Classical Conditioning: Still Going Strong

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This paper summarizes developments in the field of classical conditioning. Attention is paid to four common misconceptions of what is classical conditioning. First, classical conditioning does not ensue as a simple result of temporal pairing of conditioned and unconditioned stimuli. Rather, conditioned reacting occurs if and to the degree that the subject is able to predict the occurrence of one stimulus from the presence of another one. Second, what is learned during classical conditioning is not necessarily a response to a cue, but rather a probabilistic relationship between various stimuli. Third, classical conditioning is not only manifested in responses mediated by the autonomic nervous system, but also in immunological parameters, in motoric behaviour and in evaluative judgments. Fourth, the nature of the conditioned and the unconditioned stimulus is (often) not a matter of indifference: particular combinations of CS and US produce more powerful conditioning effects than do other combinations. In the second part of the paper, the potential relevance of these developments is illustrated. Discussions are included about anxiety, addictions and food aversions/conditioned nausea.

Introduction

Together with work on operant behaviour, Pavlov's seminal experimentation on classical conditioning was one of the main foundations of behaviour therapy. Indeed, the findings on classical conditioning long served as the raison d'être for behaviour therapy (e.g. Wolpe, 1973). By the end of the 1970s, however, learning theory appeared to many behaviour therapists to offer too narrow a basis for conceptualizing and treating abnormal behaviour. Behaviour therapy thus "went cognitive". The cognitive "revolution" was successful: cognitive concepts rapidly became accepted by practising behaviour therapists and cognitive strategies became incorporated in the standard practice of (cognitive) behaviour therapy. Although there have been many positive aspects of this cognitive shift, there is at least one drawback: it directed attention away from the important

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progress being made in research on classical conditioning (CC). (Cognitive) behaviour therapists often maintain ideas about CC that are outdated.

With the work of, for example, Rescorla and Wagner (1972) and Kamin (1972), classical conditioning has become compatible with cognitive notions. In fact, cognitive concepts are at the heart of modern views on what classical conditioning is about (e.g. Davey, 1988; Mackintosh, 1983). Many of these insights are relevant to understanding and treating abnormal behaviour.

Pavlov was no behaviour therapist and his scientific legacy is nowadays being invested by scientists for whom treatment of abnormal behaviour still lies outside their realm of interest. Meanwhile, behaviour therapists have free access to the modern pay-offs of Pavlov’s earlier work. The aim of this paper will be to illustrate the importance and relevance of recent accomplishments with regards to CC. We will not try to be exhaustive in our coverage, however. The focus will be on major changes that have taken place in the field of CC and clinical implications will also be outlined. In doing so, we will attack some all too common misconceptions of what CC is about and will draw attention to more plausible, “neo-Pavlovian” conceptualizations. Attention will be drawn to the fact that Pavlovian and cognitive accounts of learning are rapidly converging.

Common misconceptions

Classical conditioning usually refers to a procedure by which a previously neutral stimulus (CS) comes to elicit a response after pairing it with a unconditioned stimulus (US). If meat (US) is reliably preceded by a bell (CS), salivation of a dog will not only be observed during meat presentation (UR), but will come to occur upon hearing the bell (CR). The procedure works efficiently. But what does the dog learn? Textbook wisdom has it, first of all, that the animal merely learns to show a Response upon the CS. While SR learning plays a role in CC (see, for examples, Martin and Levey, 1988) it is certainly not the only way by which CRs can be established. A second misconception is that the temporal contiguity of CS and US is always of central importance. It is not. That is to say, temporal contiguity is neither necessary, nor sufficient to produce CC; temporal contiguity is only part of the story. (For an admirable analysis of this issue, see Rescorla, 1988.)

Third, textbooks by which most behaviour therapists were trained imply that the nature of CS and US and their intrinsic relation is a matter of indifference. The CS may be a bell, but a buzzer or a light would have done just as well; the US may be meat but other USs would result in an identical pattern of CC. This idea of stimulus indifference is not consistent with experimental evidence (Seligman, 1970). Lastly, responses amenable
to CC have for long been seen as reactions mediated exclusively by the autonomous nervous system. Convincing evidence shows that non-ANS mediated responses obey the laws of CC (Wolpe, 1974; Davies, 1987).

Consideration of why these notions are misconceptions (and, indeed, of the phenomena themselves) is more than a matter of mere academic importance. Behaviour therapists try to reconstruct the history of clients’ complaints as a learning sequence and to ameliorate problems by providing helpful learning experiences. A proper understanding of what CC is and is not about is vital to the validity of behavioural analyses and to the efficacy of therapeutic endeavours. In the rest of this paper, these points are considered in more detail.

(1) Temporal contiguity and probabilistic contingency

The idea that CC results from a CS preceding a US has implications consistent with established findings. First, it follows that CC does not occur if the order of CS and US is reversed, so that the US precedes the CS. (If a tone (CS) precedes and predicts a later shock (US), subsequent presentations of the tone will elicit fear responses. When, however, the shock precedes the tone, later presentation of the tone alone is unlikely to result in fear behaviour. The temporal priority view likewise implies that little CC occurs if CS and US are simultaneously presented. The weakness of simultaneous conditioning has been well documented (e.g. Smith et al., 1969). In the “traditional” studies of CC, a series of CSs was presented with every CS being followed by a UCS (see Figure 1A). In such a sequence, conditioning typically occurs and this is in line with the commonly held view. Suppose, however, that we start to administer additional CSs while keeping the number of paired cs/ucs presentations constant (see Figure 1B and compare with Figure 1A).

Under such conditions, the establishment of CRs is seriously impaired; CRs become weaker to the degree that it becomes harder to predict the occurrence of the US from the presence of the CS. This suggests that it is not contiguity per se that is responsible (contiguity is the same in Figures 1A and 1B) but that it is probabilistic contingency that counts. A different way to manipulate probabilistic contingency while keeping contiguity constant is, of course, to administer additional USs during the conditioning procedure (see Figure 1C). In such a sequence, establishment of CRs is again impaired to the degree that it becomes hard to predict the US from the CS.

These observations suggest that CC involves learning to predict an event from an earlier cue. It is not mere contiguity that is vital, but the information that the CS carries concerning the occurrence of a future event. Classical
conditioning appears to result from active processing of information. Few studies have so persuasively demonstrated this as Kamin's (Kamin, 1969) studies on "blocking". In a typical blocking experiment, experimental subjects first learn that CS1 predicts the US. Control subjects are given no prior learning experience with CS1. Then both groups are confronted with a compound stimulus, comprised of CS1 together with a new cue: CS2. For both groups, this CS1/CS2 combination is then made predictive of the US. If subjects are subsequently tested for CRs to CS2, a remarkable phenomenon occurs. There are strong conditioning effects in the control group but weak CC effects in the experimental group. Notice that both groups had the same number of CS2-US ("contiguity") experiences. The difference between the groups, as far as the CR is concerned, is due to the difference in informational meaning that the CS2 acquired. To the experimental Ss, the CS2 did not aid in predicting the US (the CS1 was enough). The important general point is that temporal contiguity of CS and US is insufficient for CC. The organism seems not so much interested in what cues happen to precede significant events. It rather actively searches for cues that predict such events. Factors that reduce the conditional probability of the US given the CS (unpaired CSs or unpaired USs; see Figures 1B and 1C) interfere with learning.

Not only is contiguity insufficient, various studies demonstrated that it is not necessary either. This can best be illustrated with so called "inhibitory" conditioning experiments. Suppose a subject has much experience with a CS that is not predictive of a US. It will be harder to condition this CS to the US (Lubow, 1973). Schematically, matters are represented in Figure 1D.

With a "latent inhibition" procedure, the subject has difficulty in realizing that a cue that earlier predicted non-occurrence of the US has now become a US predictor. Without CS/US contiguity, learning took place: what was learned was that the CS is not predictive of the US, thus making later conditioning more difficult. Conditioning here takes the form of cue-related non-responding (inhibitory conditioning) rather than the better known examples of classically conditioned cue-responding ("excitatory" conditioning).

(2) What is learned is not (always) a Stimulus Response (SR) relationship, but may be a Stimulus Stimulus (SS) association

Findings given above suggest that CC may involve more than simply learning to produce the response that was earlier evoked by the US, but now occurs after the CS. "Insight" (by the subject) into the association between CS and US appears to be a prerequisite for CC to occur in the field of human autonomic conditioning. There is broad consensus that clear awareness
of the CS–US association is necessary for the acquisition of conditioned autonomic responding (e.g. conditioned skin conductance response; SCR).

A CS, say a light, may reliably predict the occurrence of the US (say an electric shock) and this US may reliably elicit an autonomic response (e.g. SCR). Yet as long as there is no conscious contingency learning (i.e. S-S learning) no acquisition of a conditioned SCR takes place (Dawson and Schell, 1987). This is not to say that a conditioned autonomic response, once acquired, remains at full strength only as long as the subject expects the CS to be followed by an US. In fact, research shows that during extinction, dissociations between conditioned responding and subjectively perceived CS–US relationships can occur (Furedy, Riley and Fredrikson, 1983). In many instances, the conditioned autonomic response survives although the subject no longer expects the US to emerge. A similar phenomenon is seen in the neuropsychological syndrome known as prosopagnosia. Here, the patient is unable to recognize faces of his own relatives. In spite of this, the patient reacts with a conditioned "arousal" response when pictures of relatives are presented to him (Bauer, 1984). Note, in passing, that if the crucial element in fear acquisition is the subjectively perceived CS–US relation, we might anticipate also "conditioning-like" responding if the subject misperceives this relation and thinks the CS is followed by a
US while in reality it is not. This maps into clinical phenomena and we will come back to this later.

(3) CC involves more than autonomic responses

An early (e.g. Skinner, 1938) and still widespread idea is that CC is restricted to autonomic responses. The idea needs to be abandoned. First, both animal and human research shows that skeletal responses, typically held to be under the control of operant conditioning, can be classical conditioned. Examples include pecking behaviour of pigeons (Williams and Williams, 1969) and eye-blinking and avoidance behaviour in humans (Malloy and Levis, 1988).

Evaluative conditioning represents another example of non-ANS CC (for some fascinating studies, see Bayens et al., 1988). When confronted with a cue, we tend to directly assess whether we like it or not. Moreover, when we see, say, a new face, we are able to verbalize if we feel it is pleasant, unpleasant or neutral. Interestingly, when pictures of neutral cues (e.g. houses) are reliably presented before pleasant (e.g. nudes of the preferred sex) or unpleasant (e.g. mutilated bodies) pictures a hedonic shift occurs. The pleasantness of the house stimulus moves in the direction of the pleasant or unpleasant cue with which the house had been associated. Note that here CC does not involve expectancy learning (this CS will be followed by a US) but conative learning. The house picture itself becomes (un)pleasant. For conative learning, subjective awareness of CS–US associations is not necessary and this state of affairs is different to human electrodermal responding where awareness is a prerequisite (see above). In this sense, evaluative conditioning represents the blind, unconscious learning that is often erroneously believed to characterize all CC. Conditioned hedonic reactions to a previously neutral cue are stable over time and resistant to extinction; if the house picture from our example is, after pairing with the mutilated corpse picture, presented many times alone, the house picture will continue to be associated with unpleasant-affect, without the subject necessarily knowing why.

This resistance to extinction makes evaluative conditioning especially interesting to health promoting activities or advertising (e.g. McSweeney and Bierley, 1984). In passing, it may be noted that many advertisers could profit from studying CC in more detail. With evaluative conditioning, just like in ordinary CC, the effects are by far the strongest if the CS precedes the US, instead of vice versa. Note that in an amazingly high proportion of TV commercials, the to-be-conditioned, neutral cue (the product) is displayed after the US (e.g. scenes from happy family life). This is not consistent with what is known about forming associations. Experimental
data are clear: for hedonic transfer to occur the neutral cue should occur first, the US second.

Recent work indicates that, apart from autonomic, skeletal, and evaluative responses, immunological responses are susceptible to classical conditioning. A beautiful demonstration is to be found in a study by Ader and Cohen (1982) in which mice suffering from auto-immune disease (lupus) were treated with the immunosuppressive agent cyclophosphamide (US), a drug that has therapeutic properties in case of lupus. Each mouse also received saccharine tastes (CS). In half of the mice, the saccharine was paired with the drug; in the other half the saccharine was unpaired. After the initial paired or unpaired trials of saccharine and drug, mice received only saccharine. Interestingly, mice from the paired group lived longer than mice from the unpaired sample. The CS–US pairings in the paired group apparently resulted in the CS (saccharine) acquiring immunosuppressive properties and thus the CS started to function in a way which served to potentiate drug effects. Later studies demonstrated that other indices of the immune response can be conditioned as well (natural killer cell release in mice, histamine response in guinea pigs, adjuvant arthritis in rats and hypersensitivity to tuberculin injections in humans (Turkan, 1989)).

As to relevance to behavioural medicine, Ader (1985) argues that rigorous investigation of drug/placebo ratios, ways of administration and frequency/duration of placebo/drug administrations will indicate how “placebos” can best be used in clinical practice. That is to say, not as ethically questionable placebo-administration to patients who believe that they are receiving a “true” drug, but rationally programmed so as to function as a pharmacologically active, partial substitute for the original drug.

(4) The nature of CS, US and CS/US belongingness is important

Finally, the assumed indifference of CS, US and their relationships needs to be questioned. Originally, CC theory assumes that, given certain pairings with a particular US, all types of USs are equally likely to come and elicit CRs. The assumption is often described as the “premise of equipotentiality” and was seriously challenged by the work of Garcia and associates directing attention to “biological boundaries of learning” (Garcia et al., 1973). A key finding was that rats readily associate tastes (CS) with induced nausea (US) but are slow to associate taste CSs with a shock US. Conversely visual or auditory CSs are more easily associated with shock than nausea (Garcia and Koeling, 1972). There appears to be a belongingness between CS and US modalities which facilitates acquisition of CRs. It has been speculated that the interaction between CS and US is a manifestation of a genetically based, evolutionary significant “preparedness” (Seligman, 1970) to associate
taste, for example, with nausea. Preferential learning in the case of taste/nausea is evidenced from the ease of acquisition (one-trial learning of taste aversion after nausea is common), from the long CS-US intervals (up to twelve hours) that still produce CC, and from a relative resistance to extinction. Just as in the case of evaluative conditioning (see above), it appears that the taste CS after pairing with a nausea US is not so much experienced as a signal that nausea is at hand but that the taste is experienced as intrinsically bad (Garcia et al., 1972).

The most influential application of this research to clinical problems is the preparedness theory of human fears and phobias. While the preparedness concept is appealing, the theory presents serious conceptual weaknesses; experimental work with humans (cf. Öhman, 1986) suffers from a lack of reproducability across paradigms and across research centres (McNally, 1987; Merckelbach, van den Hout and van der Molen, 1989). Somewhat paradoxically, the strongest support for the preparedness theory stems from work with rhesus monkeys (e.g. Cook and Mineka, 1989).

In summary, CC involves more than learning of autonomic responding and the nature and speed of CC may also depend on the biological importance of CS/US relations to the organism. Most important perhaps is that CC may not only involve SR learning, but also SS learning. Graphically, matters are represented in Figure 2.

In both SR learning and in SS learning, research raised new and important theoretical issues. For example, the traditional view of SR learning is that the CR Mimics the UR: if the UR is vasodilatation, the CR is also expected to be vasodilatation. This assumption appears to be untenable. Alcohol, for instance, produces vasodilatation. Here alcohol can be seen as the US and dilatation of blood vessels as the UR. If, however, a certain environmental cue is made predictive of alcohol, the CS that emerges is a

![Diagram](image)

**Figure 2.**
vasoconstriction (Newlin, 1986). Note that the CR is opposite in direction to the UR. The data on counterdirectionality in CC with drugs not only raise important theoretical points, but also have therapeutic implications. We will come back to this when discussing addictions.

In many instances, SS learning is a necessary part of CC. On the other hand, it may well be that subjects are perfectly aware of the CS–US relationship but fail to react with a CR (cf. latent inhibition; see Figure 1D). Something like the reverse is also true. Sometimes subjects react with conditioned-like responses because they perceive a CS–US correlation that in fact does not exist. One might think of the obsessive compulsive patient reacting to contamination as if it were predictive of catastrophe. In which circumstances is learning of real SS contingencies necessary and sufficient to produce CRs, and under what conditions is an “illusory correlation” responsible for “conditioned like” responding? Identification of these circumstances is a major task for future studies. Let us now turn to some clinical issues.

Anxiety

The idea that any CS that is repeatedly followed by an aversive event (US) becomes a phobic cue is so beautifully simple that, if only for this reason, it ought to be true. Alas, it is not. Two of the main problems with this idea are that: (i) people often experience trauma (US) without developing phobic fear of trauma-related cues. (ii) In many instances, phobias develop without a clear history of trauma. The shortcomings of the CC approach in this respect, however, should not be overestimated. Retrospective studies by Öst and Hugdahl (1981) suggest that in the majority of phobias, conditioning experiences play a critical role. Nevertheless CC does not seem to be responsible for phobic fears in all cases.

Behavioural techniques (especially in vivo exposure) are effective in the treatment of phobias. One reaction to this state of affairs may be to retreat to a pragmatic stance, to abandon looking for the psychological history of the phobia and to concentrate on “Eliciting Stimuli” and “Elicited Responses” (Marks, 1978). Indeed, an overview of fear and avoidance eliciting cues is typically a sound basis for conducting exposure therapy (Emmelkamp, 1982; Marks, 1987). Such pragmatism is theoretically (and, ultimately, practically) unsatisfactory; given that exposure produces improvement rather than cure and that the majority but not all patients benefit, the continued investigation of learning approaches may ultimately be clinically worthwhile. But even if one chooses to take this pragmatic point of view, much can be learned from classical conditioning perspectives. Animal studies concerned with the parameters affecting fear extinction have
clearly shown which variables enhance extinction (and what is assumed to be its human analogue, in vivo exposure). Thus, long duration of the phobic stimulus, distraction strategies, counter conditioning and social facilitation might all maximize the beneficial effect of in vivo exposure (see review by Thyer, Baum and Reid, 1988).

Perhaps the most comprehensive elaboration of how the S-S theory of CC can deal with trauma without phobia and phobia without trauma has been published by Davey (1987; 1989a). Central to his S-S version of CC is the observation that the strength of a fear-CR to a CS is not only determined by the degree to which the CS predicts an aversive US. CRs can decrease or increase dramatically as the subject “revalues” the meaning of the US. Schematically, a parsimonious representation of what happens during Pavlovian conditioning is as follows:

\[
\text{CS} \rightarrow \text{Cognitive representation of the US} \rightarrow \text{Evaluation of the US} \rightarrow \text{CR}
\]

How does this model of CC explain a failure to develop phobias despite trauma? First, it predicts that if the subject has a lot of previously non-traumatic experience with the CS, it is less likely that the CS will be capable of eliciting a US representation. This corresponds “to the latent inhibition” phenomenon depicted in Figure 1D. As an example, subjects who have many non-aversive experiences with dentists are unlikely to develop a dental phobia even when, on one occasion, they are confronted with a painful dental treatment. In contrast, subjects who have little experience at the dentist are more likely to develop a dental phobia after experiencing painful treatment (Davey, 1989b). Indeed, a promising method for immunizing children against dental phobia is to give them ample non-aversive experience with dental treatment (e.g. “sham” treatments).

Second, the person may revalue the US separately from CS-US experiences. This “US revaluation” may be relevant to the finding that many phobics fail to recall conditioning experiences. To illustrate this point, consider the following procedure (described in detail by White and Davey, 1989). Subjects who are confronted with pairings of a neutral stimulus (e.g. slide of a triangle) and a very mild UCS (a tone), do, of course, not develop a conditioned skin conductance response to the neutral stimulus. If, however, these pairings are at some time followed by an isolated occurrence of a strong UCS (e.g. white noise), a conditioned SCR does occur. Apparently, the isolated UCS leads to a revaluation of the UCS representation in the subject’s mind and conditioning ensues. A clinical example might be the person who travels on a bus (CS). During his bus trip another passenger passes out (mild UCS). Some weeks later, a close relative has a heart
attack (strong UCS). This UCS “inflation” could give rise (through UCS revaluation) to a bus phobia. This scenario suggests a conditioning pathway to phobias in which first an association is learned between two relatively neutral events (for example, bus and a stranger passing out) although the learning is not, at that part, manifested in behaviour. When, later on, however, the latter event is revaluated as highly negative, the former cue may become a phobic stimulus.

Clinically, the implications are not confined to a potentially better understanding of etiology but expand to therapy. Effects of “performance based” interventions like in vivo exposure are expected to be successful because and to the degree that they break down the perceived probabilistic CS (e.g. bus) and US (e.g. heart attack) bond. Meanwhile, any other technique that reduces the perceived likelihood or perceived aversiveness of the CS (e.g. bus) predicting US (e.g. heart attack) will be expected to be effective; there is no a priori reason to believe techniques need to be performance based. One might aim to “deflate” the catastrophic meaning of the US or to reduce the perceived likelihood of the CS (e.g. bus or cardioacceleration in bus) being followed by a US (e.g. heart attack). Indeed, the parallel with cognitive approaches to anxiety disorders (cf. Hawton et al., 1989) is close.

Addictions

Experimental administration of adrenaline produces cardioacceleration and blood-sugar increases. Adrenaline can be regarded as US and increases in heart rate and blood sugar as URs. Suppose we administer a series of adrenaline injections, each preceded by a salient cue (CS). What would we expect to happen if, later on, only the cue (CS) is presented? Many psychologists firmly predict a CR comprising heart rate and blood sugar increases. This prediction is wrong. The opposite happens (see Siegel, 1983). The CS produces a decrease in heart rate and blood sugar. Thus, the CR is counterdirectional to the UR.

Counterdirectional CRs are not limited to adrenaline and alcohol as URs (see above.) The same effects have been observed with e.g. amphetamine, atropine, chlorpromazine, glucose, histamine, lithium, morphine, naloxone and more drugs (Macrea et al., 1987). Though counterdirectionality was occasionally reported by the first generation of CC researchers (cf. Subkow and Zilow, 1937), systematic research comes from more recent studies. The phenomenon of counterdirectionality between CR and UR has implications for our understanding of addictions. More specifically, drug-tolerance, drug overdose, craving and relapse can be elucidated when counterdirectional drug CRs are taken into account. The pertinent theoretical and empirical
work has in large part been done by Siegel and co-workers (for a good overview, see Siegel, 1983).

Tolerance refers to a reduced effect of a drug in cases where the drug is often taken. Drug-tolerance is, of course, characteristic of drug users. Siegel’s CC theory of addictions specifies how and under what circumstances tolerance occurs. Suppose drug intake is reliably predicted by a particular cue (CS, certain places, smells, needles), we would expect a CR (counterdirectional to the drug UR) to occur. The observed drug effect will be the net effect of the drug UR, minus the compensatory CR. Thus, tolerance is held to occur as a function of CRs. Only in cases where reliable CSs that accompany drug-intake are present, will tolerance develop. Moreover, when a “tolerant” subject is given the drug in the absence of these drug cues, tolerance disappears, as the drug effect is no longer compensated for by a counterdirectional CR. Abundant animal data show that tolerance is cue dependent. When rats receive a series of morphine injections either directly after an audiovisual cue or unrelated to the cue, a great deal of tolerance occurs in the cue-paired group, whilst hardly any tolerance occurs in the rats that are unable to predict drug intake (unpaired group).

A dramatic demonstration of the same principle (Siegel et al., 1982) is relevant to the understanding of lethal overdoses (OD), the fate of, annually, approx. 1% of heroin addicts. Naive rats that receive 15 mg/kg body weight of heroine die in 96% of the cases. Siegel et al. (1982) demonstrated that rats who were given 8 mg/kg body weight became drug tolerant during fifteen training sessions that took place in a salient cage. During a test phase, half of the heroin trained rats received 15 mg/kg dose in the same salient cage; the unfortunate other half received the mega-dose in an entirely new environment. In the same-cage-tested-rats, 32% died from the overdoses. In the other-cage-tested group, the body count amounted to 64% ODs. Apparently, the drug predicting cue (same cage) protected rats against the lethal effects of OD, presumably by giving rise to conditioned compensatory responses. A clinical parallel is the observation that OD survivors tend to report that a dose which, on a particular occasion was almost lethal, had been well tolerated on previous occasions, but that the near-lethal drug-intake took place in environments in which they were not used to taking drugs (Siegel, 1984).

Drug addicts report subjective craving for the drug. The conditioned compensatory response model described above holds that craving is a conditioned phenomenon and takes place when the addict confronts cues that predict drug intake. Craving, in other words, could be regarded as the subjective manifestation of compensatory CRs. For non-psychological
theories the situation-specific manifestations of craving (e.g. not bothering about cigarettes when watching the movies but very strong urges during the movie break) is hard to explain. Conditioning theory deals with this type of observation by postulating that the foyer of the cinema is a drug predicting cue, whereas the cinema-hall itself is an explicitly unpaired cue. From this view, guidelines for therapy can be derived. First, it follows that it is relatively simple to become and remain abstinent when the subject is not confronted with drug cues in specifically designed settings. Second, it implies that relapse is likely as soon as the abstinent addict is re-confronted with drug cues, e.g. when after discharge from the clinic the addict returns to surroundings in which drug intake previously took place. Third, and most importantly, the theory maintains that relapse prevention should be facilitated by elimination of the probabilistic relation between drug cue and drug intake. Thus, one would expect strong craving to occur when addicts are confronted with drug cues, but also that compensatory responding and craving would extinguish after repeated and prolonged exposure to the cues, provided of course that no drug is taken during such exposure. Concerning the possibilities for such extinction, animal data are encouraging; cue responsivity wanes when animals are exposed to cues in the absence of the drug (Siegel et al., 1980; Mannsfield and Cunningham, 1980). Very little clinical work has been reported. This is particularly surprising as addiction management is enormously important, the rationale is straightforward and so the techniques required should be similar to the ones that are familiar from the treatment of phobias and obsessive/compulsive disorders. For example, Rankin et al. (1983) gave a priming dose of alcohol (cue exposure) to five alcoholics and asked them to resist further alcohol intake. The control group (n=5) were asked merely to imagine having drunk a priming dose. Across sessions, there was an overall decrease in drinking urge and difficulty to resist, but the effects were stronger in the cue exposure group. Bearing in mind that the control group in essence received a somewhat weaker cue exposure, the results were encouraging. No clinical outcome measures or follow up data are provided. Laberg and Ellertsen (1987) similarly found that, in alcoholics given a priming alcohol dose, this initially elicited strong urges to drink and difficulty resisting these urges, but that both measures dropped within and between sessions. The same pattern was observed in cardiovascular and electrodermal measures. Unfortunately, here again no longer-term outcome measures were reported. As to cocaine and heroin, data consistently show greater physiological and subjective reactivity to drug related cues than to neutral cues. Cue exposure in heroine addicts reduced subjective desire, although the clinical impact of cue
exposure on heroine users was marginal in a study by McLellan et al. (1986).

While progress has been made, various questions require elucidation. Many drug CRs are counterdirectional to drug URs, but with some drugs and physiological parameters CR and UR are isodirectional (e.g. alcohol produces hypothermia and the classical conditioned response is hyperthermia). However, morphine produces hyperthermia while conditioned responses to morphine cues also include hyperthermia. Possibly, drugs that affect the afferent arm of the CNS produce isodirectional CRs while drugs that work on the efferent arm result in counterdirectional CRs (Eikelboom and Stewart, 1982). The issue of CR directionality can and should be empirically disentangled.

From a clinical stance, one might argue that therapeutic extinction procedures would not differ between isodirectional to counterdirectional CRs. What would count is the reduction of subjective desire, concomitant physiological distress experienced when cues are confronted but not followed by drug intake and, of course, longer term behaviour change and good therapeutic outcome. Plausibility aside, however, it is stressed that while Siegel's theory post facto nicely covers the relapse data, there is need for prospective studies to test whether cue dependent craving initiates relapse. Moreover, while as a matter of principle cue exposure seems a promising strategy, much is yet to be learned about spacing and duration of exposure session, interventions that stimulate generalization, the choice of cues to be employed in exposure therapy and many other parameters.

Food aversions and conditioned nausea

As mentioned earlier, food aversion learning in animals not only differs from ordinary CC because of the CS–US “belongingness” or CS–US interaction that takes place, but also by much more rapid acquisition, occurrence despite relatively long CS–US intervals, and slow extinction. Meanwhile, there is no reason to assume a qualitative difference between food aversion learning and other forms of CC (Logue, 1979). Taste aversion learning in humans seems to follow the same pattern found in laboratory animals. Usually, the pertinent food CS was present before illness (US), (forward conditioning) and food CSs are of a relatively unfamiliar type (latent inhibition in case of familiar foods). From the people that ate a given food item, became sick and developed a food aversion, most attribute the illness to the food. In one third of the cases, however, the illness can be traced to a different origin, e.g. influenza. Aversion typically concerns smell and taste rather than visual or tactile aspects (Logue et al., 1983). There are some data that alcoholics are less prone to generalize food aversions to related
food items and one might speculate as to whether this helps explain why occasional alcohol-produced sickness has so little impact on alcoholics (Logue et al., 1983). There is speculation that specific food aversions play a role in several syndromes that are (often) characterized by anorexia (cancer-anorexia; anorexia nervosa; depression). Though certainly thought provoking, these ideas (Bernstein and Borson, 1986) still remain untested hypotheses. In cases where food aversions are acquired through CC and are maintained by avoidance of the food, conditioning theory predicts extinction of aversion after repeated CS exposure. Indeed, De Silva (1988) showed that graded in vivo exposure can be highly effective in eliminating food aversions.

Although food aversions are quite common, however, they rarely present as clinical problems. This is dramatically different with the associated phenomenon of cue dependent nausea in cancer patients undergoing chemotherapy treatment. Estimates are that at least 30% of chemotherapy patients experience extreme nausea and vomiting when confronted with cues (CSs) reminding them of the chemotherapy sessions (Burish and Carey, 1986). The acquisition of conditioned nausea maps on to laboratory procedures used in food aversion studies. Acquisition can be quick and sometimes takes only one trial; long CS–US interval does not prevent acquisition, and the CSs most often conditioned are smells or tastes. Tastes experienced during the medication will later be experienced as intrinsically aversive, as experimentally demonstrated by Bernstein and Webster (1980). Experientially, there seem no difference between Unconditioned and Conditioned nausea, while the unpredictability of conditioned nausea may make the latter an extra burden for the patient. Though the conditioning history can easily be traced, the most plausible therapy, graded in vivo exposure, is rendered difficult if not impossible because CSs are regularly and strongly reinforced by the chemotherapy induced nausea (US). While little systematic research is available, it is noted that neo-Pavlovian perspectives provide rational guidelines for prevention and management of conditioned nausea (van den Bergh et al., 1989). It is essential that the experienced predictivity of the CS for the US should be as low as possible.

Note that familiar tastes and smells are less susceptible to the development of aversive associations, and that it takes, on average, six chemotherapy sessions before serious conditioned nausea is established. One strategy could capitalize on effects of latent inhibition; to reduce conditioned nausea in the chemotherapy room, one may have the actual chemotherapy sessions preceded by several inactive-infusions. Second, findings on "overshadowing" may be helpful. Overshadowing refers to the phenomenon that if two neutral stimuli are simultaneously presented in a CC set-up, the CR will
typically only develop to one of the two CSs. Typically, subjects start reacting to the most salient one and in the case of aversion/nausea the most potent CSs will be gustatory or olfactory. Bernstein et al. (1982) showed that food eaten before the infusion can be protected from becoming a CS by having the infusion preceded by eating icecream with a highly unfamiliar and therefore salient taste. Likewise, the physical conditions in which the infusion is given should be as novel as possible. Unfamiliar smells in the chemotherapy room may have beneficial effects and casuistic evidence indicated that lemon flavour during the infusion reduced conditioned nausea and vomiting (Greene and Seime, 1987). These suggestions are direct extrapolations of laboratory procedures to possible clinical techniques. It remains to be seen how adequate such extrapolations are. The main purpose here is to demonstrate that the important clinical phenomenon of conditioned nausea in chemotherapeutically treated cancer patients can be understood in terms of CC and that this theoretical analysis can be applied to the development of treatment innovations.

Concluding remarks

In this paper, recent developments in neo-Pavlovian research have been outlined. New findings and concepts were related to clinical phenomena. It will not have escaped the reader that clinical implications were tentatively formulated; the authors believe that they are dealing with promises and potentials rather than with established facts. This state of affairs is curious. Compared to the 1960s, we now know much more about CC and it is striking how quickly early accomplishments were accepted and integrated in clinical practice and how slow more sophisticated modern insights have been applied in the clinic. Of course, the rising tide of cognitive psychology, and especially cognitive therapy, is involved here. Just as conditioning paradigms were a source of inspiration to early behaviour therapists, information processing paradigms nowadays provide a credible and fruitful paradigm for empirically oriented clinicians. Worries about neglect of newer insights from the CC field may be reduced by acknowledging the convergence between cognitive and behavioural conceptualizations (Turkann, 1989). It is likely that important discoveries made in one area will be picked up or be independently identified in the other field. Besides, essential to the behaviour therapy enterprise was not the adherence to any specific theory, but rather the attachment to scientifically based theories and techniques. Changes in view and practice in the light of new evidence, indeed, is the sort of manoeuvre one would anticipate in a profession that justifies its very existence by the application of science.

The next ten to fifteen years will show if and how the potentials from
the rather specialized and sometimes esoteric field of CC are realized. To some extent this will depend on popularization of CC applications and on training of students, and, in the final analysis, it will depend on the theoretical rigour of the work in CC.

The amendments made by neo-Pavlovian researchers described above boil down to an increased ability of CC theory to accommodate clinical phenomena: conditioning can take place with very long CS-US intervals; phobias can occur without CS-US contiguity and CS-US contiguity can occur without later phobias; CRs can be isodirectional or counterdirectional to URs, and so on. This increased capacity to explain post facto a host of phenomena (cf. psychoanalytic theory) can only be described as progress if it is paralleled by specific, a priori predictions concerning the conditions under which pertinent phenomena should or should not occur.

When it comes to the future relevance of CC theory to psychopathology, the main issue is whether the notion of “laws” of learning can be taken seriously. Laws should specify under what circumstances certain things should not take place. Clearly, traditional CC theory was too narrowly defined in these terms, and its limitations could easily be identified. This weakness, in a sense, was its strength. On the other hand, a great diversity of clinical phenomena (phobias with conditioning history; phobias without conditioning history) can now be interpreted in neo-Pavlovian terms. However, this strength may turn out to be a weakness.

Classical conditioning research has increased our insight as to how organisms learn from experience. The insights were gained in well defined and circumscribed laboratory studies that were typically not designed to answer clinical questions. The implication is that much of what’s going on in CC laboratories runs the risk of being of only limited relevance to clinical practitioners. However, the CC research tradition is highly empirical and if the paradigms created in the lab map on clinical reality (e.g. taste aversion and conditioned nausea in chemotherapy), clinicians are provided with a powerful heuristic for analyses and intervention. Clinicians may benefit from keeping abreast of the progress that is being made in fundamental research. On the other hand, CC researchers would do their clinical colleagues a great service if some of their research concentrated on specifying conditions under which learning processes can result in maladaptive behaviour.
References


