally identical causal and remedial orientation. After examining Freud's adaptation of germ theory I shall argue that its limitations are largely responsible for the limitations of our understanding of what happens in the various forms of psychological treatment.

That Freud derived his methods for investigating the causes of neuroses from Pasteur's and Koch's germ theory was first explicitly pointed out by Carter (1980)[2] although the indebtedness to Koch had been noted earlier by others (Macmillan, 1976). According to germ theory, diseases came about through infection by microbes. The specific organism responsible for a given disease was identifiable through the procedures enshrined in Koch's postulates: first, the suspect microbe had to be different from any other by taking up staining material in a unique or specific way; second, it had to be found in every instance of the given disease and not in any other; third, innoculation of a culture from it had to produce the disease experimentally. Only this last criterion identified the organism as part of the sufficient conditions for the disease; otherwise all that

[2]Until Carter drew attention to it, the pioneering nature of this aspect of Freud's work had gone unnoticed. Carter's evaluation is not impugned by the fact that he supposes Freud to have first developed the conceptualization for hysteria and not, as I have shown, for the actual neuroses.

had been described were the necessary conditions.

Freud relied especially heavily on Koch's postulates in trying to find the *specific causes* of both of the very first neuroses he investigated — the actual neuroses of neurasthenia and anxiety neurosis.[3] He also used Koch's reasoning in linking the specific causes with *preconditons* and *concurrent causes* in his "aetiological equation". However, as we see from Table 1, Freud did not bother with an equivalent of Koch's innoculation condition.

Freud's Adaptation of Koch's Postulates

Koch	Freud
Specific staining	Specific sexual practice
Found only in the one disease	Found only in the one neurosis
Innoculation produces the disease	Specific practice not found in the healthy ("control")*

*No formal investigation actually conducted by Freud

This would have meant searching for cases in which the presumed cause might be present but its effects absent. That is, Freud did not examine normal subjects — 'controls' if you wish — to see whether the presumed cause was at work in them. Although Freud spoke of the specific causes as "sufficing" to produce the neuroses only the necessary conditions had been identified (Macmillan, 1976). For example, Freud held the specific cause of anxiety neurosis to be incomplete sexual gratification primarily because he claimed it to be present in every instance. Naturally, nothing solely identified in this way could be a specific cause.

Freud's unwarranted conclusion about the role of sexual factors in the actual neuroses was transformed into an equally unwarranted expectation that a specific sexual factor was at work in hysteria (Macmillan, 1976, 1977b). Initially he had thought that the quota of affect came from the emotion of *any* sufficiently forceful traumatic experience. Gradually he restricted its source to libido, the energy of the sexual instinctual drive. Having suspected that Freud had begun an explicit search for a sexual basis to hysteria in the second half of 1983, I was therefore very pleased to see this dating directly confirmed in a letter translated for the first time in Masson's recent edition of the complete Freud-Fliess correspondence. Nothing in anything Freud wrote connects sexuality with hysteria until a letter of 29 September, 1893:

> I happen to have very few new sexualia [his term for his investigation of sexuality in the actual neuroses]. I shall soon start investigating hysteria. (Masson, 1985, p. 56)

Almost inevitably the vigour with which Freud prosecuted his investigation interacted with his ideas about determinism and led to the ill-fated childhood seduction theory (Macmillan, 1977b). According to this hypothesis the specific cause of hysterical symptoms and the determinant of their sensory content was the traumatic seduction of children into perverse sexual activity by adults, usually their fathers.

Freud's conceptualizations of the symptom as reflecting the traumatic experience and of the seduction memory being dissociated from consciousness by repression were, of course, simple consequences of the ideas he acquired at the Salpêtrière. What was original was his placing this cause within the framework he developed for the actual neuroses. In doing so, he again claimed it was unnecessary to look for cases in which the presumed cause was present but the hysteria absent. He said that all he needed to show was the presence of the presumed cause in every case (Freud, 1896, p.209) and once again his confusion of necessary with sufficient conditions led to a misidentification of the cause.

Freud held the childhood seduction theory in this form for only a few months before abandoning it. As we know, he eventually concluded that what had been recalled were fantasies. With but slight regard for observable fact (Macmillan, 1980; Lindner, 1879-1880/1980), he then invented the notion of a perverse childhood sexual instinctual drive capable of creating fantasies that contributed to symptom formation but were realistic enough to have been mistaken for the memories of real events.

Souvenirs of the Salpêtrière are still readily discernable in this new conceptualization. First, Freud placed as much weight as Charcot on prior experiences as determinants of symptoms although, for him, they were internally driven rather than externally

[3]From Freud's contributions to the American medical press we know how fully he understood the significance of these postulates (Grinstein, 1971).

caused. Second, Freud gave the same importance to sensations as Charcot although, for him, they were generated auto-erotically and transmuted *via* fantasies before becoming symptoms rather than by realizing themselves automatically. Finally, both drew similar implications from the uniformity of the phenomena they observed: Charcot that there was a change in physiological functioning, Freud that a biological instinctual drive was at work. We have also seen that Freud, like Charcot, rejected unconscious, inadvertent suggestion as a basic determinant of clinical phenomena. Charcot had been wrong, of course. In the upper left hand corner of the famous painting of one of his lectures by Brouillet there is a sketch showing the next of the regular stages in the development of the hysterical attack — the famous *arc de cercle* — that the patient being demonstrated is about to fall (Ellenberger, 1970). Charcot's disregard of the effects of this and other kinds of indirect suggestion was, even in his own day, quite notorious. Freud's was to prove fatal to both his causal and therapeutic propositions.

Souvenirs of the Salpètrière

	Charcot	Freud
Past experience	External	Internal
Sensation-symptom link	Realized via ideas	Transmuted via fantasies
Uniformity implication I	Physiological function	Biological drive
Uniformity implication II	Suggestion not important	Suggestion not important

Before concluding this discussion of Freud's adaptation of germ theory, I should perhaps stress again that what Freud learned about mechanisms at the Salpêtrière gave him at most an *orientation* toward a causal search. Not until returning to Vienna did he begin to apply Koch's postulates. When he did so he actually tried to move away from the practice at the Salpêtrière (and almost everywhere else) of defining neuroses by enumerating their symptoms. Freud's placing of his mechanism of symptom formation in a more general causal framework indicated an approach quite different from Charcot's. Charcot was not responsible for Freud's 'germ theory' framework.

Specific remedies

Early in his work on neurasthenia and anxiety neuroses Freud began prescribing to his patients what I shall call a *specific remedy* — the adoption of more normal sexual practices. As we have seen, once he had added affect to Janet's thesis, he proposed a remedy just as specific for hysteria: the abreaction or discharge of the quota of affect. Where the idea that remedies might be specific actually comes from I do not know, but a simple derivation from germ theory seems likely. Each of Freud's remedies was directed toward removing or negating a specific cause. Now, the fact that his remedies worked in the way they did was reason enough for Freud to rule out suggestion as a therapeutic factor. As Grünbaum (1984, pp. 177-189) has observed, what prevented Freud from ruling it out from catharsis was the very closeness of the connection he saw between the mechanism that created the symptom and the results of abreactive therapy (Breuer & Freud, 1893; Breuer & Freud, 1895, pp. 255-265; Freud, 1893a). But, if the causes were not specific neither could be the remedies. Nor could suggestion be ruled out. We now know that Freud was wrong not to see that suggestion was the main component of the advice he gave those of his patients suffering from the actual neuroses (Oerlemans, 1949) and no less an authority than Ferenczi (1908/1952) equated catharsis cures with suggestion.

After Freud located the new cause of hysteria in the vicissitudes of an internal drive, he set his patients a therapeutic task quite different from abreaction and more complex than it. What they now had to do was to transfer on to their psycho-analysts the combination of positive and negative feelings which the infantile drive had once caused them to have toward their own parents and so reproduce an infantile form of their neurosis. Re-creating infantile feelings in this way seemed to lead patients to an understanding of the forces that had shaped them and it seemed to be the resolution of the transferred neurosis that removed their symptoms. Again Freud ruled out suggestion as the basis of the therapeutic effects and again he was as wrong as he had been previously about the effect of catharsis on the seduction 'memories'. Freud's failure to apply Koch's postulates

completely in investigating causes was matched by his failure to rule out 'suggestion' from his therapies.

The equivalence of therapeutic effects

Freud argued that what were ordinarily called suggestion therapies, especially hypnosis, were effective to the extent that they contained significant transference components. Like every therapeutic zealot, Freud believed that the real truth resided only in his scriptures. Despite this, and similar attempts by other psychotherapists, a monolithic explanation of therapeutic effects proved impossible. Gradually the view emerged that all therapies worked to about the same extent and that their effectiveness was due to some component other than the specific ones nominated by their protagonists. As far as I can tell, this proposition was first advanced by Saul Rosenzweig (1936). No comparative studies of the effectiveness of different kinds of psychotherapy had by then been carried out, so that Rosenzweig had no choice but to begin with the therapeutic folklore that "It has often been remarked upon that no form of psychotherapy is without cures to its credit" and to make what he recognised as the doubtful assumption "that all methods of therapy when competently used are equally successful". Rosenzweig's conclusion was implied in his introductory quotation, "At last the Dodo said, '*Everybody* has won, and *all* must have prizes' ". Nearly forty years later when Luborsky, Singer, and Luborsky (1975) questioned the Dodo's pronouncement they found the differences between therapies to be only marginal. In any case, what differences there are may be due to the theoretical allegiance of the therapists rather than to their specific techniques (Berman, Miller, & Massman, 1985).

Did the non-runners really deserve prizes? Perhaps any effects of psychological or behavioural treatments were due to their non-specific or placebo properties. Prior to the 1940's no study of any form of psychotherapy, including psycho-analysis, took these non-specific effects into account. Systematic interest in the psychological effects of placebos arose in the 1940's and the early 1950's in connection with the first psychotropic drugs like chlorpromazine (Shapiro, 1960). At that time little or no attention was given to the mechanism of the changes caused by the placebo itself. The psychological or social psychological context was simply not of interest despite medical folklore having it that the efficacy of many remedies depended in large measure on the attitude of the prescribing physician. Even after Feldman's (1956) demonstration that differences in the proportions of psychiatric patients responding to chlorpromazine were related to the enthusiasm of the psychiatrists, the placebo effect was still not studied in its own right (Whitehorn, 1958). Although Feldman's finding has been so often confirmed that it is now a commonplace (e.g. Uhlenhuth, Canter, Neustadt, & Payson, 1959; Feldman, 1963; Rickels & Cattell, 1969) an explanation for it has yet to be formulated.

No one doubts the existence of placebo components in psychological treatments or that they are of considerable magnitude (Shapiro & Morris, 1978, p.369). Bergin's (1978) recent evaluation of studies of therapy using placebo control groups shows that between 20% and 50% of controls are improved or cured. The size of the effect depends to a large extent on how closely the patient sees the placebo procedure as resembling an active treatment (Frank, Gliedman, Imber, Stone, & Nash, 1959; Paul 1966, 1968; McReynolds, Barnes, Brooks, & Rehagen, 1973; McCardle & Murray, 1974). From time to time the question has even been raised whether placebos might not be the treatment of choice for some conditions (Whitehorn, 1958; Fish, 1973; Frances & Clarkin, 1981).

We can be fairly certain that some part of the results of psycho-analysis is due to placebo effects even though few studies have evaluated psycho-analytic treatment proper and in none have placebo controls been used. Further, therapists of almost all persuasions have followed Freud in disregarding the need for placebo controls (Wilkins, 1984). They have thereby continued to misidentify the basis of therapeutic effects.

Rather than arguing for the need to use placebo controls or to investigate placebo effects directly, Rosenzweig foreshadowed the two substitute methods that have been subsequently used. He asked what the apparently diverse therapies had in common

that made them equally successful. He listed as possibilities such implicit factors as "social reconditioning" and catharsis, the "indefinable effect of the therapist's personality", the less implicit role of "the formal consistency of the therapeutic ideology" for structuring the patient's disorganised experience, and the similar potency of alternative interpretations of the same behaviour for modifying personality structure and so beginning "the work of rehabilitation". What we see in this list are the two methods of "combination" and "distillation" both of which require a search for features common to different therapies. "Combiners"pick out one or two of the elements they regard as essential from each of a number of different therapies and mix them together into a single system, for example, social reconditioning and catharsis. "Distillers" seek some kind of essence in variant forms of therapy such as the therapist's personality. Each method involves selection from among aspects of therapy already identified and it is clear that it is based on the unlikely assumption (or at least an untested one) that the set of elements or the essence of the therapy is in some sense *already known* but incorrectly appreciated.

Wolberg (1954), one of the first of the combiners, believed therapies to be alike in that all therapists gave emotional support, provided cathartic release, helped in handling stress and altering defences, and assisted in self re-evaluation. As you see, he chose only (and indeed he could not do otherwise) from already known favourites from the pre-behavioural era. Most often the ingredients chosen by combiners result in pretty insipid dishes, as Prochaska's recent "trans-theoretical therapy" illustrates (Prochaska, 1979; Prochaska & Di Clemente, 1982). After analysing some eighteen therapy systems, Prochaska concluded that most involved, among other things, some process of consciousness raising. Thus, in psycho-analysis the therapist sought to follow Freud's famous slogan and make the unconscious conscious. Prochaska has it that this is just one of a number of equivalent procedures which raise consciousness by giving patients feedback about their actions or educating them about the impact of environmental events. In so grossly missing the point of Freud's famous slogan, Prochaska's equating of overcoming repression with education or feedback points all-to-clearly to the main weakness of almost all of the work of the combiners: superficiality.

"Distillation" has resulted in three products: first, catalogues of characteristics differentiating patients who do well from those who do not; second, the somewhat shorter lists of the differentiating personal characteristics of therapists; third, the factors in the relationship which develops between therapists and clients when therapy is successful. Little has emerged from these studies.

Studies of patient or client characteristics have led to the conclusion that outcome is best predicted by severity and duration of 'illness' and acuteness of onset. This conclusion has not been displaced by any of the candidates from the more personal, but mind-numbing catalogue of socio-economic status, sex, body-sway suggestibility, expectation, liking for therapy, motivation, and diagnostic test results including Wechsler, Rorshach, TAT, MMPI, Sentence Completion, Locus of Control, and a swag of anxiety scales (Garfield, 1971; Lambert, 1982, Ch. 2). Because none of these patient characteristics is related to therapeutic success none can be at the core of the placebo effect.

Nor are therapist qualities responsible. Whitehoren and Betz (1954), who seem to have begun this line of investigation, showed that psychiatrists successful in treating schizophrenics (Type A) could be differentiated from the less successful (Type B) on, of all things, the Strong Vocational Interest Blank or scales derived from it (Betz, 1962). Despite many subsequent studies, Razin's (1977, p. 320) review emphasised the poor predictive power of the distinction:

> *The A-B variable is not a powerful predictor of any important process or outcome parameters in real, ongoing therapy.*

Rogers' (1957) specification of the necessary and sufficient conditions for change as the therapist's accurate empathy, non-possessive warmth, and genuiness has been disposed of just as emphatically by Mitchell, Bozarth, and Krauft (1977, p. 483):

> *empathy, warmth, and genuineness are related in some way to client change but . . . their potency and generalizability are not as great as once thought.*

Even if we accept Lambert's (1982, p.68) conclusion that there is a "modest" effect on therapeutic outcome, the characteristics of successful therapists do not explain placebo effects.

Are the effects due to the relation which develops between therapist and patient? Emphasis on the relationship seems to have begun with Black's (1952) listing of "common factors" or "universal elements" in the psychotherapies. For him, therapists had to generate rapport and provide acceptance and support within the context of a limited emotional relation in which the therapist maintained superiority. However, most of those investigating the therapist-patient relationship seem to think that its essence is determined by the Rogerian characteristics of warmth, genuineness, and empathy (Truax, 1963). As one might expect, the review by Mitchell et al. (1977) also dismisses that possibility. Placebo effects cannot be due to the relationship.

Even were it the case that the therapeutic relation was of primary importance there is an hiatus between it and the effects it supposedly produces. Crudely put, it is by no means evident that being nice to patients should affect their symptoms. Something has to bridge the gap between the supposed conditions for change and the change itself. Candidates like Rogers' self-actualizing tendencies do not begin to meet the need; they are too profoundly mystical in scope and operation for the explanations they generate to be taken seriously.

Trying to identify the elements common to therapy or to distill its essence by studying patients, therapists, or the relation that develops between them has failed. We are as much in the dark as ever about placebo components and placebo effects.

The process of change

What then brings change about? In the investigations so far considered there is an absence of any genetic account of how either active therapies or placebos make their impact on the patient. For the psychological therapies the best we have are lists of more or less permanent social, psychological, illness, or relationship characteristics having more or less some connection with each other but very little to outcome. Liberman (1964) found the same shortcoming in placebo research in medicine, especially in the concept of there being a personality type that defined the placebo reactor. Is it the case, perhaps, that in fixing on the readily describable permanent properties of the individual we have failed to see what is really going on in both the placebo response and in active treatment? I am, of course, well aware that many investigations of what is called 'process' have been conducted (Luborsky & Spence, 1971; Truax & Mitchell, 1971). However, most of these are inward looking studies, devoted to the minutiae of the internals of particular therapeutic systems and not to identifying processes related to outcome (cf. Robbins in Bergin & Strupp, 1972, pp. 140-141). With a double turning of Oscar Wilde's famous epigram, we may say that in the outcome investigations we have the unchangeable in pursuit of the unpredictable and in the process studies the introverted in contemplation of the inconsequential.

Only one type of 'process' account comes near to what I think the area demands and it is that exemplified by Frank (1961). It was Frank's merit to place psychotherapy within the context of other healing and persuasive procedures and on the basis of his follow-up studies to make this extremely significant suggestion:

> the function of psychotherapy may be to *accelerate* a process that would occur *in any case* as the result of a patient's interactions with help-givers. (Frank, 1961 p. 214. Italics added)

Frank (1974, cf. Bergin & Strupp, 1972, pp. 109-111) has since repeated this idea and made its underlying premise explicit: at the time of seeking help, the patient has *already* begun to change. The wise therapist fosters this self-initiated process.

Although many prominent workers in the field, for example, Bergin and Suinn (1975) and Lambert (1982, p. 32), have endorsed the importance of changes within the patient, the notion does not seem to have influenced research thinking. Where it has, the results have been rather vague conceptual analyses rather than empirical investigations (e.g. Wilson, 1980). Yet, if the notion is correct, we should no longer delay trying to determine what it is that leads patients to seek help,

for this ought to throw light on the nature of the internal process. We ought also to be studying the impact of the initial help-seeking for this may tell us what acts to inhibit or promote the change.

Here we strike difficulties. There is a paucity of formal studies on the initiation of treatment. Neither Gurin, Veroff, and Feld (1960), nor Roth, Rhudick, Shaskan, Slobin, Wilkinson, and Young (1964), nor Garfield (1980) cite much literature on the subject. Garfield's (1980) chapter on initiation is especially interesting in that he gives only three references in contrast with the very full documentation he provides for his other chapters. Similarly, neither Wimberger and Millar (1968), in their earlier investigation of the effects of initial contact, nor Roth et al. (1964) in their study of initial treatment conditions, cite many other studies. You will therefore have to pardon the intrusion of a personal note into the next few remarks.

When I worked at Travancore Clinic I was struck several times by the therapeutic power of appointment making. At their wit's ends, clients would seek urgent appointments and, while most kept to the times given, some did not. Occasional and quite fortuitous follow-up among those who did not sometimes showed that improvement had nevertheless taken place. It did not seem to matter whether the problem was one of family conflict over the care of a severely retarded adolescent, or the difficulties of controlling the behaviour of an over-active pre-school child, or the near breakdown of a marriage. After the decision to seek help had been made the problem "disappeared".

Therapeutic power seems also to inhere in the diagnostic process. Although I have no recollection at all of the problem itself, I still vividly recall an initial appointment with a mother and father who practically drowned me in a flood of problems. At the end of our time all I did was to list the issues which they seemed to have told me about as topics for future exploration. However, they did not keep their next appointment and I did not see them again until a chance social meeting some years later. Much to my surprise, they thanked me for my help, attributing the solution of their problem to the very cogent advice they said I had offered on that one occasion. Of course, I am as sure that I am not a miracle worker as I am that every one who has worked in a clinical or quasi-clinical setting has had similar experiences.

Many people "know" about the therapeutic power of appointment making and of diagnosis, but there are very few studies that explore either of these first public acts of the patient or investigate the reasons for seeking help. Occasionally there are incidental comments like those of Saslow and Peters (1956) and Endicott and Endicott (1963) who reported that "several" of their patients had been helped by a single interview. Significantly, some of Endicott's and Endicott's patients saw their initial *evaluation* interviews as *treatment*. Beiser (1976) thought the annual visits paid to his subjects might have had the same effect, but in only two other studies does this reaction seem to have been considered further. After spending thirty days on the waiting list, the ten of Wimberger's and Miller's (1968) forty child patients who were most spontaneously improved were compared with the ten worst. The parents of nine of the improvers but none of those of the non-improvers had seen the intake interview as *therapy*. What is striking is the discrepancy between the minimal advice given at intake and the magnitude of the behavioural change. Malan, Heath, Bacal, and Balfour (1975) make the same point: after a single diagnostic interview, their otherwise untreated neurotic adults recovered symptomatically and dynamically to "a depth and extent that none of us had believed possible". What is the bearing of this odd reaction to diagnosis on what happens in therapy?

What happens in therapy?

The general lines along which I believe we should interpret these observations on diagnosis and associated clinical lore is as follows. After a period of barely coping with their difficulties patients begin to want to be different, to be better adjusted, or to be rid of their symptoms. Moreover, this need is present whether they have simple problems or complex ones, like the dissociated symptoms so characteristic of nineteenth century hysterics, or the habitual self-defeating patterns of behaviour and addictions of our own time. Clarifying problems and making

appointments mark a critical point in satisfying this need. For some, these actions are enough — they are then able to work out solutions for themselves. For others, some contact or perhaps some minimal advice is needed to help them find the right direction. For those who require formal treatment it may still be the case that it does not much matter what therapeutic techniques are used. Like the most effective placebo conditions, what matters is how patients see the treatment method. I believe it likely that successful therapy offers these patients a kind of temporary support while they move toward their goal of being different, better adjusted, or symptom free. If you like, we give these patients a frame upon which they lean while learning to walk in a new way. We should recognise that many are able to do so without any help at all. Of those 464 subjects in the study by Gurin et al. (1960) who admitted that they had at some time felt close to a nervous breakdown only 29% sought formal help while 69% relied on self-help. It may be that only a minority of potential patients require the use of our walking frame.

If seeking help and formulating problems indicates an important stage in the patient's desire to change we should not be puzzled by the reaction to diagnosis, or to *the phenomena of the waiting list,* or perhaps it is its *paradox:* waiting list controls do fairly well but less well the longer they have to wait before treatment is begun. Lambert's (1976) analysis of waiting list data reached what he himself saw as a somewhat misleading median recovery rate of 43% because improvement rates as high as 70% have been found (Wallace and Whyte, 1959; Jurjevich, 1968; Schorer, Lovinger, Sullivan, & Hartlaub, 1969; Gottschalk, Fox, & Bates, 1973). Of the waiting list controls in Rogers' and Dymond's study of less serious counselling problems, four out of nine were regarded as successes and one a moderate success (Gordon & Cartwright, 1954). Waiting list patients do sometimes turn to others for help but this can hardly be termed "treatment". In any case, as the results of Christensen, Birk, and Sedlacek (cited in Lambert, 1982, p.9) show, only about one half of improved patients do so. Delays in moving from the waiting list to active treatment are negatively related to final status, as Gordon and Cartwright (1954) also found. Generally the longer the delay, the smaller the change (Roth et al., 1964; Uhlenhuth & Duncan, 1968). I recognise that these facts are capable of other interpretations, but they are at least consistent with the view that most patients are particularly ready for treatment at the time they seek help.

To some extent, my thesis resembles Bergin's and Strupp's (1972, p. 17) appreciation of the role of "self-control", Bandura's (1977) notion that therapy ought to be directed toward increasing "self-efficacy", and Goldfried's (1980) formulation of psychotherapy as coping skills training, but it is much broader than any of these. To begin with, I would want to endorse Bowers' (1980) warning: reducing the main issue to the extension of control, especially through rational methods, may state the task too narrowly. Second, while it may well be the case that when the neurotic or maladjusted choose to be different they wish to bring some part of their behaviour back under their own control, I think a more fundamental process is at work. Sartre advanced the proposition that people *choose* to be neurotic. While I do not think that at all, I do believe that patients make more-or-less conscious choices to no longer live in the old way.

I admit that this is rather vague. But there are other than anecdotes. The only process variable so far identified that predicts outcome is Gomes-Schwartz's (1978) combined measure of the extent to which patients actively interact with their therapists, for example by initiating discussions with them and are not negative, hostile, or distrustful of them. Gomes-Schwartz named this variable "patient involvement" and it accounted for some three to four times more of the outcome variance than either the quality of the therapeutic relationship or the extent to which psychodynamic hypotheses were explored. What makes the finding especially significant is that it held for the combined comparision groups treated by psycho-analytic therapists, client-centered therapists, and non-professional counsellors.

We may also note two almost identical findings about the factors that patients think important to their improvement. Sloane, Staples, Cristol, Yorkston, and Whipple

(1975, pp. 206-207) compared the judgements of psycho-analytic and behavioural therapies by neurotics while Cross, Sheehan, and Khan (1982) contrasted opinions about insight (transactional and gestalt) and behavioural (social-learning) oriented treatments among non-psychotic outpatients. Within each study, patients in the treatment groups improved to the same degree and in both studies they retrospectively rated the same factors as of almost equal importance. Even though the rank ordering of the factors in Cross et al. creates a different impression, the ratings of the factors were all quite high. What patients thought important were: being helped to understand one's problems and one's self and talking to an understanding person; the personality and skill of the therapist; and practicing facing bothering things, being helped to relax, and being encouraged to shoulder one's responsibilities. With caution due to its analogue status, one may add the conclusion of Strong, Wambach, Lopez, and Cooper (1979) that interpretations identifying causal factors about which clients can do something produce more change than interpretations that cannot be so acted upon.

Now we come to the two data sets of most relevance: Malan's et al. (1975) detailed study of "spontaneous" change in neurotics and Quarrington's (1977) little known observations on stutterers who had managed to rid themselves of their problem. Malan et al. looked in some detail at the eleven of their 45 untreated patients who had improved after only a single initial interview. Two features stood out. First, the interview had often provided patients with an understanding of the bases of their behaviour ("insight") or provoked its development. Second, the interview had caused many patients to take "responsibility" for their own lives. This second factor is also very evident in Quarrington's 27 adult chronic stutterers, none of whom had ever been treated in any formal sense. While in seven, no definite mechanism was discernable — they just seemed to begin speaking normally — in the remaining 20:

> about half claimed that changes in their self-attitudes were of primary importance and about a half attributed their recovery to some new approach to the mechanics of speaking. (Quarrington, 1977, p. 77).

Quarrington went on to say that all twenty were actually mistaken. *All* of them had altered their attitudes and *almost all* had adopted a different approach to speaking. Now the "mechanics of speaking" were peculiar in the extreme. "Talking more clearly", "speaking slowly", "speaking in a deeper and firmer voice", which are the instances cited by Quarrington, have at best only a marginal relation to the principles on which a speech pathologist might base treatment or psychologist find intelligible. But, from the perspective I am sketching, the change is perfectly understandable. Well before changing their speech each had successfully met some considerable challenge in their lives that had enhanced their self-worth. The challenge itself had not involved any need to alter their speech nor were there any later incentives. Nevertheless, as a consequence of their successes:

> the majority of subjects *somehow felt less helpless with regard to their stuttering and decided to do something about it.* (Italics added) (Quarrington, 1977, p. 77).

Not surprisingly, Quarrington raised the possibility that the benefits from behavioural treatments might be due to these "non-specific" effects. It would not be inappropriate to identify Quarrington's non-specific factor as an attempt by his stutterers to take responsibilty for themselves and achieve fluency.

I believe that we ought to investigate whether the patient's taking responsibility is not the most central feature of our therapies. Quarrington's remarks strongly imply it to be the key to the behavioural therapies, a view which seems to be shared by others. For example, after Turner and Ascher (1979) had found paradoxical intention, stimulus control, and progressive relaxation to be equally effective in the treatment of insomia, they proposed that all three methods worked by "investing the client with the perception of self-control or self-attribution". Some remarks by Ross and Olson (1981) incidental to their discussion of placebo effects also interpret the basis of prescribing the patient's symptom in the same way. The more patients practise their symptoms, for example by intensifying sensations of pain, the more they learn that they are controllable. O'Connell's

(1983) comments on the effect of symptom prescription are also consistent with this interpretation. The same seems to me to be true of one of the most famous studies in the behavioural literature, that of Yates' (1958) investigation of the effects of massed *versus* spaced trials of the voluntary evocation of facial tics. Yates was testing the hypothesis that the tics had been learned and were subject to the ordinary laws of learning. Therapeutic consequences followed in that there was some reduction in the frequencies of the tics. Without detracting from the implication of the confirmation of Yates' prediction, we may ask whether the therapeutic by-product was not due to his patient coming to feel that she was regaining responsibility for a part of her behaviour.

As Malan et al. (1975) have discovered, taking responsibility clearly applies in the psychoanalytic framework. By his claim that dynamic therapists must convey to patients the necessity for them to "learn to accept *personal responsibility*" for their actions Strupp (1975) has also drawn attention to it in the dynamic psychotherapies. Taking responsibility is, of course, a central if unstated ingredient in the treatment of addictions.

Before concluding, let us consider the most difficult aspect of this conceptualization: its clear emphasis upon subjectivity, not to say mentalism. Many years ago when Frank (1961) marshalled the evidence against the specificity of therapeutic techniques he pointed to the central role played by mobilising the patient's hope and instilling confidence and similar non-specific factors. About all that eventuated were some studies of the effects of expectation and motivation and few of these yielded clear results (Frank, 1974). For the most part Frank's thesis has not been properly tested. As Bandura (1977) has observed, expectations have been thought of globally, considered to act statically, assessed inadequately, and then only at the one point in time. We seem to be more at home with simple measures of simply defined concepts than with the analyses of important but complex processes.

We also like to think of ours as a scientific discipline and we are uncomfortable with mentalistic concepts. Yet they are the ones that patients use. For example, in the data from people, who have given up smoking or are intending to give it up, Salvina Failla, one of my Honours students, has found that almost all her subjects pose success or failure in terms of whether or not they "really want to", have enough "resolve", "will-power", "desire", or "motivation", and so on. Hangovers from a positivist past make it difficult for some of us to think about mentalistic notions like self, responsibility, hope, will, and desire and to incorporate them into theories is often considered virtually impossible. Yet, if we are to understand what goes on in therapy we have no choice but to follow these leads.

Does the road I have pointed to lead to mysticism? Not necessarily. Frank attributed the initial marked reduction in the severity of his control patients' symptoms to "hope". Is this mysticism? Let me answer by asking how mystical Wimberger's and Millar's (1968) adolescent patient was when he responded to being placed on the waiting list with:

> That's okay, Doctor. I guess a shipwrecked person starts to feel better the moment a rescue plane has spotted him. (p. 180).

If you do not like that answer you might like to attribute the change to the measurable reduction in anxiety that accompanies therapeutic intervention. If you do, you have only to assume that it takes place slightly earlier than has actually been demonstrated (Murray and Jacobson, 1978).

This is one place where we should have no hesitation in following Charcot. In a paper published in the year of his death Charcot (1893) made an analysis of the well attested faith-cure of Mlle. Coirin. A "tumour" of her left breast had led to the loss of her nipple and the subsequent development of a ulcerous cavity through which, over a period of some twelve years, there was "discharged incessantly a fluid with an offensive smell". When Mlle. Coirin applied some earth obtained from the neighbourhood of the tomb of St. Francis of Paris to the hole in her breast the discharge was immediately staunched and a healing process commenced that was complete within about two weeks. Charcot had no difficulty in explaining how oedema due to vasomotor constriction had first caused the cutaneous "gangrene" or how the removal of

the constriction had modified "the local conditions affecting the nutrition of the tissues" and allowed healing to take place. The healing was governed "by virtue of physiological laws as well known as those which had previously operated to produce the gangrene". Given our experimentally based knowledge of the cognitive control possible over internal physiological processes like blood flow we should not be surprised by this case and others like it. If extraordinary cases can be explained without appeal to mystical or supernatural processes we need not be fainthearted about tackling the "mental" effects of placebos or psychological treatment.

My thesis has been that because we misconstrue what we take to be the specific effects of our therapies we do not understand their incidental or so-called non-specific effects. If there are idiosyncratic theoretical conceptualizations, specific causes, and specific remedies we shall not discover them until we put aside Freud's causal blinkers. It is they which have prevented us from seeing either the need to use placebo controls or the necessity of placing their direct study at the centre of our endeavours.

Let me now conclude by returning to my starting point: Charcot and the Salpêtrière. Charcot had supposed, as we have seen, that symptoms were caused by an unconscious mental process that had escaped the controlling influence of the ego. We have also seen that Freud's original theses were simple extensions of those of the French school of psychopathology. When Freud left Paris on 28 February, 1886 his *souvenirs* therefore included rather more this photograph (see Figure 1) given him by Charcot. Perhaps we might include the similar Napoleonic and imperious attitude that Freud took up when he incompletely adapted the causal assumptions in Pasteur's and Koch's germ theory — after all, he did once describe himself as a Conquistador. What is definite is that it was a similar lack of caution to that which forced Napoleon back from Moscow that caused Charcot's rout over uniformities in hysteria and Freud's over its specific cause and remedy.

If we are to learn from history and avoid the farce of a third repetition we must be doubly careful about conducting research along the lines proposed. Symptom removal must not be tied to too definite a conceptual framework until we learn to see things from the perspective of the patient. The framework through which we investigate how our patients' varied attempts at their solutions have developed must be generated from their perspectives.

Otherwise we too will be as soundly defeated as were our predecessors at their Waterloos.

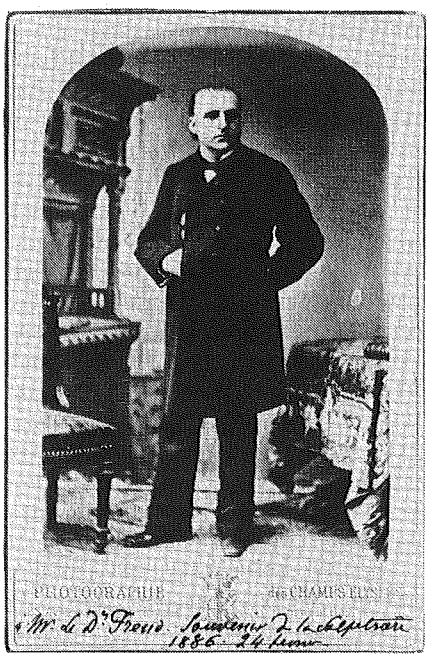

Figure 1. Freud's "souvenir" of Charcot. Reproduced by permission of Mary Evans/Sigmund Freud Copyrights Ltd.

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