THE USE OF OPERANT RESPONDING AS A MEASURE OF MOOD FLUCTUATION IN PERIODIC PSYCHOSIS[1]

Ullin T. Place

AUTHOR'S NOTE: This paper was written some thirty years ago at a time when I was in the process of transferring from a post in Clinical Psychology in the Department of Psychiatry at the University of Leeds to one in the Department of Philosophy in the same university. Apart from one abortive attempt to have it published at the time, it has been gathering dust ever since. I was encouraged to resurrect it by two events. Firstly the interest that has recently been shown in this work by Michael Apter and his followers because of its anticipation of some of the ideas which lie at the foundation of the so-called 'Reversal Theory' which he has developed in recent years (Apter 1982, etc.; Place and Wheeler 1999). The second event was a presentation, by Craig H. Kennedy (1999) of Vanderbilt University at the Annual Conference of the Experimental Analysis of Behaviour Group in London in March 1999 of a paper entitled 'Early to bed, early to rise ... Sleep deprivation causes response patterns to reorganize'. In this paper Kennedy reports, quite independently as one of the motivational effects of sleep deprivation, the phenomenon which is central to my paper, the independent variation in sensitivity to positive and negative reinforcement.

Abstract

Ryle (1949) has suggested that to be in a happy mood or frame of mind is to have (a) an increased capacity for enjoyment and (b) a reduced sensitivity to distress. It is a natural corollary of this view that to be in an unhappy or miserable mood or frame of mind is to have (a) a reduced capacity for enjoyment and (b) an enhanced sensitivity to distress. Assuming that an individual's capacity for enjoyment can be measured by the rate of operant responding under conditions of positive reinforcement and his or her sensitivity to distress by the rate of responding under conditions of negative reinforcement, it should follow, on Ryle's theory, that in elation the rate of response under conditions of positive reinforcement will be high with a correspondingly low rate of response when reinforcement is negative. In depression, on the other hand, a low rate of response is predicted for the positive reinforcement condition with a high rate of response for the negative reinforcement condition. In this study, the rate of operant responding under

[1] An earlier version of this paper was presented to the Annual Conference of the Experimental Analysis of Behaviour Group, Nottingham, 1968.

conditions of positive reinforcement is compared with that under conditions of negative reinforcement in two manic depressive patients with regular and predictable mood cycles. Longitudinal studies extended over several months confirm a number of the predictions drawn from Ryle's theory and throw some new and unexpected light on the nature of pathological mood states.

Introduction

The experiments described in this paper were designed to provide an empirical test of two theories of the nature of elation and depression each of which implies a different set of predictions as to the effect of fluctuations of mood between these two poles on the emission of an operant response. The first of these theories is one stated by Skinner in his book *Science and Human Behavior* (1953). The second is a theory put forward by Ryle in his book *The Concept of Mind* (1949). The passages from which I have extracted these theories are in both cases somewhat cryptic and obscure, and I cannot be certain that the respective authors would subscribe to them in the form in which I have stated them.

Skinner's Theory of Elation and Depression

Skinner has this to say:

> Instead of responses commonly observed in grief, sorrow or fear, we may observe responses characteristic of elation or joy. Emotional pre-dispositions ... stand in ... polar opposition, the general depression of activity in the one case is matched by a general heightening of activity in the other (Skinner 1952, p.180).

I infer from this that in Skinner's view, elation consists in a general increase in the rate and probability of operant response emission and that depression consists in a corresponding general reduction in the rate and probability of operant response emission.

Ryle's Theory of Elation and Depression

Ryle's theory is stated as follows:

> If a person is too gay to brood over a rebuff, he is not undergoing so violent a feeling that he can think of nothing else; and therefore not of the rebuff; on the contrary, he enjoys much more than usual all the things he does and all the thoughts he thinks, including thoughts of the rebuff. He does not mind thinking of it as much as he would usually do. (Ryle 1949, p. 100).

As I interpret it, what Ryle is saying here is that a mood of elation or happiness consists of two things,

(a) an increase in the individual's capacity for enjoyment, and

(b) an overall reduction in the individual's sensitivity to distress.

Ryle makes no specific mention of the depressed or unhappy frame of mind in this connection; but since depression is presumably the opposite of elation, it would seem to be a corollary of his view that depression consists in

(c) a reduction in the individual's capacity for enjoyment, and

(d) an increase in the individual's sensitivity to distress.

The behavioural implications of a theory stated in terms of concepts like "enjoyment" and "distress" are not perhaps immediately apparent; but if we refer to what Ryle has to say about enjoyment later in the same chapter, we find that "a person who is so absorbed in some activity, such as golf or argument, that he is reluctant to stop or even think of anything else, is ‘taking pleasure in’ or ‘enjoying’ doing what he is doing, though he is in no degree convulsed or beside himself, and though he is not, therefore, experiencing any particular feelings." (Ryle 1949, pp. 107-8). It is evident from this quotation that Ryle is offering an account of ‘pleasure’ and ‘enjoyment’, as those words are used in ordinary language, which parallels almost exactly the account which Thorndike gave of ‘a satisfying state of affairs’ in connection with his original formulation of the Law of Effect as a principle of learning, where he defines it as something "which the animal does nothing to avoid, often doing things which maintain or renew it" (Thorndike 1911, p. 245). Now a satisfying state of affairs defined in this way is what Skinner would call (following Pavlov[2]) ‘a reinforcing state of affairs’ or ‘a reinforcing stimulus’, where ‘a reinforcing stimulus’ "is defined as such by its power to produce the resulting change" (Skinner 1938, p.62). More specifically a satisfying state of affairs, as defined by Thorndike, and hence one that is

[2] While it is true that the *term* ‘reinforcement’ comes from Pavlov, the term is applied rather differently in connection with instrumental/operant responding from the way Pavlov used it in connection with his so-called ‘classical’/’respondent’ conditioning. The difference between the two usages can be expressed by saying that in Pavlovian conditioning a reinforcer is an event which ‘confirms an expectation’ established by previous occurrences of the CS followed by the US, whereas in instrumental operant/operant responding it is an event which ‘encourages’ the organism to repeat what it has just done either on the same occasion or on some future occasion when similar circumstances arise. [Footnote added 1993]

enjoyed or which gives pleasure, as defined by Ryle, is what Skinner would call ‘a positive reinforcer’, a state of affairs whose appearance in the stimulus environment of an organism serves to maintain and strengthen the organism's disposition to repeat whatever behaviour immediately preceded its appearance, as contrasted with ‘a negative reinforcer’ or ‘aversive stimulus’ whose removal from the stimulus environment of an organism serves to maintain and strengthen the organism's disposition to repeat whatever behaviour immediately preceded its removal.

Following these conceptual connexions we may, I suggest, translate the notion of an individual's capacity for enjoyment as construed by Ryle into Skinner's terminology as `the sensitivity of the individual organism to positive operant reinforcement', meaning by this the degree to which the individual's behavioural dispositions (response probabilities) are susceptible to being maintained and strengthened by *additions to* the content of the individual's stimulus environment. We may likewise, I suggest, translate the notion of sensitivity to distress into Skinner's terminology as `the sensitivity of the individual organism to negative operant reinforcement', meaning by this the degree to which the individual's behavioural dispositions are susceptible to being maintained and strengthened by *subtractions from* the content of the individual's stimulus environment.

Using these translation equations we can restate Ryle's theory in Skinner's terminology as follows:

A happy state of mind, on Ryle's view, is a condition in which

(a) the individual's sensitivity to positive reinforcement is enhanced, and

(b) his sensitivity to negative reinforcement is diminished.

An unhappy or depressed state of mind is a condition in which

(c) the individual's sensitivity to positive reinforcement is diminished, and

(d) his sensitivity to negative reinforcement is enhanced.

The Different Predictions of Skinner's and Ryle's Theories

The different predictions of Skinner's and Ryle's theories in the hypothetical case of an individual whose mood is fluctuating between happiness or elation on the one hand and unhappiness and depression on the other, with periods of normality in between, is illustrated in Fig. 1.

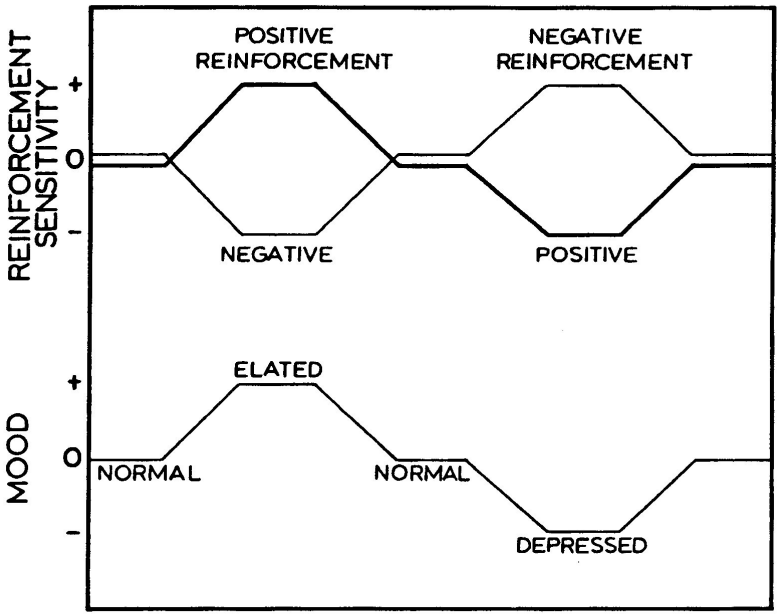


FIG.1. Schematic representation of Ryle's theory .

In such a case as this, in which an individual's mood fluctuates between elation and depression with periods of normality in between, Skinner's theory would predict that the individual's rate of operant responding on any schedule of reinforcement, other than one in which there is a differential reinforcement of low rate (drl), would go up and down in direct relationship to his mood at the time, regardless of whether the reinforcement was positive or negative. In the elated phase, the response rate would be higher than normal, in the depressed phase it would be lower.

The predictions of Ryle's theory are more complex. In the first place, since it is the individual's sensitivity to reinforcement that is assumed to change rather than his potential response output as such, the changes would be reflected, on any schedule reinforcement, in an increase or decrease in the amount of reinforcement secured by the behaviour. On most schedules of reinforcement this would involve a

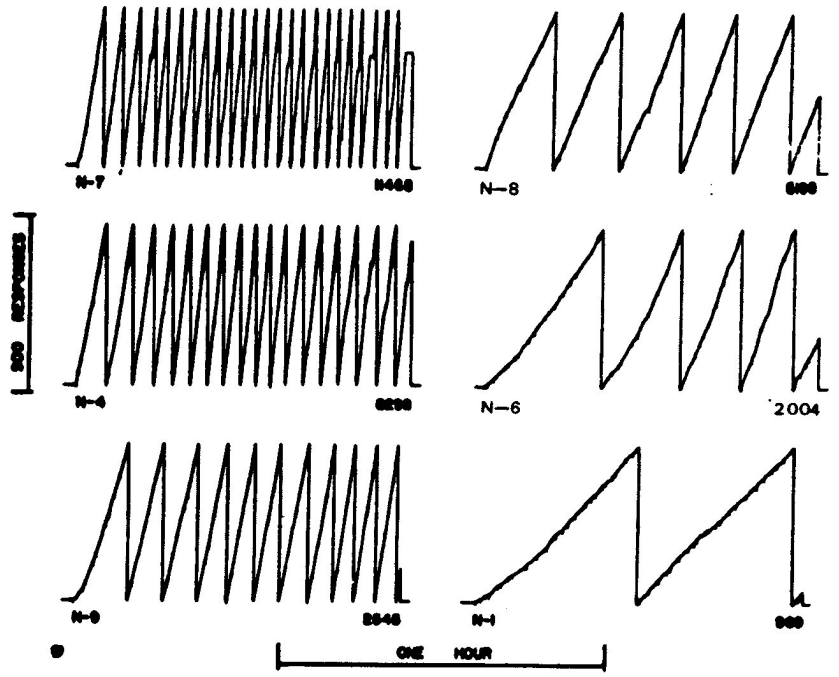
rise in response output with increased sensitivity to reinforcement, but with a drl in operation it would be reflected in an increased efficiency of the performance, possibly with an overall reduction in response rate due to the more efficient elimination of responses which have had the effect of delaying reinforcement. On other schedules, where the increased sensitivity to reinforcement would be reflected in a rise in response output, Ryle's theory would predict that elation and depression would have an opposite effect on the response rate according to whether the reinforcement was positive or negative. The rate of responding under conditions of positive reinforcement would be expected to follow the same course as predicted by Skinner's theory, rising above normal in elation and falling below normal in depression. The rate of responding in conditions of negative reinforcement, on the other hand, would be expected to do the opposite, to fall below normal in elation and rise above normal in depression (Fig. 1).

Previous studies

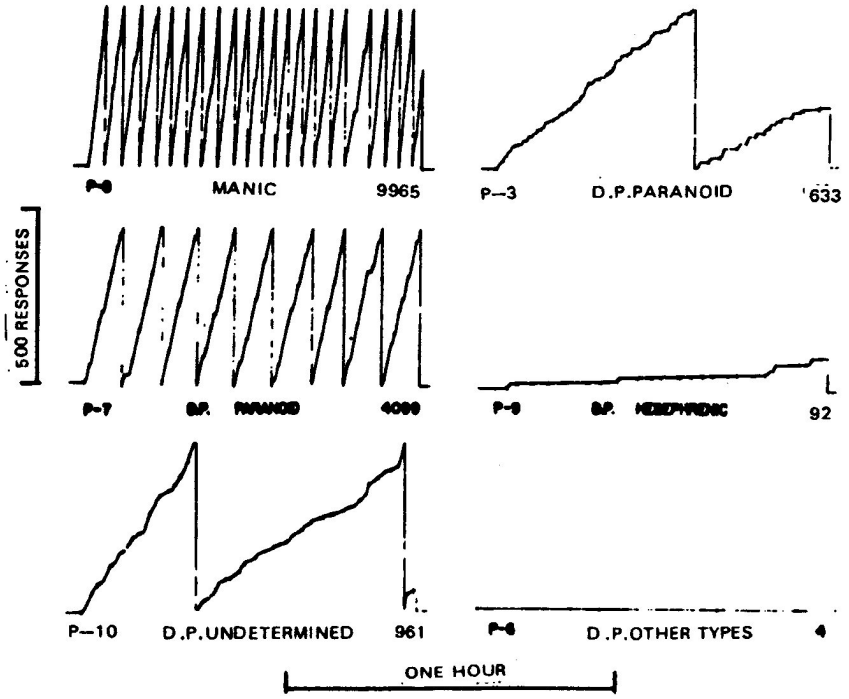
Is there any evidence about the rate of operant responding when an individual's mood is elated or depressed which would enable us to discriminate between these two theories? The only relevant published evidence known to the writer comes from Ogden Lindsley's pioneer study of "the characteristics of the behaviour of chronic psychotics revealed by the operant conditioning methods" (Lindsley 1960).

Lindsley's results, in so far as they affect our present problem, are given in Fig. 2. This shows a marked difference between the high operant response rate emitted by a manic (elated) patient and the low rate emitted by a number of depressed patients. This at first sight would seem to be in agreement with Skinner's theory, as indeed it is. However, if we examine the text we find that the reinforcements used in these experiments were uniformly positive - cards, cigarettes, coins, etc. - and that therefore the results conform equally to the predictions of Ryle's theory.

There is, however, another point that is brought out by Lindsley's observations, namely the large individual differences in overall response rate between individuals who fall into the same classification as far as mood is concerned. One of the consequences of these individual differences is that it would be



Cumulative response records for six unhospitalized normal adults, responding on a one-minute variable-interval schedule of intermittent reinforcement with five-cent coins. Each record is of the fifth hour of responding on this schedule. Under each record is printed the number of the subject on the left, and on the right the number of responses made during the hour.



Cumulative response records for six hospitalized adult chronic psychotics, responding on a one-minute variable-interval schedule of intermittent reinforcement for small assorted pieces of candy. Each record is of the fifth hour of responding on this schedule. Under each record is printed the number of each patient to the left, the most recent psychiatric diagnosis, and to the right the number of responses made during the hour.

FIG. 2. Data from Lindsley (1960).

quite impossible to differentiate the performance of an individual manic subject from that of a normal individual or between the performance of some normals and manics on the one hand and some of the less deeply depressed patients on the other. If anything, there is a suggestion that the response rate of the manic patient is lower rather than higher than the mean response rate of the normal subjects.

Method

In the light of the results obtained by Lindsley, it seemed reasonable to argue that there are two sets of conditions which would need to be fulfilled in order to test the predictions of Ryle's theory as against Skinner's with respect to the effect of mood fluctuations on an ongoing operant response. In the first place there is a need for an independent measure of the operant response rate under conditions of positive and negative reinforcement respectively, on a schedule of reinforcement which, apart from being positive in one case and negative in the other, would be otherwise identical. Secondly, in view of the individual differences found by Lindsley, it seemed more appropriate to carry out longitudinal studies of individual cases showing consistent and repeated fluctuations of mood between elation and depression, so as to compare rates of responding within the same individual under different mood conditions, rather than rely on comparisons across individuals. These were the principles on which the design of the present series of experiments was based.

The first problem was to develop a schedule of operant reinforcement suitable for use with human subjects which would permit a direct comparison of the effects of positive and negative reinforcement on response rate. For this purpose it was not possible to use any of the standard reinforcement schedules which have been studied in detail in the laboratory. The reason for this is that all the standard reinforcement schedules used with a positive reinforcement, such as continuous reinforcement (crf), fixed and variable ratio (FR, VR), fixed and variable interval (FI, VI), presuppose a reinforcer which occurs as an isolated explosive event such as the delivery of a food pellet, a piece of candy, a cigarette or a coin. In negative reinforcement schedules such as Sidman avoidance, on the other hand, the reinforcing state of affairs is present continuously and persistently as long as the aversive stimulus remains absent under conditions where the organism has learnt to expect it. Negative

reinforcement can occur as an isolated explosive event only when the aversive stimulus is tied to a specific discriminative conditioned stimulus of short duration, as in the traditional trial by trial avoidance learning situation. This type of situation does not generate repeated response emission in the absence of the discriminative conditioned stimulus and is consequently unsuitable in the present application, where we are interested in fluctuations in the rate of spontaneous response emission. What was needed, therefore, was a schedule in which positive reinforcement can occur, as does negative reinforcement in a Sidman avoidance schedule, as a continuous uniform process extended over time like water running from a tap. As far as I am aware, the only published schedule of reinforcement that satisfies these conditions is that provided by Ogden Lindsley's 'conjugate servo' device (Lindsley *et al.* 1961; Lindsley 1962). This is a device which automatically relates the volume of a stimulus input to the responses of the subject. When used in the negative reinforcement condition, the volume of an aversive stimulus such as an unpleasant noise is progressively increased in the absence of a response on the part of the subject, while each response of the subject lowers the volume by a predetermined amount. Conversely in the positive reinforcement condition the volume of a positive reinforcer such as pleasant music is progressively reduced in the absence of the subject's response and is increased by a predetermined amount when a response occurs.

Apparatus

In the present study, as originally planned, it was intended to use a conjugate servo with auditory stimuli - music and white noise - as the positive reinforcer and aversive stimulus respectively. Music, however, was soon abandoned as the projected positive reinforcer due to the problems of individual differences in musical appreciation and satiation effects. After some experimentation with both noise and shock as aversive stimuli, the reinforcement system shown in Figure 3 was ultimately adopted.

Figure 3 shows the manipulandum used by the subject, a morse key, with a counter in the subject's line of regard above it. The counter is reversible; it is in fact a revolution counter taken from a tape recorder driven by a d.c. motor which can be reversed by reversing the polarity of the supply current. To the right and left of the counter are two lamps, one red, one green. When the green light is on, the

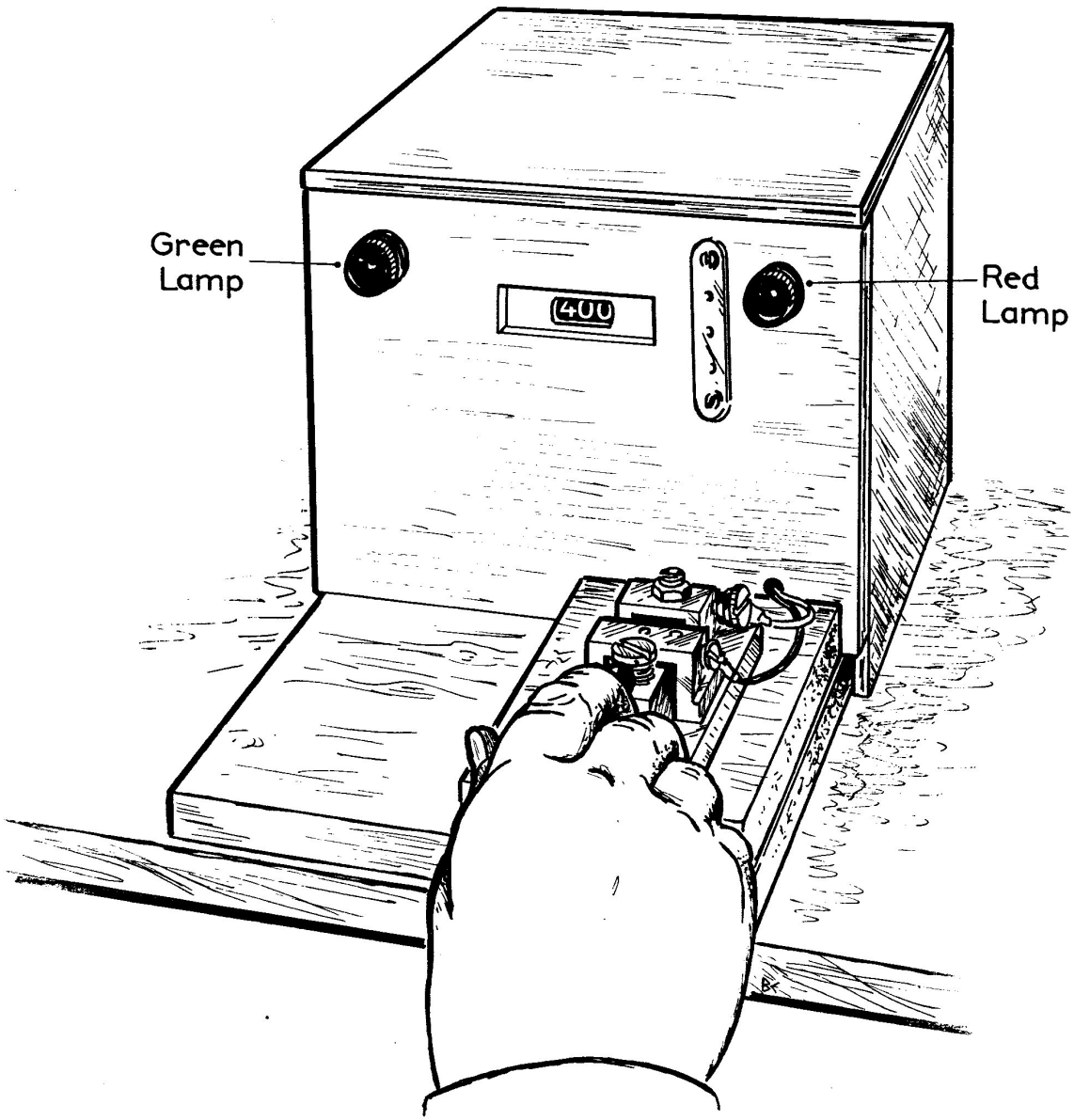


FIG. 3 Morse Key and Reinforcement Counter.

reinforcement is positive. In this condition pressing the key will cause the counter to start counting up. When the red light is on, the reinforcement is negative and the counter counts down unless and until the subject responds so as to stop it. The movement of the counter up and down derives its value as a positive reinforcer and aversive stimulus respectively from a cash payment that is made at the end of the experimental session, the amount of which is determined by the score showing on the counter. Each experimental session consists of two fifteen-minute subsessions, one with positive reinforcement, the other with negative reinforcement.

The two subsessions are separated by a three-minute rest pause or `time out' period when neither lamp is lit. The order of the two reinforcement subsessions is varied from session to session according a semi-random programme. In order to avoid the negative score which would accrue to the subject if he failed to respond during either subsession, he starts with a bonus of 400 points, slightly more than he would lose if he failed to respond altogether. The counter displayed to the subject counts up at a constant rate when positive reinforcement is being given and counts down at the same rate when reinforcement is withheld under conditions and counts at the same rate when reinforcement is withheld under conditions of negative reinforcement. This means that for a given amount of work done during the 15-minute subsession, the reward in terms of money gained in the case of positive reinforcement and saved in the case of negative reinforcement is the same.

The method used to programme the contingencies relating response to reinforcement is the least satisfactory aspect of the technique employed in the present experiment and would be substantially modified, if the experiments were to be repeated. A pair of uniselectors is used to add up the number of responses made within a period of two seconds. If the number of responses in the preceding two-second period equals or exceeds a predetermined figure, reinforcement is given during the following two-second period. If the number of responses falls below the required figure, no reinforcement is received. The rate of responding required for reinforcement varies between 1 and 5 responses per second during the experimental session. Each fifteen-minute subsession is divided into five three-minute periods during which a given minimum response rate is required for reinforcement. Each of the five rates is represented

once in each subsession. The change of rate is not announced to the subject and the order in which each rate occurs varies in a semi-random manner from sub-session to sub-session. As far as I have been able to discover these changes in rate requirement served no useful purpose and had the effect of obscuring what turned out to be important spontaneous changes in response rate during the session.

Subjects

The subjects in this study were patients suffering from a form of manic depressive psychosis which is characterised by very regular and predictable fluctuations of mood between the two poles of manic excitement and depression. Patients showing these very regular mood-fluctuations are comparatively rare, but fortunately for our purposes they are of special interest to physiologists and biochemists who are trying to discover a metabolic and biochemical basis for mental disorder, since the clockwork regularity of the mood-fluctuations in these cases virtually precludes an explanation of these conditions in terms of environmental contingencies. Research units interested in this problem collect patients of this kind and subject them to intensive study over considerable periods of time. This makes them ideally suited to the kind of longitudinal study of an individual case which, in view of the individual differences reported by Lindsley, would seem to be the method of choice in studying the effect of mood on operant response rate. I am reporting the results from two patients of this kind, one from the M.R.C. Unit for the Study of Chemical Pathology of Mental Disorders, based at that time at Hollymoor Hospital in Birmingham under Dr. F.A.Jenner, now Professor of Psychiatry at Sheffield University, and another, a patient from the Metabolic Research Unit at High Royds Hospital, Menston under Dr R.P. Hullin, Reader of Biochemistry at the University of Leeds.

Results: Case 1

Figure 4 shows the results obtained with Professor Jenner's patient at Hollymoor in 1967. This is a 56-year old male patient, previously described by Jenner *et al.* (1967), with a twelve-year history of a regular 48-hour manic depressive cycle since a road accident in 1955. The 48-hour cycle is shown clearly on the mood rating at the bottom of the graph. These mood ratings were made every four hours during the day by trained nursing staff. The chart in fact shows only one rating per day. On days when the

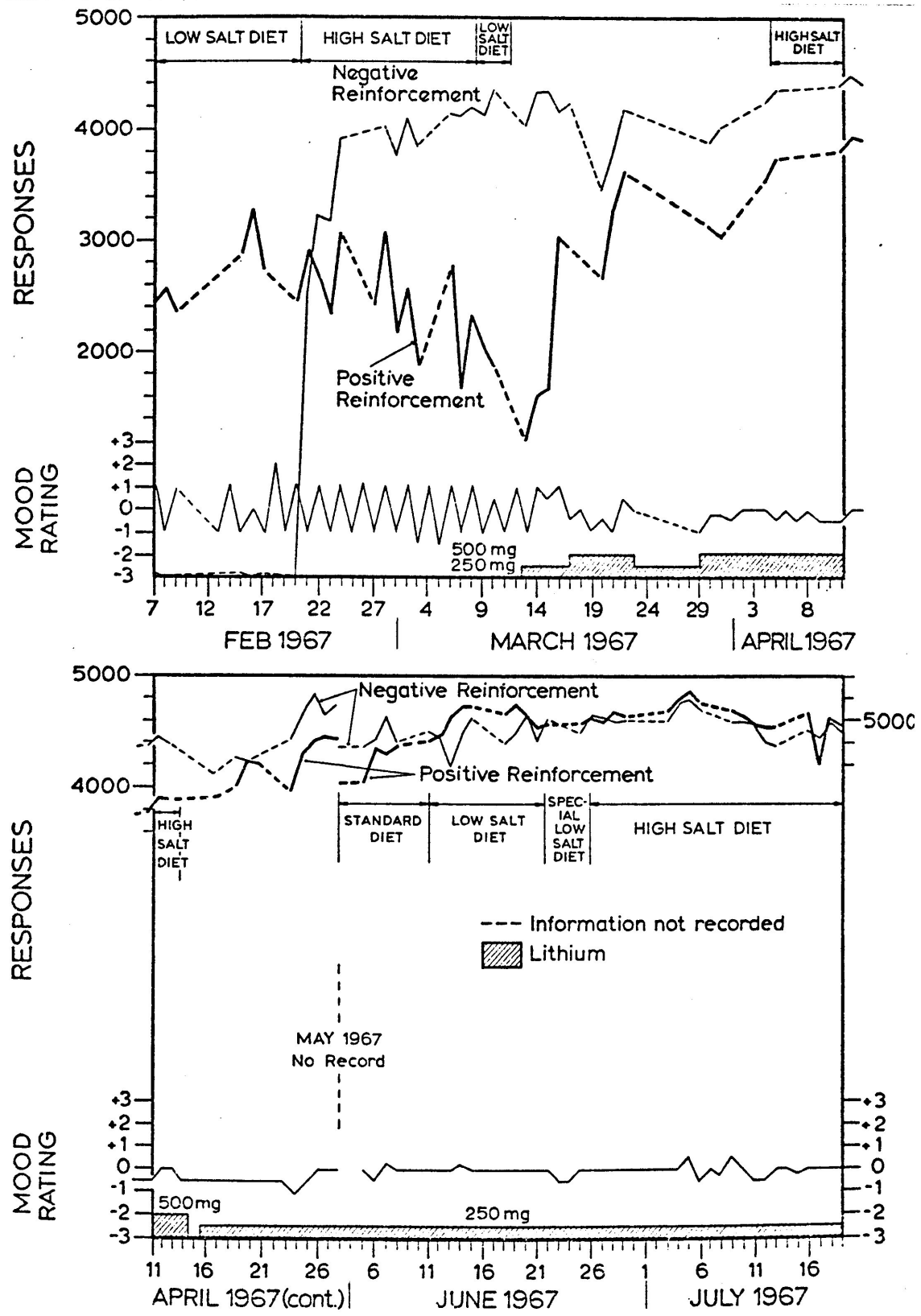


Fig. 4. Operant Response Rates for patient J.W.

patient was run on the operant responding device the rating is that closest to the time when the patient was run. On other days, it is the mean rating for the day as a whole. In Jenner's unit an individual rating scale from +3 to -3 is devised for each patient and the behavioural criteria for assigning each rating are specified in terms of behaviour found to be characteristic of the individual patient as the mood changes from one pole to the other.

On the thirty-fourth day after the operant response sessions had been introduced, this patient, who had previously been without drugs of any kind, was put on a dose of 250 mg. of lithium. This was increased three days later to 500 mg. and then reduced again to 250 mg. after the patient had developed side effects in the form of nausea and vomiting. As can be seen from Figure 4, the introduction of lithium had the effect of abolishing the mood cycle and returning the patient to an essentially normal condition.

During the period represented on the graph, the patient was given one 33-minute session in the operant response situation every weekday, except when he was allowed home on leave or when, as happened not infrequently, the apparatus was out of commission due to mechanical failure.

The sections of the graph connected by continuous thin lines represent periods when the patient was run on successive days, while those connected by thin dotted lines represent periods when there was a gap of more than 24 hours between runs. The height of the points connected by these lines show the total number of responses made per 15-minute sub-session under conditions of negative reinforcement. The points connected by continuous and dotted thick lines show the total number of responses made per 15-minute sub-session under positive reinforcement conditions.

It will be noted that during the first fortnight, although the patient was responding during positive reinforcement at a level similar to that which was maintained until the lithium was introduced, responses under conditions of negative reinforcement were at a very low level indeed. The rate of responding in this reinforcement condition was so low that no reinforcements were received at all and in consequence such responding as had been occurring initially tended to extinguish altogether. The reason for this was that during a previous pilot phase of the experiment, which is not shown on the graph,

the cash payments at the end of the session had been based on the amount accumulated on the counter in the positive reinforcement condition only.

An attempt was made to provide negative reinforcement by using a loud noise generated by a pair of motor car sirens as the aversive stimulus. Unfortunately, the subject, an ex-naval rating, regarding this aversive stimulus as a challenge to his powers of endurance, rather than as something which he could work to avoid. As a result the sirens, which were not designed to work continuously for periods as long as 15 minutes, very soon burned out. An electric shock was then substituted as the aversive stimulus with similar results. The patient quickly adapted to the strongest shock which could be generated by the inductorium employed without making any significant attempt to respond so as to remove it. As a result of this experience the habit of not responding appears to have become firmly conditioned to the red lamp which signalled the presence of the negative reinforcement condition. Consequently when, in view of the success of financial gain as a positive reinforcer, a corresponding financial loss was finally substituted as the aversive event, the rate of responding continued at a level well below that required to secure reinforcement on the schedule in operation. Eventually, after the patient had been told on more than one occasion that if he worked as hard when the red lamp was on as he did when the green lamp was on, he would double the amount he was paid at the end of the session, he finally had, what one can only call, `a flash of insight', with the result that his rate of responding on negative reinforcement rose rapidly to a level well above that on positive reinforcement. Once established the rate of responding under negative reinforcement continued on a slowly rising course and remained virtually unaffected by the introduction of lithium.

Apart from this initial dramatic rise, responding under conditions of negative reinforcement is also relatively unaffected by fluctuations of mood from day to day. There is in fact a slight fluctuation in the response rate during the period over which mood was fluctuating on the 48-hour cycle which correlates positively with the mood rating. For the period of three weeks immediately prior to the introduction of lithium this correlation is +0.17 which is not significant.

Apart from this positive correlation between the mood rating and the rate of response under conditions of negative reinforcement which, as we have seen, is not statistically significant, there is no evidence of the overall reduction in the rate of response emission in depression predicted on Skinner's theory. The rate of responding under conditions of positive reinforcement on the other hand rises and falls as the mood goes up and down, as predicted by both Ryle's and Skinner's theories, yielding a positive correlation of +0.476, which is significant at the 3 per cent level of confidence, between mood rating and positively reinforced responding for the period up to the introduction of lithium. But since the rate of responding under conditions of negative reinforcement is at a very much higher level and is relatively unaffected both by mood changes in the pre-lithium phase and by the return to a normal condition after the introduction of lithium, it is evident that the fluctuations in the rate of positively reinforced responding are not due to any general change in reactivity. They can only be accounted for, in accordance with Ryle's theory, as the result of specific changes in the patient's sensitivity to positive reinforcement.

I conclude therefore that at least one of the propositions of Ryle's theory is confirmed by these results, namely that there is a reduction in sensitivity to positive reinforcement in depression. What is not confirmed is

(a) an increased sensitivity to negative reinforcement in depression,

(b) a reduced sensitivity to negative reinforcement in elation, and

(c) a higher than normal level of sensitivity to positive reinforcement in elation.

The fact that the rate of responding under conditions of positive reinforcement on days when the patient's mood is elated is significantly lower than the rate of response under this condition when the patient's mood returns to normal is not readily accounted for on either theory. And this leaves us with the problem of how to account for the hyperactivity which is characteristic of this, as of other patients of this type, during the manic or elated phase.

A possible solution, however, is suggested in terms of Ryle's theory when we consider that although we would certainly describe a patient in the manic phase as being in a happier frame of mind that he is when depressed, we should not be inclined to describe his mood as happier than it is when it returns to normal. As many psychiatrists have observed, the feverish hyperactivity of the manic phase suggests an underlying unhappiness. This could be accounted for on Ryle's theory by supposing that the sensitivity to positive reinforcement in mania although higher than in depression is subject to an abnormally rapid adaptation or satiation effect. An individual in whom positive reinforcement tended to satiate with abnormal rapidity would, in an environment in which there were large numbers of different sources of positive reinforcement available, tend to flit in a butterfly fashion from one source of reinforcement to another, which is precisely the kind of behaviour that is referred to as hyperactivity in the manic patient. It was certainly very characteristic of the patient we are considering when in the manic phase. On the other hand, in a situation, such as the one we are studying here, in which the individual is required to respond continuously for 15 minutes for a single source of reinforcement, it would be predicted that his response rate, though perhaps high initially, would fall off rapidly as the session proceeded, yielding a lower response total than when his mood returns to normal and the effect of the reinforcer satiates more slowly.

This theory would account for the fact that in this case, as in Lindsley's (1960) study, the positively reinforced response rate in the manic phase when the mood is fluctuating is lower than it is when the mood returns to normal. But it also predicts that the response rate would tend to fall or fall more rapidly as the session proceeds when the patient is manic than it does when the mood is normal or depressed. Unfortunately no evidence of such a trend was found in this case when responses for successive three-minute periods on the elated days were compared with those on the depressed days.

Results: Case 2.

Figure 5 shows the results obtained from a female patient aged 52, studied in the Metabolic Research Unit, High Royds Hospital, Menston, in 1968. This patient has suffered from periodic bouts of elation and depression since she was 17. As can be seen from the mood rating graph at the bottom of Figure 5,

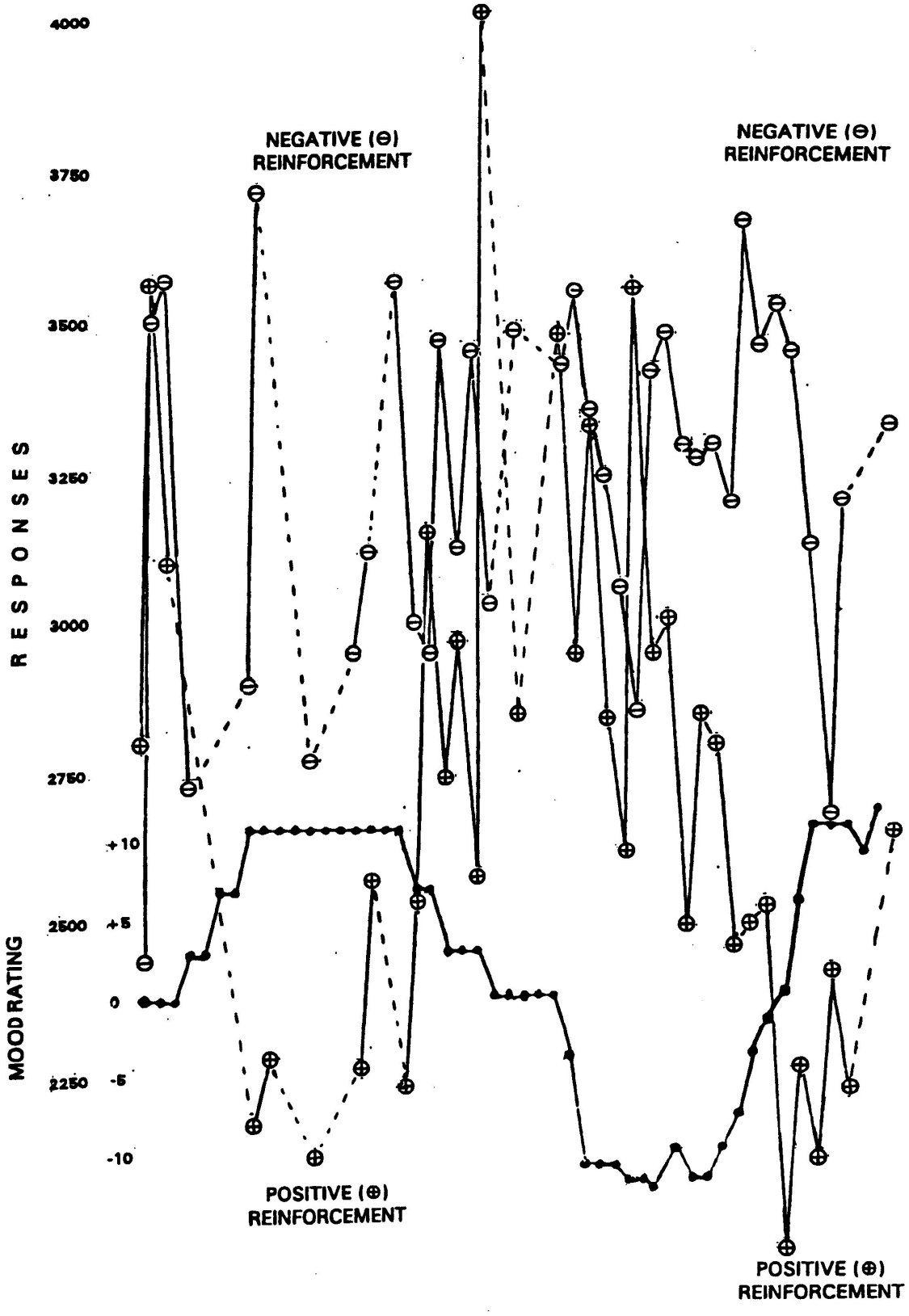


Fig 5. Operant Response Rates for patient M.W.

she has a much longer elation-depression cycle than the previous patient - about 52 days from peak to peak, as compared with 48 hours in the previous case. It will be noted that the mood rating used in the Unit at High Royds Hospital differs from that used in Professor Jenner's unit; instead of an individual rating scale for each patient, there is a common rating scale for all patients obtained by adding together the individual ratings on 7 - 7 point subscales giving a range of total scores between -21 and +21.

During the period represented by the graph, the patient was on a dose of 500 mg. of lithium. This has had the effect of reducing the severity of both the manic and the depressed phases of the cycle and of cutting down the length of the cycle by about 50 per cent; it has not however abolished the cycle as it did in the case of the previous patient. Although the scale along both axes is different from that on Figure 4 it will be seen that the pattern is very different. The variation in response rate from day to day is much greater and the current mood rating appears to account for a much smaller proportion of the variance. But, whereas in the previous case the rate of responding for positive reinforcement was almost invariably lower than that for negative reinforcement, even when the gap between the two had narrowed markedly after the introduction of lithium, in this case it is the rate of responding to negative reinforcement that is the lower of the two.

The principle factor contributing to this negative relationship between mood and overall response rate is the rate of response under conditions of negative reinforcement which, in agreement with Ryle's theory and in direct contradiction to Skinner's, falls in elation and rises in depression. There is in fact a negative correlation of -0.419 between responses to negative reinforcement and the mood rating on the same day which is significant at the 1 per cent level.

Moreover, if we correlate responses to negative reinforcement with the mood rating on the days subsequent to that on which the run in question was made, we find that the correlation gradually rises to a maximum of -0.572 which is significant at the 0.1 per cent level on the second day after the one on which the run was made, suggesting that changes in negative reinforcement sensitivity occur a day or two before the corresponding changes of behaviour are observed clinically.

To this extent, the results are in agreement with the predictions of the theory. However, when we examine the correlation between responses to positive reinforcement and mood rating which ought to be positive both on Skinner's theory and on Ryle's, we find that there is in fact a small negative correlation of -0.22 between response positive reinforcement and mood rating. Although the correlation is small and did not quite reach statistical significance, the fact that it is in the wrong direction is a matter of some concern. However, if we examine what happens to the response rate within the 15-minute sub-session, we find that the situation is not as inconsistent with the prediction as it appears at first sight. If we correlate the number of responses made in the first three minutes of the positive reinforcement sub-session with the mood rating, we find that in spite of variations due to differences in rate requirement for the period from session to session, there is a positive correlation with the mood rating of +0.165 which just fails to reach significance at the 5 per cent level. By contrast in the case of negative reinforcement, we find that the correlation between the number of responses during the first 3-minute period, though not, as we might expect, as high as in the case of the responses for the 15-minute sub-session as a whole, is still negative at -0.206. What this seems to mean is that although the positive reinforcement sensitivity does, as predicted, rise with elation and drop with depression, in elation the effect of the positive reinforcement satiates very rapidly with the result that by the end of 15 minutes the rate of response has dropped well below the level on the depressed days yielding the overall slight negative correlation observed for the whole 15-minute period. We thus have a strong confirmation in the case of the second patient of the hypothesis of the rapid satiation of positive reinforcement in elation which was introduced to account for the lower than normal positively reinforced responding in the case of the first patient.

Conclusion

Although any conclusions based on longitudinal studies of only two cases, particularly when they show such marked individual differences, must necessarily be tentative, we are justified in concluding, at the very least, that the theory I have attributed to Skinner of a simple rise and fall in the rate of operant response emission in elation and depression will not account for the data in these two studies. On the

other hand the theory which I have attributed to Ryle, of a differential rise and fall in reinforcement sensitivity in the case of positive and negative reinforcement respectively, while not confirmed in all its predictions, has at least opened up what promises to be an interesting line of empirical investigation in this important and difficult area of clinical research.

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