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EFFECTS OF AROUSAL IN HUMAN INSTRUMENTAL LEARNING: A TEST OF BERLYNE'S AROUSAL THEORY BY MEANS OF INFERENTIAL SIMULATION [1]

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One of the purposes of the investigations reported here was to test Berlyne's 1960 theory of arousal, i.e., in relation to learning. Among the fundamental hypotheses of this theory there is one implying that reinforcement depends on arousal regulation. This theory was translated into three mathematical models incorporating different assumptions about the relationship between arousal regulation and the effects of learning. Furthermore, on the basis of the assumption that reinforcement is not motivationally regulated but should be treated descriptively, two models were developed, matching all of the assumptions of two of the three motivational models except that of motivational regulation of reinforcement. Because of the complexity of the models, they were investigated by means of inferential computer simulation. Parameters were estimated by Monte Carlo methods. The performance of the models was compared with the performance of a group of 50 human subjects in an instrumental learning task with a 100:0 CRF schedule. The performance of the motivational models proved to deviate at a high level of significance from the performance of the subjects (Tab. 2 & 3, Fig. 4). Moreover, the distribution of the performance of the motivational models is bimodal. This can be explained by the fact that most of the replications strive toward the opposite asymptote, closely approximating a criterion of several consecutive incorrect responses. Further explorations showed that when negative reinforcement is disconnected from arousal regulation, performance is ameliorated (Fig. 5). This improvement is, however, rather small, so it must be concluded that the motivational models, and consequently the theory from which they are derived, are not useful for generating predictions about learning performance. The non-motivational models, on the other hand, show fairly good agreement with the performance of the subjects. Even a model with variable "learning operators" shows a reasonably good fit for some perseveration statistics as well as the general learning curve statistics (Tab. 2, 3 and 4). It is concluded that models with variable learning operators deserve further exploration, and that Berlyne's arousal theory, as far as it is concerned with learning, seems to be defective in its most fundamental assumption.

Current learning theories can be classified as motivational or non-motivational according to whether or not motivational factors are deemed to influence learning. Motivational learning theories explain learning processes by identifying reinforcement of responses with some

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kind of aversive drive-reduction (e.g. Hull 1943; Berlyne 1960; Seward 1970). Non-motivational theories, on the contrary, avoid the attribution of any motivational forces to reinforcement and try to predict learning performance on the basis of contiguity of stimuli and responses (e.g. Estes 1950; Guthrie 1959) or some form of cognitive evaluation (e.g. Minsky & Papert 1969; Selfridge 1959).

Since the present investigation concerned this general question of the relative importance of motivational factors in learning, Berlyne's (1960, 1963) arousal theory was taken as a starting point. This theory is closely related to Hull's (1943) earlier attempts to design a coherent behaviour theory. Moreover, it tries to deal with some of the deficiencies imputed to this older drive-reduction theory: besides behaviour motivated by primary drives, it covers such areas as exploratory, creative, and ludic behaviour. Although Berlyne's arousal theory is not strongly dependent on the view that all reinforcement is accompanied by the reduction of an aversive motivational state – room being left for other possibilities – he discusses only motivational reinforcers. Consequently, empirical evaluation of this theory should yield evidence supporting, or at least reinforcing, one of the alternatives in the above-mentioned controversy.

Because of the complexity of the problem, computer simulation seemed to offer the best method for this investigation, especially since it has been shown that inferential simulation, a recently developed method of computer simulation, is not only permissible but even useful in hypothesis testing (Vandierendonck 1971, 1975). When this method is applied, the theory to be tested must be converted into a program or model showing a clear correspondence with the theory itself. Once a representative model is obtained, comparisons of the outputs of the model (computer program) and of the subjects provide evidence about the validity of the theory. To elucidate the consecutive steps clearly, we shall start with a short description of Berlyne's theory and show how this theory can be converted into a representative model, before dealing with the design of the experiment, the analysis of the data, and a discussion of the results.

CONSTRUCTION OF REPRESENTATIVE SIMULATORS

BRIEF STATEMENT OF BERLYNE'S AROUSAL THEORY [2]

Arousal which forms the central concept in Berlyne's theory, refers to the common core of all primary drives: the energizing of behaviour.

[2] It should be kept in mind that Berlyne's arousal theory has been fundamentally modified since 1967, so that the term arousal-reduction theory is no longer adequate. Consequently, the model developed here does not represent the more recent theory. The reported investigation concerns Berlyne's original version of the arousal theory. We see no a priori reason not to test this "older" theory. After all, acceptance or rejection of theories is not a matter of individual decisions, but should be based on empirical evidence (cf. de Groot 1969).

Arousal thus concerns the degree of undirected activity, of readiness to react. In essence, the degree of arousal refers to a dimension, the lower pole of which is represented by sleep and comatose states, the upper pole by excitation and emotion.

Berlyne defines arousal as an intervening variable by linking it to antecedent conditions and to consequent behaviour. Therefore, he supposes that arousal is influenced by intensive, affective, and collative variables. Intensive variables include all stimulus conditions bearing on the intensity or amount of stimulation. Bright light, loud stimuli, high-pitched sounds, large figures, and chromatic colours would arouse more strongly than their opposites. Collative variables are related to resemblances and differences between stimulus elements, responses, attitudes, and thoughts. A number of investigations have shown that such variables as novelty, complexity, uncertainty, ambiguity, and surprisingness affect arousal (Berlyne 1958, 1965, 1968, 1969, 1970; Nicki 1970).

Consequences of arousal appear in the orientation reaction, exploratory behaviour, and learning. It is assumed that the subject strives for a low level of arousal, and that arousal reductions have rewarding influence, i.e., responses accompanied by or closely followed by arousal reduction will have a greater probability of appearing in similar situations in the future (learning by reinforcement).

The diagram in Fig. 1 represents the part of the theory relevant for the test we wished to undertake. The set of affective variables is not included in this diagram, because this set is so poorly defined that it is virtually impossible to distinguish it from other sets of external variables affecting arousal. In our opinion, this set of variables and the sets of intensive and collative variables overlap to a high degree. Therefore, instead of trying to make a far-fetched interpretation of the concept affective variable, we attempt to work without it. This must be possible, because the list of variables affecting arousal is not exhaustive and therefore in principle the theory should function whenever at least one of these variables is present.

Besides those already mentioned, the theory includes some other important concepts: conflict, arousal potential, and inhibition. The term conflict is used to refer to a competition of habits associated with the same set of stimuli. The degree of conflict increases with: a. the number of simultaneously instigated responses; b. the total absolute strength of the instigated response tendencies; c. the nearness to equality in strength of the competing response tendencies; and d. the degree of incompatibility of the competing responses.

According to Berlyne (1960, 1968), all collative variables induce a number of competing responses, so that the degree of conflict depends at least partially on the collative variables. Moreover, he assumes that collative variables exert an influence on arousal only through mediation by conflict.

The set of all antecedent variables capable of affecting arousal are called the arousal potential. It is assumed that the effects of all sources of arousal potential are additive (Berlyne 1960, p. 209). Arousal potential is thus nothing else than the union of all sources of arousal induction, i.e., intensive and collative variables and conflict.

Arousal potential affects arousal. Since stimulation is characterized solely by arousal-increasing facilities, arousal will always increase (at least never decrease) through arousal potential. Since arousal cannot

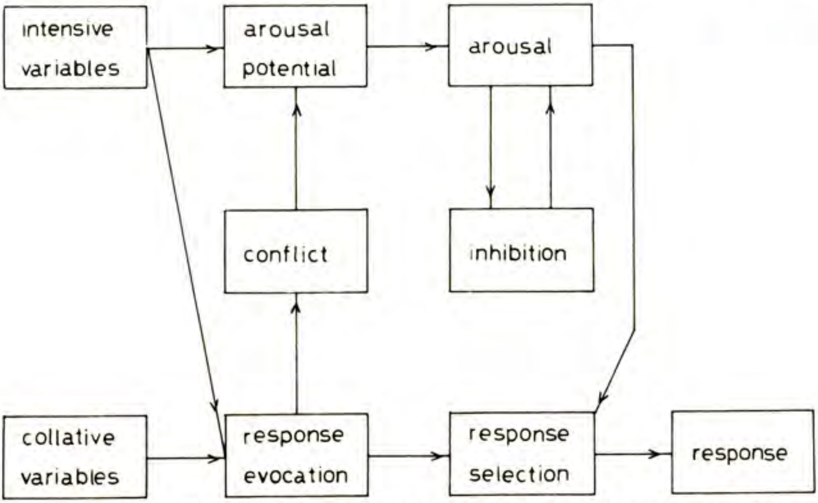

FIG. 1. SCHEMATIC REPRESENTATION OF PART OF BERLYNE'S (1960) THEORY OF AROUSAL REDUCTION

diminish spontaneously, some kind of inhibition is necessary. Berlyne supposes that the cortex is the inhibitory agent. This cortical inhibition, then, is a force counteracting arousal potential and thus restoring the original level of arousal. How the quantity of inhibition is related to other variables, is not explicitly stated in Berlyne's writings. It is, however, plausible to assume that only arousal and arousal potential affect inhibition. This would give rise to a feedback loop, which is consistent with Berlyne's views on this point (Berlyne 1960, p. 181).

Finally, arousal affects response selection, not only directly through the selection of certain kinds of behaviour but also indirectly through the reinforcement of responses such that certain response-classes become dominant over others.

QUANTIFICATION OF THE AROUSAL THEORY

We shall try now to quantify Berlyne's (1960) arousal theory in a way approximating the original formulation as much as possible, and starting with the definition of a function that relates arousal potential to arousal.

Berlyne (1963) hypothesizes a curvilinear relationship between arousal potential and arousal. The illustrations he gives (1963, pp. 318-320) suggest that a parabola should give a close approximation of the postulated relationship[3]. Let x be arousal potential and y arousal; then, by taking the directrix parallel to the abscissa, we define

$$y = \left[(a-x)^2 + 2bd\right]/2d, \tag{1}$$

where a and b are coordinates of the lowest point of the parabola and d is the distance between focus and directrix (for details of the

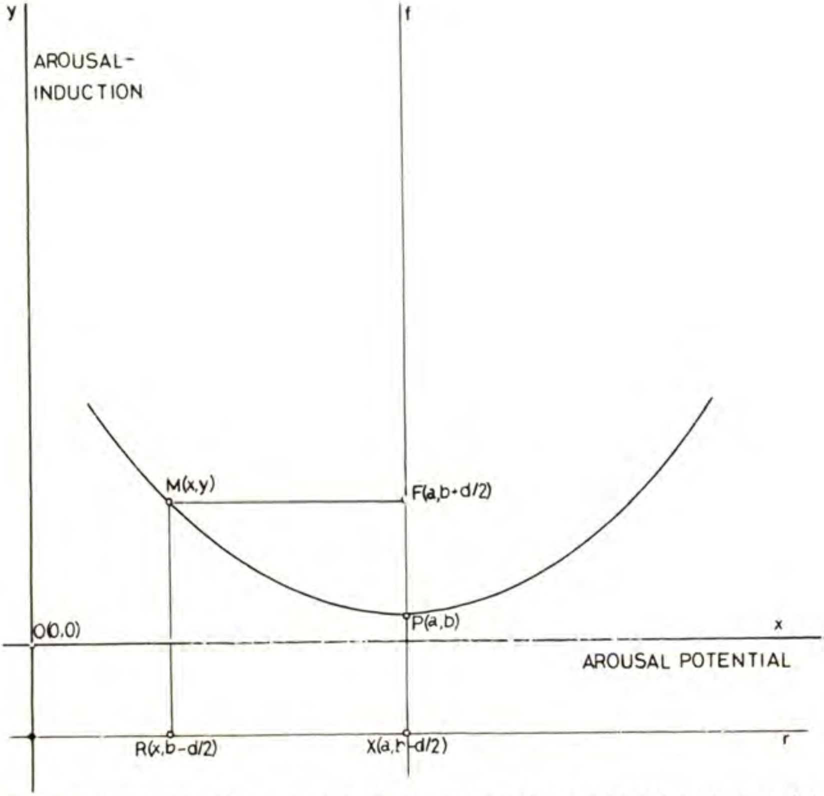

FIG. 2. RELATIONSHIP BETWEEN AROUSAL POTENTIAL (x) AND AROUSAL INDUCTION (y): CONSTRUCTION OF A PARABOLA. F is the focus, r is the directrix.

construction, see Fig. 2).

Equation (1) contains three free parameters: a, b, and d. We shall try to define these parameters in terms of the other quantities in the model, so as to minimize the number of free parameters.

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[3] As an important consequence of the hypothesis of the inverted-U (here interpreted as a parabola) relationship between arousal potential and its effects on arousal, it may be stated that both very high and very low quantities of arousal potential have substantial arousal inducing effects.

Since point (a, b) corresponds to an optimal quantity of arousal potential, i.e., a quantity that affects arousal minimally, we can identify a with that optimal value of arousal potential, x. Because the quantity of optimal arousal is influenced by earlier experiences, it appears justifiable to identify a with the arithmetic mean of the relevant source of arousal potential. To conserve the additivity of effects (an assumption made by Berlyne, see above), the mean must be taken for each source of arousal potential. In the current development we assume two sources – intensity and conflict – each having an optimal value, which we define as follows :

$$O_n(I) = (1/n) \sum_{i=1}^{n} I_i \, ; \tag{2}$$

where $O_n(I)$ refers to the optimal intensity at moment n, I_i to the intensity at moment i; and

$$O_n(C) = (1/n) \sum_{i=1}^{n} C_i, \tag{3}$$

where $O_n(C)$ refers to the optimal conflict at moment n and C_i to the degree of conflict at moment i.

Intensity, I_n, can be defined as a function of the quantity of stimulation at moment n. As we shall see later, the model is constructed such that stimulation comes in via a 16 by 16 binary matrix, s, where each cell $s_n(i,j)$ takes the value 1 if the cell is stimulated and 0 otherwise. We then have :

$$u_n = \sum_{i=1}^{16} \sum_{j=1}^{16} S_n(i,j), \tag{4}$$

where u_n is the intensity at moment n. However, instead of working with this variable, we shall work with standardized variables. Assuming that stimulation of any cell of s is random and independent of the stimulation of the other cells, it follows that the sum of all cells, u_n, is binomially distributed with a mean of 128 and a standard deviation of 8. Standardizing u_n, we get :

$$I_n = (u_n - 128)/8. \tag{5}$$

Berlyne (1957, 1960) defines conflict, C_n, quantitatively as (in our notation) :

$$C_n = H_n . \sum_{i=1}^{m} v_n(R_i), \tag{6}$$

where $v_n(R_i)$ is the associative strength of response R_i at moment n, and H_n refers to uncertainty, which is defined as

$$H_n = -\sum_{i=1}^{m} p_n(R_i).\log_2 p_n(R_i), \tag{7}$$

where $p_n(R_i)$ designates the probability of response R_i at moment n.

Equation (7) is clearly identical with Shannon's (1959) definition of information. The kinship between conflict and information is already apparent in the verbal definition of conflict given above. Moreover, it should be noted that the quantification of conflict does not completely cover the concept as defined, being valid only when the instigated responses are completely incompatible. For our purpose this is quite satisfactory, because we have limited ourselves to experimental situations with incompatible responses. And since in almost all experimental situations response-classes are mutually exclusive (incompatible) and exhaustive, no restrictions are involved.

As Fig. 2 implies, parameter b corresponds to the amount of arousal induction caused by an optimal influx of arousal potential. Because in a learning situation there are no indications to support any particular value for parameter b, we simplify the model by putting $b = 0$, so that (1) becomes :

$$y = (a\text{-}x)^2/2d. \tag{8}$$

We have already seen (see Fig. 2) that d corresponds to the distance between focus and directrix. The larger this distance, the "flatter" the parabola becomes, or the more slowly arousal increases as a function of the distance between arousal potential and its optimum, everything else being held invariant. Therefore, the value of d should not be chosen arbitrarily, because neither too large nor too small arousal inductions are permissible for an adequate functioning of the model. In fact, the amount of arousal induction (y) should not attain its maximum before the distance between arousal potential (x) and its optimum (a) becomes maximal. In this context maximal means infinite. However, since a rather large number of factors influence the arousal potential, it can be argued, on the basis of the central limit theorem, that the distribution of this variable will be approximately normal. Assuming a normal distribution, if the distance between some value and the mean amounts to four standard deviations, we agree to call it maximal, because the probability of the occurrence of such a value is less than .0001.

It follows, then, that y becomes maximal if $a\text{-}x = 4s$, where s designates the standard deviation. Now, the variable y can also be interpreted as a stochastic variable that takes on many different values centred around a mean, i.e., the optimal arousal level or arousal tonus, with some variance greater than zero. Because of the central limit theorem (many stochastic phenomena affect arousal) we may again assume a normal distribution, so that it is meaningful to say that an increase in arousal (y) is maximal if it amounts to four standard deviations. Expressing y in standard deviations, it follows that

$$y = 4 = (a\text{-}x)^2/2d = (4s)^2/2d \rightarrow d = 2s^2.$$

For the sources of arousal potential included in this development of the model, s^2 is defined as

$$V_n(I) = (1/n) \sum_{i=1}^{n} I_i^2 - O_n(I)^2, \tag{9}$$

where $V_n(I)$ designates the variance of intensity (I) at moment n; and

$$V_n(C) = (1/n) \sum_{i=1}^{n} C_i^2 - O_n(C)^2, \tag{10}$$

where $V_n(C)$ designates the variance of conflict (C) at moment n.

Now we can rewrite (8) completely as a function of quantities of the model. We write P_n instead of y to refer to the amount of arousal induction at moment n. Writing P_n as a function of both sources of arousal potential, i.e., intensity and conflict, and preserving the additivity of sources, we get

$$P_n = \frac{1}{2} \left\{ \frac{[I_n - O_n(I)]^2}{4V_n(I)} + \frac{[C_n - O_n(C)]^2}{4V_n(C)} \right\}. \tag{11}$$

In equation (11), additivity of sources is obtained by taking the average of the effects attributed to both sources of arousal potential, so that the model is easily adapted for additional sources of arousal potential without the necessity for completely new derivations.

From Fig. 1 we know already that arousal at any particular moment is a function of the preceding arousal level, of arousal potential and inhibition. Consequently, we define

$$A_n = A_{n-1} + P_n - K_n, \tag{12}$$

where A_n refers to the arousal level at moment n and K_n to the amount of inhibition at moment n.

Before defining inhibition, K, we first introduce the concept of optimal arousal or arousal tonus. According to Berlyne, this is a level of arousal low enough to be attractive and high enough to enable the subject to respond immediately if some event necessitates action. In certain respects it is comparable to the concept of muscle tonus. Moreover, the location of the arousal tonus depends on all kinds of circumstances, such as "…how often the environment has been issuing calls for urgent action" (1960, p. 193). Therefore, arousal tonus is characterized as the average arousal level:

$$O_n(A) = (1/n) \sum_{i=1}^{n} A_i, \tag{13}$$

where $O_n(A)$ refers to arousal tonus.

In Berlyne's view, inhibition functions principally to reduce arousal such that the optimal level is restored. Therefore, we assume that inhibition will increase as arousal exceeds the optimal level. Moreover, it seems justifiable to postulate a non-linear relationship. This interpretation is strongly supported by Gray's (1964) reinterpretation of Pavlovian theory in terms of arousal. Gray suggests, in fact, that inhibition may be identified with transmarginal inhibition (1964, p. 324). Because this kind of inhibition comes into play only after a certain

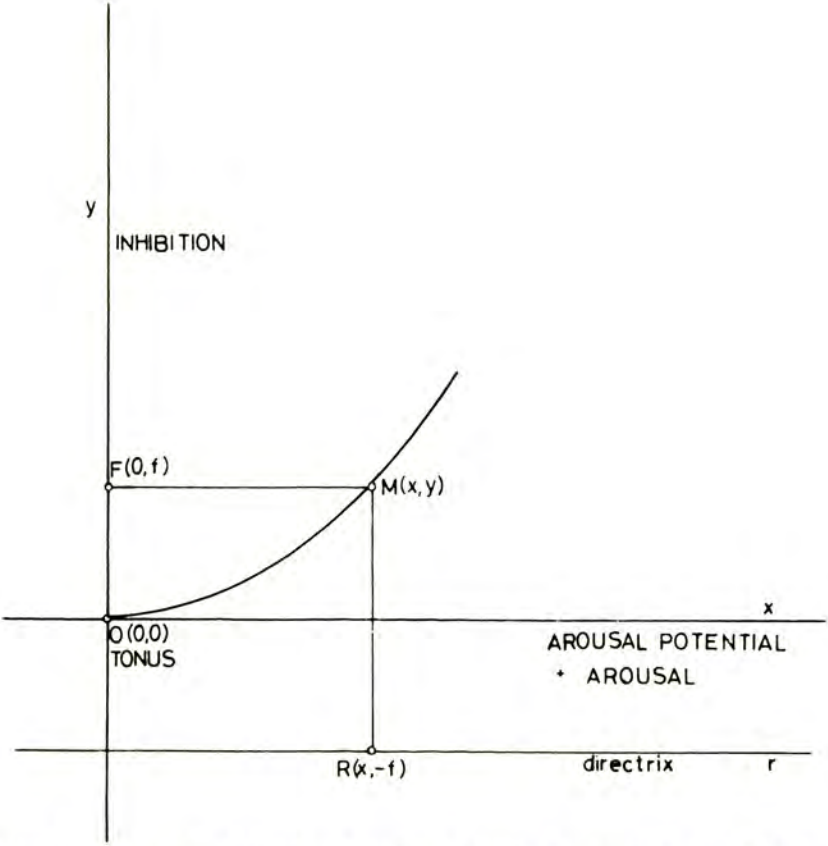

FIG. 3. DEFINITION OF A FUNCTION RELATING AROUSAL POTENTIAL AND AROUSAL TO INHIBITION

quantity of stimulation has reached the sense organs, it follows that the quantity of stimulation (and thus arousal) increases. The best-suited function again appears to be a parabola.

We define a half-parabola in the interval $[o_n(A), \infty]$ (see Fig. 3). We put $o_n(A) = (0, 0)$ and the focus at $(0,f)$. We construct the parabola such that its minimal value coincides with the origin of the

Cartesian system, because with optimal arousal no inhibition is generated. Let x stand for the value of $A_{n-1} + P_n - O_{n-1}(A)$; then, by definition of a parabola, taking the directrix parallel to the abscissa, it follows that

$$y = x^2/4f, \tag{14}$$

where y designates the amount of inhibition and $x = A_{n-1} + P_n - O_{n-1}(A)$. The parameter f can be defined as a function of the other variables of the model, by letting maximal inhibition correspond with maximal arousal. Solving this problem in the same way as for parameter d, we get

$$y = 4 = x^2/4f = (4s)^2/4f \rightarrow f = s^2,$$

and f is interpreted as the variance of the probability distribution of arousal. From (14) we get by substitution :

$$K_n = [A_{n-1} + P_n - O_{n-1}(A)]^2/4V_{n-1}(A), \tag{15}$$

where

$$V_n(A) = (1/n)\sum_{i=1}^{n} A_i^2 - O_n(A)^2. \tag{16}$$

We have thus quantified the main points of arousal regulation as conceptualized by Berlyne (1960, 1963). All necessary information is concentrated in equations 2-7, 9-13, and 15-16. Before relating these arousal-regulating mechanisms to reinforcement, we must develop a model that perceives, evokes, selects, emits, and evaluates or reinforces responses.

A RESPONSE MODEL : LEARNING AND REINFORCEMENT

Most current learning theories embody an interactionistic viewpoint, the learning process being seen as an interaction between the subject's actions on the one hand and influences originating from the environment on the other. This interaction may be described in a cyclic form (cf. de Groot 1969). The cycle begins with a stimulation (the environment affects the subject); the subject responds to this stimulation. This response affects the environment, producing some change in the situation. In turn, this change is perceived, which permits evaluation of the response that caused the change. In essence, this is a feedback loop (cf. TOTE-unit : Miller, Galanter & Pribram 1960), but it is also the typical course of actions in a learning experiment; the main difference appears to be one of terminology : most learning theorists prefer to speak of reinforcement instead of feedback.

A question at the heart of the present investigation concerns the nature of the reinforcing events. Non-motivational theories adopt a descriptive approach, defining reinforcement as an activity of the experimenter (environment) resulting in an increased probability of correct responses. One knows from experience that certain manipulations are reinforcing, and these are preferentially used in experimental situations. Motivational theories, on the other hand, postulate a mechanism that explains why certain stimulations are reinforcing and others are not, the mechanism being some kind of motivational regulation.

With respect to simulation experiments, the difference between the two types of theory is important. Because non-motivational theories are merely descriptive, they can be simulated by simply programming the particular equations. A separate channel must be created to display feedback information, because the theory lacks specification of the perceptive and interpretative processes required by the organism for adequate selection of the feedback information from all incoming information. In motivational theories, on the other hand, these processes are specified, which leads to a more complex model of behaviour: at least a primitive form of perception must be specified in the computer program to enable it to select information adequately. Since non-motivational models lack this specification, the separate feedback channel replaces extensive elaboration of the perceptual processes. Expressed in terms of costs, programming of the perceptual processes of motivational theories surpasses by far those involved in the creation of a separate feedback channel. Because our main interest is in motivational theories and because of the low additional cost of programming non-motivational models, we have implemented models of both types.

Since it was our intention to simultate a motivational theory, we had to allow for these considerations and requirements. Hence, we designed the model such that each learning trial consists of a number of stages corresponding to the following divisions: a. stimulus presentation, the subject or model responds; b. administration of reinforcement, the subject or model evaluates; and c. during a certain interval, no specific stimulation occurs; this interval separates the trials.

We assume that each trial consists of these three parts. It is obvious that in each part a stimulus is presented, causing the subject or the model to respond and to evaluate the preceding response. Consequently, every stimulus is a potential reinforcer. To realize this property, the model is constructed such that every stimulus presentation evokes a chain of activities: perception, response evocation, response selection, evaluation, and response execution. We can now turn our attention to a description of these activities as they are conceived of in the model.

Perception consists of the reception of stimulation introduced into a 16 by 16 binary matrix. This matrix is subdivided into 8×8 blocks each containing 2 by 2 cells. These blocks are called the subsystems of the model. At every given moment, each subsystem receives one of sixteen possible contents, each associated to a certain degree with each of two response classes R_1 and R_2 (the program actually admits more than two classes). In the response-evocation stage, each subsystem instigates one of the two response classes on the basis of a probabilistic decision. Thus, two lists are filled such that there is a fixed one-to-one mapping from the set of subsystems to the cells of each list. If we let i take any value between 1 and 64, then the ith cell of the first list contains the name of the response class instigated by the ith subsystem, while the ith cell of the other list contains the associative strength of that response class in the ith subsystem. For each response class the total strength over all subsystems is calculated by summing all associative strengths. These total strengths are then inserted into the degree-of-conflict formula (eq. 6). Furthermore, on the basis of these total strengths a response is selected by means of a probabilistic process. Finally, the response is executed. Evaluation or estimation of the effect of learning occurs after response selection, although it could equally well be programmed simultaneously with response selection and execution, because in principle these processes are parallel, i.e., their order of occurrence does not affect the result.

Probabilistic response evocation and response selection follow the principles of a model that closely resembles Luce's beta model (see Bush, Galanter & Luce 1959; Luce & Galanter 1963; Restle & Greeno 1970). A short outline of the rationale of this model is in order here.

The model is derived from Luce's choice axiom, which states that omission of any elements of a set of alternatives from which some alternatives are chosen, has no influence on the relative choice of the other elements in the set. Let U be the universe of elements to which the choice is confined, let R and T be finite subsets of U such that $R \subseteq T \subseteq U$, and let P_T and P_U be the probability functions defined on T and U, respectively, then

$$P_U(R/T) = P_T(R). \tag{17}$$

We show how a scale of associative strength, v, can be introduced on the basis of equation (17). Applying the definition of conditional probability ($P_U(R/T).P_U(T) = P_U(R \cap T)$; cf. McCord & Moroney 1964) to (17), we get

$$P_T(R) = P_U(R \cap T)/P_U(T) = P_U(R)/P_U(T) \tag{18}$$

since $R \subseteq T$ implies $R \cap T = R$. We define the strength of an arbitrary element x of R as $v(x) = kP_U(x)$, with $k > 0$. By eq. (18) we get :

$$P_T(x) = kP_U(x)/kP_U(T) = v(x)/\sum_{y \in T} v(y).$$

When the elements of T are response classes it follows that

$$P(R_i) = v(R_i)/\sum_j v(R_j). \tag{19}$$

Given some random number Q between 0 and 1, response R_i is instigated or selected if and only if $P(R_i) \geqslant Q$. During response evocation, $P(R_i)$ is defined on the basis of the associative strengths in the subsystem;

during response selection, $P(R_i)$ is defined on the basis of the total strengths.

For the model to learn, some mechanism responsible for evaluation is needed. As we have already seen, learning occurs only when the probability that some response occurs in some situation is changed and perhaps becomes maximized or minimized. This is achieved by changing the associative strengths of the relevant responses. In this process of changing the strength, we consider only linear transformations of the form

$$v_{n+1} = t.v_n \quad \text{with } t > 0, \tag{20}$$

because, by definition of the v scale, only those transformations make sense.

It is useful to distinguish two kinds of transformations: the first increases the probability of a response by means of increases of the associative strength, whereas the second kind decreases the probability of a response by means of decreases of associative strength. In the terminology of (20), if $t > 1$, t is a transformation of the first kind; if $0 < t < 1$, t is of the second kind.

Usually, increases in probability accompany positive reinforcement. Therefore, we call these learning operators positive operators, and we write L_1. The other operators, which correspond to negative reinforcement, are similarly called negative learning operators; we designate them by L_2.

To arrive at a general definition of evaluation in our models, we introduce some variables: $v_n(j,i)$ represents the v-strength of response j in subsystem i at moment n (it should be noted that v always represents the strength of the association between a particular stimulus content and a specific response class). Further, $x_n(j,i) = 1$ if response j is evoked in subsystem i at moment n; otherwise, $x_n(j,i) = 0$. If positive reinforcement is administered at moment n, we define $z_n = 1$; otherwise $z_n = 0$. Applying (20), evaluation is defined by

$$v_{n+1}(j,i) = v_n(j,i).L_1^{z_n.x_n(j,i)}.L_2^{(1-z_n).x_n(j,i)}, \tag{21}$$

where $L_1 > 1$, and $0 < L_2 < 1$.

MOTIVATIONALLY STEERED EVALUATION

In motivational learning theories, reinforcement is coupled with motivational conditions. This means that for our model z_n is defined as a function of changes in arousal. Moreover, it does not seem implausible to suppose that these changes influence the rate of learning; this might be mediated by the magnitude of the learning operators.

With respect to this latter supposition, Berlyne's theory is not clearly formulated. At least three interpretations are possible: a. the magnitude of the learning operators, L_1 and L_2 in (21), is proportional to the amount of change of arousal; b. the magnitude of the learning operators is invariable for specific learning tasks; c. the magnitude of the learning operators is not invariable but depends on several factors.

We shall develop all three interpretations : the first and third lead to models with variable learning operators (MV and MN), the second leads to a model with constant operators (MK).

Motivational model with constant operators (MK) : In the MK model evaluation follows (21) and $z_n = 1$ if and only if $A_n - A_{n-1} - \sqrt{V_n(A)} \leqslant 0$; otherwise, $z_n = 0$. This implies that all decreases of arousal and those increases that are less than a standard deviation have a positive influence on the probability of a response. In this definition allowance is made for the facts which compelled Berlyne to modify his theory [4].

Motivational model with variable operators (MV) : In the MV model, which is based on the first interpretation given above, we have to define learning as a function of the magnitude of arousal changes. The function defining this operator, E_n, must meet several requirements :

a. E_n is a function of the difference between A_n and A_{n-1} ;

b. the function is continuous;

c. if $A_n = A_{n-1} + \sqrt{V_n(A)}$, then $E_n = 1$;

d. if $|A_{n-1} - A_n + \sqrt{V_n(A)}| > |A_{m-1} - A_m + \sqrt{V_m(A)}|$, then $E_n < E_m$; and

e. $0 < E_n \leqslant 1$.

It is evident that by reason of condition (e), the variable E_n is not applicable as a positive operator; it is easily shown, however, that $1/E_n$ fits this purpose perfectly.

Consistent with these five conditions, we define E_n as :

$$E_n = V_n(A)/(\sqrt{V_n(A)} + |A_{n-1} - A_n + \sqrt{V_n(A)}|). \qquad (22)$$

Defining z_n as in the MK model, and taking $L_1 = 1/E_n$ and $L_2 = E_n$, (21) remains useful.

Motivational model with normally distributed operators (MN). If the magnitude of the learning operators depends on several factors, as in the third interpretation given above, it appears justifiable to assume that the magnitude of the operators is normally distributed. Especially when the number of influential factors is rather large, the central limit theorem provides a useful basis for argumentation. According to this line of reasoning the positive learning operator, D_{1n}, is sampled from a normal distribution with mean L_1 and an empirically estimated

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[4] Empirical evidence suggests that in addition to arousal reduction, slight increases of arousal are also rewarding (see Berlyne 1971). In a rather arbitrary way we therefore assume that increases of arousal amounting to less than a standard deviation are rewarding. We have followed the same reasoning for all of the motivational models.

variance. Analogously, D_{2n} is sampled from a normal distribution with mean L_2. With z_n defined as in the MK model and taking $L_1 = D_{1n}$ and $L_2 = D_{2n}$, (21) remains valid for this model too.

NON-MOTIVATIONALLY STEERED EVALUATION

Since non-motivational models handle reinforcement as a descriptive concept and are easily programmed, it requires no great effort to enlarge the research design to encompass such models. Therefore, two models were included, one with constant and one with normally distributed learning operators. Except for the evaluation, both models are identical to motivational models MK and MN. In motivational models evaluation depends on the interaction of arousal changes and information from the environment, whereas in non-motivational models evaluation depends solely on the environmental information.

Non-motivational model with constant operators (NK): In the NK model evaluation is defined as in (21) with $z_n = 1$ when positive reinforcement occurs and $z_n = 0$ otherwise.

Non-motivational model with normally distributed operators (NN): The only way to construct variable operators in a descriptive model is by sampling them from a probability distribution. For this model to match the MN model, the normal distribution is assumed. (21) gives the rule for evaluation with z_n defined as in the NK model [5].

VALUE OF THE MODELS AS REPRESENTATIONS OF AROUSAL THEORY

Before confronting these models with empirical data, the degree to which they represent Berlyne's earlier arousal theory must be determined. In inferential simulation, the appropriate method here, it is indispensable that the model or program represent the theory at least approximatively.

Proceeding from Berlyne's 1960 theory of arousal, we first indicated the aspects that are relevant for our purpose. Next, adhering consistently and closely to his formulations, we developed a quantification of Berlyne's theory. It is striking that very few difficulties have arisen, which implies that, at least as far as the part under study is concerned the theory is relatively well formulated. We were only forced to use interpretations for the concepts inhibition and effect of learning (the learning operators). For the interpretation of inhibition, Gray's (1964) view was adopted. After elaborating this position quantitatively, we arrived at a formulation consistent with Berlyne's statements about inhibition.

[5] It may be remarked that the MK model matches the NK model and that the MN model matches the NN model, whereas the MV model stands alone. This is not completely true. We suppose that the NN model can serve as a basis for comparison for the MV as well as for the MN model. The extent to which this supposition is valid depends on the degree to which the distribution of MV operators approximates a normal distribution.

Concerning the interpretation of the effects of learning, we put forward three possible candidates and based a model on each of them, i.e., MV, MK, and MN. It is obvious that these models cannot be valid simultaneously : research is required to show which interpretation would survive the data.

Roughly speaking, the models we developed give an adequate representation of Berlyne's theory. However, a difficulty arises. In his first formulations of the theory Berlyne (1960, 1963) suggested that increases of arousal are unattractive and thus not rewarding, but punitive. In more recent formulations (e.g. Berlyne 1971) he claimed that slight increases of arousal are rewarding. And although it would seem more appropriate to quantify this recent version of the theory, we decided not to do so because the postulated neuronal mechanism is very complicated and deviates materially from the mechanism hypothesized in the older version of the theory. Since the present models were already programmed when these new formulations came to our attention, the best procedure seemed to consist of an adaptation of the models such that slight increases of arousal are rewarding (see also footnote 2).

SITUATIONAL PARAMETERS

We tested the models in an instrumental learning task. This task had already been used by Sternberg (1959b) in a comparative study of some learning models.

In this task the subjects, human adults, have to handle an apparatus (two-armed bandit), closely resembling a gambling device. It is provided with two response keys, one on the left, the other on the right. On each trial, subjects have the choice between these two response keys, only one of which yields gain or positive reinforcement.

The beginning of a trial is announced by a signal (light plus buzzer). The subject is then allowed to insert a chip to activate the device, and to press the key of his choice. After he makes this response a light appears above the key that has been pressed and another light illuminates the payoff slot. If the subject presses the correct key, a chip drops into the payoff slot within .5 seconds after the response. If the incorrect key is pressed, no chip is delivered. About one second after the response, the end of the trial is announced by the extinction of all of the lights. Two or three seconds later, a new trial can begin. The reinforcement schedule is a 100:0 CRF schedule, positive reinforcement, always ensuing from the left key.

For comparison of the results obtained with our models with the performance of human subjects, we shall use Sternberg's data. Each subject participated in the experiment until he reached a criterion of 15 consecutive correct responses, with the assumption that all responses that would follow this series are correct.

This learning task contains four stimulus situations of importance for our models : a. the state of the device before the subject responds, the ready-signal included (experimental stimulus); b. the state of the device after a correct response has occurred (positive reinforcement); c. the state of the device after an incorrect response has occurred (negative reinforcement); d. the state of the apparatus between two trials (intertrial stimulus).

By virtue of the construction of our learning models, they will receive three of these four stimuli, on every trial. Furthermore, these stimuli must be adequately selected, because in the motivational models it depends partly on them whether arousal reduction ensues. Therefore, the following conditions must be met : a. the stimuli must be composed such that transfer of responses from one to the other is minimized : in other words, the number of common components must be minimized; b. optimal intensity and variance of stimulation must remain as stable as possible throughout the experiment; c. the positive reinforcer must be arousal-reducing; d. the negative reinforcer must induce arousal to a moderate degree.

On the basis of these conditions it is possible to infer what intensity must be assigned to the reinforcers by using the equations of the model. Once the values of these stimuli are known, the other intensities can be determined according to condition b : this yields two quadratic equations with two unknowns. (For an elaborated presentation of this solution process, the reader is referred to Vandierendonck 1973). Thus, in solving the problem we got an intensity of 136 for the experimental stimulus, 128 for the positive and 115 for the negative reinforcer, and 133 for the intertrial stimulus. On the basis of these results there was no difficulty in satisfying condition a.

ESTIMATION OF FREE PARAMETERS

In all of the developed models, except MV, the learning operators are free parameters. Moreover, the initial probability of a correct response is arbitrary for all models. The parameter values are usually chosen such that the model resembles the data as much as possible. Consequently, to estimate the parameters, the data must already be known.

We did not wish, however, to follow the common practice of estimating the parameters on the basis of the data themselves. Yet we wanted to work with optimal values. Therefore, we took a random sample comprising 27 of Sternberg's 77 subjects as the basis for our estimation. By means of Monte Carlo computations, the three free parameters of the NK model were estimated so as to give maximal correspondence with the data of these 27 subjects. We used these values for all our models. In the NN and MN models the values for the learning operators were used as means of the normal distribution. The variance of this distribution must, however, be determined arbitrarily. To differentiate maximally between the models with variable operators and those with constant operators, we chose variances of operators as large as possible, but avoiding non-allowed values of the learning operators by not taking over-large variances. The values of the parameters are shown in Tab. 1.

parameter	estimated value
initial probability of a correct response	.52 (a)
positive learning operator	1.33 (b)
negative learning operator	.93 (b)
variance of positive learning operator	.35 (c)
variance of negative learning operator	.15 (c)
intensity of experimental stimulus	136.00 (d)
intensity of positive reinforcer	128.00 (d)
intensity of negative reinforcer	115.00 (d)
intensity of intertrial stimulus	133.00 (d)

TAB. 1. ESTIMATED VALUES OF FREE PARAMETERS. a. For all models. b. For all models except MV. c. Only for MN and NN. d. Only for motivational models.

To evaluate the usefulness of the parameter values, we compared the performance of the NK model with that of the sample of subjects used for estimation. The Wilcoxon matched-pairs signed-ranks test (Siegel 1956, p. 75 ff.) yielded a T of 155; this corresponds to a standard normal z of .52. The probability under the null hypothesis of finding a difference corresponding to a z greater than or equal to .52 amounts to .603. The product-moment correlation coefficient between both series of performances over 26 trials yielded a value of .887. The probability of getting a value of at least that magnitude is, under the assumption of linear independence, smaller than .001.

These findings suggest that a certain degree of confidence in the estimated values of the parameters is not unwarranted. Consequently, application to a new sample may teach us something about the value of our models.

A TEST OF THE MODELS

Although our models are labeled either motivational or non-motivational, they are all learning models. Consequently, we are interested mainly in learning performance. This performance is characterized by the number of incorrect responses and by the course of correct and incorrect responses, i.e., the learning curve. For a more thorough study of these models, statistics may be used that give a picture of the degree of perseveration and of the degree to which errors are related to each other. These statistics yield especially information about the tenability of the assumption of independence of path, which is at the basis of all our models. This assumption implies that the probability of a

trial	S	MV	MK	MN	NN	NK	MV(+)	MK(+)	MN(+)
1	31	22	21	25	15	21	4	10	12
2	25	21	23	31	16	20	8	10	15
3	32	12	17	19	16	16	0	6	7
4	28	17	24	21	19	14	0	14	9
5	19	16	17	18	18	17	1	7	7
6	21	24	18	21	15	9	3	7	6
7	14	29	15	22	14	7	5	5	8
8	9	24	18	19	17	10	3	10	5
9	13	28	13	20	14	3	4	3	3
10	5	27	14	21	0	1	5	3	3
11	5	34	11	20	10	2	6	1	4
12	3	32	14	18	7	0	6	1	4
13	2	33	15	19	6	2	6	2	4
14	1	40	14	22	6	0	5	0	5
15	2	38	15	18	3	0	4	1	2
16	2	31	13	16	4	1	3	0	2
17	1	33	15	17	3	0	4	1	1
18	3	33	14	18	2	0	1	0	0
19	0	31	13	20	2	0	1	0	0
20	0	29	14	20	3	0	2	0	0
21	0	33	14	18	1	0	2	0	0
22	2	34	15	20	3	0	2	1	1
23	1	34	13	19	1	0	1	0	0
24	0	33	13	16	1	0	1	0	0
25	0	34	14	16	1	0	1	0	0
26	0	30	11	18	1	0	1	0	0
27	0	33	12	18	1	0	0	0	0
28	0	34	9	18	1	0	0	0	0
29	0	33	8	19	1	0	0	0	0
30	0	32	11	17	2	0	0	0	0
31	0	31	10	17	2	0	0	0	0
32	0	28	8	18	0	0	0	0	0
33	0	29	7	18	0	0	0	0	0
34	0	28	8	14	0	0	0	0	0
35	0	30	9	17	0	0	0	0	0
36	0	28	8	18	0	0	0	0	0
37	0	30	6	14	0	0	0	0	0
38	0	28	8	17	0	0	0	0	0
39	0	27	11	18	0	0	0	0	0
40	0	29	5	14	0	0	0	0	0
total	219	1172	518	749	215	123	79	80	78
N	50	50	50	50	50	50	12	36	29
mean	4.4	23.4	10.4	15.0	4.3	2.5	6.6	2.2	2.7

TAB. 2. NUMBER OF INCORRECT RESPONSES PER TRIAL UNDER ALL CONDITIONS

given response-class depends solely on its probability on the preceding trial and the kind of reinforcement that followed. This means that the performance of response A on trial n depends only on its probability and not on the fact that response A was already performed on any preceding trial. Consequently, neither perseveration nor correlation of errors should show up in the data.

Such a study of the fundamental properties of the learning models is only of value if the gross characteristics of the models yield an adequate description of the learning curves and the number of errors. Therefore, we turn our attention first to these general characteristics. Tab. 2 shows the results for the subjects and the models in 40 trials, on the basis of 50 replications for each condition. Inspection of this Table indicates that learning is slower for the motivational models than for the non-motivational models or the subjects, which already suggests that the motivational models are less adequate than the non-motivational ones.

Table 3 shows the results of an analysis of variance of the data in Tab. 2. The most suitable kind of design for investigations of this type seems to be a two-factorial design with repeated measures on one factor (for technical details : see Winer 1962, p. 302 ff.). The argumentation on which this conclusion is based is as follows. Since simulation is always oriented toward null hypothesis acceptance, it is expedient to use this design, because when it is assumed that the variance between models and subjects must be smaller than the variance of performances, the null hypothesis can only be accepted after several conditions are met (this point is discussed at length in Vandierendock, 1975). When this design is combined with the use of correlations between performances, the quality of the null hypothesis is put to the test quite severely, as stipulated by Grant (1962).

Part A of Tab. 3 shows an over-all analysis of all conditions, i.e. for models and subjects. It is evident that several of these conditions do not correspond well with each other. The differences between conditions explain about 18% of the total variance, the differences within conditions due to performance in trials explain only 3%. Moreover, it may be concluded that the learning curves are not quite parallel, since the interaction between conditions and trials explains another 6% of the variance. Therefore, it seemed appropriate to try to find the cause of these rather large deviations.

For this purpose – and above all because we also wanted to compare each model individually with the subjects – we made comparisons of the means, each involving one model and the subjects. The results of this analysis are shown in part B of Tab. 3. The performance of the motivational models deviates significantly from that of the subjects. The performance of the non-motivational models, on the contrary, shows close agreement with that of the subjects. So we may conclude tentatively that the non-motivational models give a sufficiently approximative description of the subjects' performance. An analysis of the interaction (interaction of comparisons) indicates that the NN curve takes the same course as the NK curve. However, neither of these curves agrees well with that of the subjects, which means that although the general level of performance of both non-motivational models is fairly close to that of the subjects, the course of learning, and more specifically (as can be inferred from Tab. 2) the rate of learning, are less similar. Nevertheless, the resemblance is quite satisfactory.

Part C of Tab. 3 gives the product-moment correlation coefficients between the mean learning curves of each model and the subjects. All correlations deviate significantly from zero; moreover, all of the correlations are high, even that of the MV model, which bears a negative

A. main analysis

source	SS	df	MS	F	p	ω^2
conditions (1)	408.11	5	81.62	37.78	< .001	.18
replications within conditions (2)	635.04	294	2.16			
between subjects	*1043.15*					
trials (3)	73.87	39	1.89	21.24	< .001	.03
(1) x (3)	109.94	195	.56	6.29	< .001	.06
(2) x (3)	1021.04	11466	.09			
within subjects	*1204.85*					
total	2249.00					

B. planned comparisons

source	SS	df	MS	F	p	ω^2
S-MV	227.05	1	227.05	105.11	< .001	.10
S-MK	33.67	1	33.67	15.60	< .001	.02
S-MN	70.23	1	70.23	32.51	< .001	.03
S-NN (4)	.00	1	.00	.00	ns	
S-NK (5)	2.30	1	2.30	1.06	ns	
(3) x (4)	8.42	39	.22	2.47	< .001	.00
(3) x (5)	7.00	39	.18	2.02	< .001	.00

C. correlation coefficients

r(S,MV) = −.80	$p < .001$	
r(S,MK) = .76	$p < .001$	$P(.68 \leqslant \rho\,(S,MK) \leqslant .82) = .95$
r(S,MN) = .60	$p < .001$	$P(.48 \leqslant \rho\,(S,MN) \leqslant .69) = .95$
r(S,NN) = .89	$p < .001$	$P(.85 \leqslant \rho\,(S,NN) \leqslant .92) = .95$
r(S,NK) = .95	$p < .001$	$P(.93 \leqslant \rho\,(S,NK) \leqslant .96) = .95$

TAB. 3. RESULTS OF STATISTICAL ANALYSES

sign. As can be seen from Tab. 2, the learning curve of the MV model rises from the sixth to the fourteenth trial, after which it shows a slight downward trend. The curve of the subjects, on the other hand, is completely different, which explains the negative correlation. Again, we find a closer approximation between the non-motivational models and the performance of the subjects than between the motivational models and the subjects. Moreover, the correlation between the NK model and the subjects is significantly higher than that between the NN models and the subjects; in other words, the NK model is significantly less different from the subjects than the NN model.

From this initial analysis of the results it is unequivocally clear that the motivational learning models fail to give a correct description of the subjects' behaviour in an instrumental learning task. We shall postpone discussion of this point, and concentrate here on the non-motivational models to inquire into the so-called detail statistics, which provide indications about the tenability of the assumption of independence of path.

Suitable statistics for this purpose (Sternberg 1959b) comprise the number of runs of i consecutive errors (e_i), the autocorrelation (or error correlation) lag k (c_k) which yields an expression for the degree to which an error on trial n is correlated with an error on trial n+k, for all n, and the influence of an error on trial n on the response on trial n+1, for all n (B). More formally, in accordance with Sternberg, who developed some of these statistics to test his path-dependent model (1959a, 1959b), these statistics are defined as follows (see also Atkinson, Bower & Crothers 1965):

$$c_k = \sum_{n=1}^{N-k} x_n . x_{n+k} ; \tag{23}$$

where $x_n = 0$ if the response on trial n is correct, and $x_n = 1$ otherwise; and N is the total number of trials.

$$B = \sum_{n=1}^{N-1} x_n . x_{n+1} - (1-x_n)x_{n+1} . \tag{24}$$

In equation (23) the product $x_n . x_{n+k} = 0$ until both responses on trials n and n+k are incorrect. Therefore, equation (23) yields a picture of the extent to which incorrect responses go together 1, 2, ..., k trials apart. The B statistic may be considered a corrected error correlation lag 1: the first term in (24) is c_1, while the second one stands for the degree to which correct responses are followed by an incorrect one, or in other words the extent to which errors occur without being evoked by a directly preceding error.

We used these statistics not to score models NN and NK, but rather to find out the extent to which their scores deviate from those of the subjects. Tab. 4 shows the relevant scores of the subjects and models NN and NK, with indication of whether the differences are statistically significant. In calculating these statistics we selected only the most important values: e_i and c_k for i and k varying from 1 to 5.

From Tab. 4 we can infer that with respect to the e_i statistics, both models (NN and NK) show too many error runs of length 1, which is compensated for by a shortage of error runs of lengths 2, 3 and even 4. As far as the error correlations are concerned, there is almost no difference between the NN model and the subjects, but all differences between the subjects and the NK model are statistically significant.

Finally, concerning the B statistic – the measure of choice for the evaluation of short-term perseveration – both models differ significantly from the subjects.

These results indicate that the assumption of independence of path is not quite tenable: for almost all of the perseveration statistics the NK model fails. The NN model, on the other hand, shows, besides some manifest deviations, a certain degree of resemblance to the subjects. This evidence does not lead to a decisive answer to the question of whether the independence assumption is valid for the subjects, although it points in the direction of untenability. In any case, the possibility of a model that incorporates this assumption and nonetheless yields a suitable description of human performance in instrumental learning tasks must be allowed for. However, the fact that most path-independent models fail (see also Sternberg 1959b) should not be underestimated.

statistic	mean			difference	
	subjects	NN	NK	S-NN	S-NK
e_1	1.22	1.70	1.54	ns	ns
e_2	.36	.78	.10	< .01	< .01
e_3	.24	.06	.06	< .05	< .05
e_4	.18	.06	.02	ns	ns
e_5	.10	.04	.06	ns	ns
c_1	2.22	1.60	.66	ns	< .001
c_2	1.94	1.46	.84	ns	< .001
c_3	1.34	1.26	.46	ns	< .01
c_4	1.02	1.20	.46	ns	< .05
c_5	.80	.98	.32	ns	< .05
B	.68	-.80	-.72	< .01	< .01

TAB. 4. ERROR RUNS AND ERROR CORRELATIONS OF SUBJECTS AND OF NN AND NK MODELS

EXPLORATORY INVESTIGATION OF MOTIVATIONAL MODELS

The most striking result of the present investigations is the failure of the motivational models to describe the performance of human subjects in an instrumental learning task. Therefore, the possible causes of this phenomenon deserve attention.

It is obvious that the failure of these models does not generally hold for all replications. Therefore, it does not seem justified to conclude that motivational models learn more slowly than their non-motivational counterparts. An inspection of the raw data (not presented here because of lack of space) shows that some replications reach the criterion of consistently correct responding quickly, whereas others give the impression of striving toward an asymptote of consistently incorrect responding. Therefore, we divided the replications of each motivational model into two sets, the first comprising those attaining the criterion of 15 consecutive correct responses within 40 trials, the second includes all other replications. This leads to some remarkable results which we will now analyse.

For convenience, we shall designate the set of replications attaining the criterion by a plus sign, e.g. MV(+), and the other set by a minus sign, e.g. MN(–). Tab. 2 shows the results for models MV(+), MK(+),

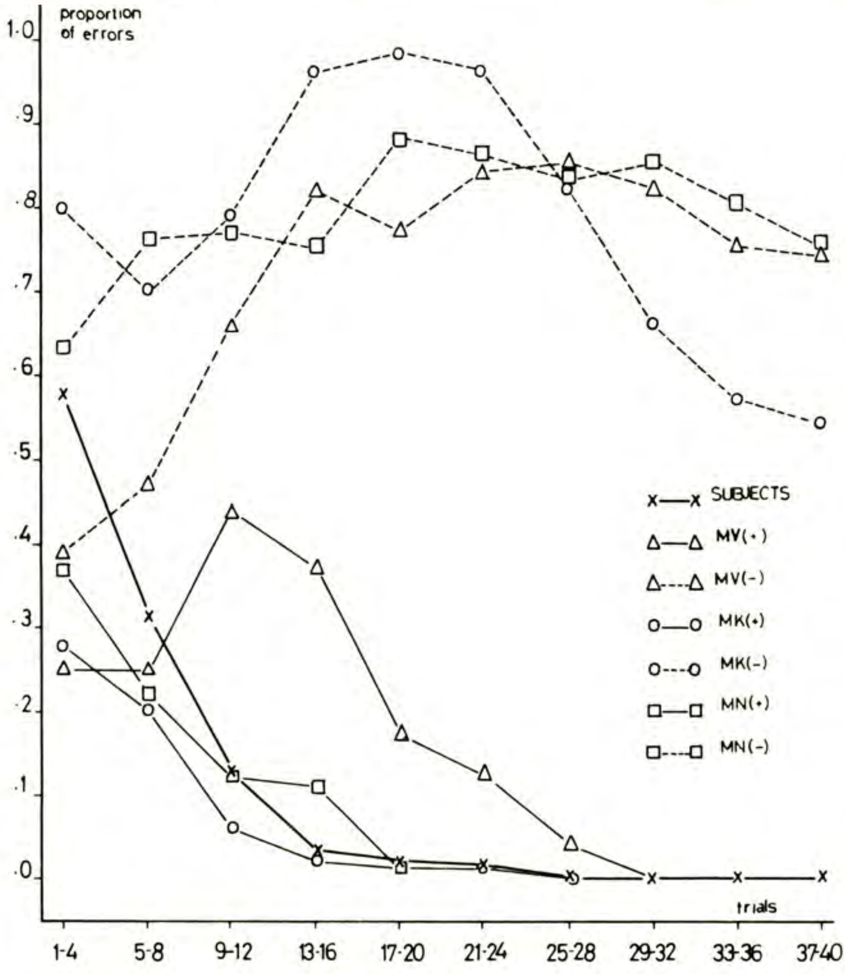

FIG. 4. LEARNING CURVES OF SUBJECTS, MK(+), MK(–), MV(+), MV(–), MN(+), AND MN(–)

and MN(+), with indication of how many replications satisfy the (+) criterion. Fig. 4 shows the learning curves of MV(+), MK(+), MN(+), MV(–), MK(–), and MN(–) and the subjects in blocks of 4 trials.

It should be stressed that the partition of the replications into plus and minus sets is not arbitrary but is based on a clear cut in the data. None of the replications of the minus set reaches the criterion within 40 trials, but none of them would reach the criterion within 50 trials either. The reader can convince himself of this striking phenomenon from Tab. 2 : of the 38 MV(–) replications, 29 still err on the 40th trial; of the 14 MK(–) replications, 11 err on trial 39 and of the 21 MN(–) replications 18 are still erroneous on trial 39. This implies that even if these replications made no further errors they would need about 55 trials to reach the criterion. It is even extremely unlikely that this would happen, especially since the computer trace shows that for most of these minus replications the probability of an incorrect response amounts to .80 or more. This undoubtedly concerns a bimodal distribution of performance, implying that the plus replications learn to execute the correct response, whereas the other learn to respond incorrectly.

In view of this bimodality of the data, it is less surprising to note that the learning curves of MK(+) and MN(+) approximate the learning curve of the subjects quite closely (see Fig. 4). Except for the MV(+) curve, which differs visibly from the subject curve, the plus curves show a noteworthy resemblance to the subjects' learning curve. Like the NK curve, the MK(+) curve lies lower than that of the subjects. Application of the Wilcoxon matched-pairs signed-ranks test indicates that the probability of the differences is smaller than .01 under H_0. The MN(+) curve shows good agreement with that of the subjects: the probability of the difference is about .31 under H_0 by the same test.

This analysis shows that there really is some evidence supporting the conclusion that motivational models (except possibly the MV model, whose learning operators are too variable) can approximately describe learning of human subjects if no circumstances disrupting learning occur. How these circumstances can be characterized remains questionable. Yet, the degree of variability of the learning operators certainly plays a part, although it cannot be the whole story. Even though variability is limited and controlled in the MN model and there is no variability of operators at all in the MK model, both models show deficiencies too often to ignore.

Another factor is suggested by the fact that the minus replications tend to reach a criterion of consistently incorrect responses. Since all of the models start with a slight bias for correct responses (by estimation of the initial probability of a correct response), it follows that these replications must learn in some way to perform the incorrect response, which can only be achieved by positive reinforcement of the incorrect response. In other words, the negative reinforcer must frequently have been paired with too little or too much arousal induction. This is not altogether unlikely, because by definition the intensity of the negative reinforcer increases arousal. If, however, at the moment of reinforcement the arousal level is already moderately high, i.e., above tonus level, arousal can increase so much that it becomes substantially reduced by inhibition. Consequently, arousal is reduced or only slightly increased, so that positive reinforcement occurs when negative reinforcement was indicated.

From this line of argument it is obvious that a model that learns only by positive reinforcement, should not show this deficiency at all or at least to a lesser extent. Such a model will clearly have other draw-

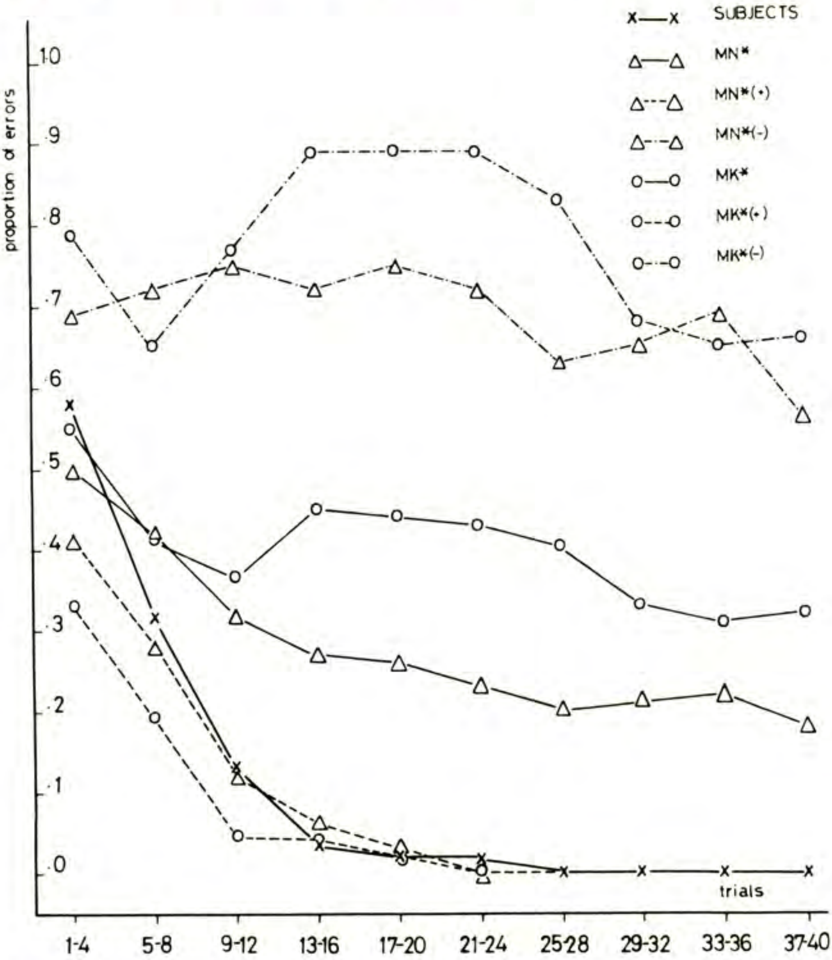

FIG. 5. LEARNING CURVES OF SUBJECTS, MK*, MK*(+), MK*(–), MN*, MN*(+), AND MN*(–).

backs, such as slower and less efficient learning, because negative information is ignored. To check this prediction, we have developed two models in which learning occurs only on positively reinforced trials. These models, called MK* and MN*, are variants of MK and MN, respectively, with which they are, except for reinforcement, in all respects identical – even in the value of the parameters.

We ran 25 replications of each model. The over-all learning curves are shown in Fig. 5. Once more, the deficiency persists, but not to the same degree. For model MN*, 17 of the 25 replications are of the plus type, but for model MK* only 13 out of 25.

Fig. 5 also shows the mean learning curves of replications MK*(+), MK*(–), MN*(+), and MN*(–). With respect to these curves we wish to stress that :

a. although a delay is expected when negative information is ignored in the learning process, learning of MK*(+) and MN*(+) is faster than that of the subjects (see also Fig. 4);

b. although on average the earlier models with variable learning operators learn more slowly than identical models with constant operators the MN* model proved to learn more rapidly than the MK* model. This suggests that the restriction of reinforcement to cases of arousal reduction has more influence on the MN model than on the MK model. Moreover, learning is slower in MK* than in MK, but faster in MN* than in MN. This implies that the modifications of the MK model have an effect, but not the intended one. Therefore, we tend to conclude that disconnection of negative reinforcement and arousal induction is only useful for the model with variable operators.

Although a certain degree of constraint on the variability of the learning operators and disconnection of reinforcement and arousal increases are factors that manifestly influence the learning performance of motivational models, the average number of errors made remains too high for these models to be useful. Yet these models were constructed adequately, and appropriate parameter values were used. Consequently, we are confronted with a finding which has far-reaching consequences. Since the conditions of the test appear to be optimal, the deviating performance of the motivational models seems to indicate that the fundamental assumption about the learning process, viz. the motivational steering of reinforcement, is wrong, We do not deny that arousal is a relevant concept for the psychology of motivation; we do, however, doubt its relevance for reinforcement theory. The research reported here, indicates that it is not plausible to assume that at least negative reinforcement is dependent on arousal regulation.

DISCUSSION

Several of the findings discussed in this report are of particular interest :

a. as far as learning is concerned, the non-motivational models (NN and NK) agree better with subjects than do the motivational models;

b. the NN model describes subject behaviour at least as well as the NK model does, and moreover, yields a slightly better approximation for some perseveration statistics;

c. in certain as yet undefined and unidentified circumstances motivational models describe human instrumental learning more or less adequately; in other, likewise unidentified, circumstances, learning is disrupted.

On the basis of this last finding it may be suggested that for learning of motivational models to follow the expected pattern, certain limiting conditions are necessary. In some replications it would not be necessary to activate these conditions, viz. in those reaching the criterion. In the other replications the limiting conditions should function to avoid disruption of performance.

Whatever the validity of this suggestion, it is obvious that motivational models are defective. Apparently, the theory on which these models are based is defective. As has already been pointed out, Berlyne, who formulated this theory, is not convinced of its value and has since developed a new version.

The evidence discussed above gives good grounds to doubt the validity of any theory that identifies reinforcement with arousal regulation. We have shown, for instance, that negative reinforcement does not function that way, and this may also be true of positive reinforcement. In any case, our research has shown that the difficulties in Berlyne's 1960 theory are rooted more deeply than at the level of the problem of the exact relationships between arousal and reinforcement, which was the principal motive for the reformulation of the theory (cf. Berlyne 1971). Rather, the fundamental hypothesis stating that reinforcement is regulated by arousal is called into question. Because this hypothesis also underlies the reformulated theory, the second version can be questioned too. Studies based on the revised version of the theory are needed to settle the issue decisively.

The present investigation has also shown that a non-motivational model with normally distributed variable learning operators describes the behaviour of the subjects fairly well. Unfortunately, the number of free parameters is quite large : 5. Nevertheless, the NN model predicts a larger variability in performance than the NK model; this leads to a more realistic description. Moreover, variability of operators may be interpreted in a broader context, offering points of view for theory construction. Thus, it may be supposed that this variability is caused by fluctuations of attention, motivation, and the like. Although this does not seem very promising ground for new research, at least exploratory studies along these lines are indicated.

Of the present results, the one with a small degree of certainty is the finding that the assumption of independence of path is not tenable in human instrumental learning. The evidence indicated here is not completely convincing and could easily be predicted by a model showing only slight deviations from this assumption. Another interpretation argues that in the learning task in question, perseveration must be attributed to the subject's hypothesis-testing behaviour. Just as in concept learning, subjects would try different hypotheses concerning the cues inherent in the situation. In any case, continued research on the tenability of the independence assumption seems very important for the formulation of an adequate learning theory.

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