ATTENTION, NOT ANXIETY, INFLUENCES PAIN

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Summary—Four hypotheses about the influences of anxiety and attention on pain impact were tested in a critical experiment: (1) anxiety increases pain; (2) anxiety decreases pain; (3) attention to pain increases pain; (4) only the combination of anxiety and attention to pain increases pain (interaction hypothesis). In a 2 × 2 design, anxiety (low vs high) and attention (attention vs distraction from the pain) were experimentally manipulated. Subjects received 20 electrically produced painful stimuli. Subjective pain experiences, skin conductance responses and heart rate responses gave no support for a pain impact increasing effect of anxiety. The anxiety-attention interaction hypothesis did not receive any support either. There was some support, only from the heart rate responses, that anxiety reduces pain impact. The critical factor appeared to be attention. Attention to the pain stimulus was related to a stronger pain impact (indicated by all measures) and to less subjective habituation, compared to distraction.

INTRODUCTION

It is still a controversial issue whether or not anxiety increases pain. Clinicians often seem to believe that anxiety intensifies pain (e.g. Chapman & Turner, 1986). This notion can also be found in theoretical views. For example, Melzack's (1973) model on the psychological influences on pain states that anxiety increases pain. In the dual-process theory of habituation (Groves & Thompson, 1970) it is stated that arousal increases the responses to a stimulus. Although the concept of arousal is not clearly defined in the dual-process theory, anxiety could be hypothesized to increase the state of arousal, thereby intensifying pain. On the other hand, it has also been proposed that anxiety decreases pain. In a influential article, Bolles and Fanselow (1980) reasoned that fear and pain are mutually exclusive states. Fear is assumed to have priority, and therefore to reduce pain and activate the organism (flight-flight response), whereas pain is assumed to deactivate the organism, promoting healing. A possible mechanism of the pain reducing effects of anxiety may be endogenous opioid release. There are, indeed, animal studies (reviewed by Bolles & Fanselow, 1980) and anecdotal reports of severely wounded anxious human Ss who appear to feel no pain (e.g. Beecher, 1956), which seems to support the idea that anxiety has a pain-reducing effect.

As for physiological processes, both views can be defended. Chapman and Turner (1986) argued that anxiety increases sympathetic activity and the release of epinephrine at the sympathetic terminals, which may sensitize or directly activate nociceptors. These authors have also suggested that increased muscle tension, which often accompanies anxiety, may cause additional pain, especially when muscles near the site of the source of the pain (e.g. the wound) are involved. On the other hand, there are physiological processes accompanying anxiety that may reduce pain, such as increased endogenous opioid release (Thyer & Matthews, 1986). Moreover, it is far from settled that reducing anxiety and physiological arousal, by relaxation or biofeedback for instance, directly causes pain reduction (Arntz & Schmidt, 1989). Increasing physiological arousal may be as effective as decreasing arousal, as long as the S. believes in the effectiveness of the response (Holroyd, Penzien, Hussey, Tobin, Rogers, Holm, Mancille, Hall & Chila 1984; Bush, Ditto & Feuerstein, 1985).

Two types of studies have sought to illuminate the relationship between anxiety and pain in humans: correlational and experimental studies. As for the first type of studies, several studies have found that state anxiety and (experimentally caused or internal spontaneous) pain are correlated (Kleinnekht & Bernstein, 1978; Dougher, 1979; Weisenberg, Wolf, Mitwocx, Mikulincer & Aviram, 1985; Ahles, Cassens & Stalling, 1987; Malow, West & Sutker, 1987, 1989). However, many studies did not find significant relationships (Arntz, van Eck & Heijmans, 1990; Bowers, 1968; Chaves & Brown, 1987; Klepac, McDonald, Hange & Dowling, 1980), or even negative correlations (Kent, 1984). Moreover, a positive correlation should not be confused with a causal...
effect of anxiety on pain: pain can, of course, be accompanied by anxiety and physiological arousal, but this leaves any causal direction open. Pain may as well lead to anxiety, or a third variable might be involved. Similarly, increased pain responses, as found in high trait anxious Ss (e.g. Dougher, 1979) may be caused by a third variable, and not by anxiety itself. To clarify the issue of causality, studies are needed in which anxiety is experimentally manipulated.

To the present authors' knowledge only six studies have investigated the effect of experimentally induced anxiety on pain. Unfortunately in two of these (Haslam, 1966; Bobey & Davidson, 1970) the effectiveness of the anxiety induction was not assessed. Three studies did find (sometimes mixed) evidence for a pain sensitivity-increasing effect of anxiety (Haslam, 1966; Dougher, Goldstein & Leight, 1987; Cornwall & Donderi, 1988), one failed to demonstrate any clear effect (Weisenberg, Aviram, Wolf & Raphaeli, 1984), and the other two found indications for the opposite effect: anxiety decreased pain sensitivity (Bobey & Davidson, 1970; Malow, 1981). Interestingly, in the Dougher et al. study only pain-related anxiety had pain sensitivity-increasing effects. Anxiety that was not related to pain did not lead to lower pain thresholds and lower pain tolerance levels. The Weisenberg et al. study also reports some evidence for this influence of focus of anxiety. However, in the study of Cornwall and Donderi, pain-irrelevant anxiety had also a pain impact increasing effect. The main differences with anxiety manipulations in other studies is that Ss in this condition were not confronted with an anxiety object during pain stimulation (the anxiety was induced by telling the Ss that they would get a stressful interview after the pain test). Thus, the effect of anxiety on pain might be related to the presence vs absence of an anxiety evoking stimulus during pain stimulation.

In summary, empirical consensus on the influence of anxiety on pain is lacking. One reason for this might be that a third, hitherto overlooked, factor modulates the relationship between anxiety and pain. One obvious candidate in this respect is attention. In most studies possible attentional effects were not controlled for. There is evidence that attention towards pain increases the pain experience, whereas distraction reduces it (Arntz & Schmidt, 1989; Kanfer & Goldfoot, 1966; Grimm & Kanfer, 1976; Worthington, 1978; Beers & Karoly, 1979; Tan, 1982; Turk, Meichenbaum & Genest, 1983). Therefore, it seems evident that if the focus of fear/anxiety is directed away from the pain, the pain experience is reduced; and conversely, if the pain itself is focused upon because of pain-related fear/anxiety, the pain experience will be stronger. The influence of such an attention factor might explain why some studies found a positive anxiety-pain relationship, whereas other studies found the opposite to be true. Thus, in the empirical studies on the influence of anxiety on pain, anxiety may have been confounded with attention. Therefore, it is worthwhile to re-examine the influence of anxiety on pain, controlling for the effects of attention.

The present study was designed as a critical experiment of several hypotheses that can be put forward with regard to the influence of anxiety and attention on pain. A 2 × 2 design was employed, the first factor being anxiety (low vs high), and the second being attention (towards pain attention vs towards an external object (distraction)). In the low anxiety/attention condition, Ss were given non-threatening information about the stimulus and were asked to concentrate on the pain stimulus as much as possible. In the high anxiety/attention condition, Ss were made anxious for the stimulus and were instructed to concentrate on the stimulus. In the low anxiety/distraction condition, Ss were distracted and were instructed to disregard the stimulus as much as possible. In the high anxiety/distraction condition, Ss participated in an exposure in vivo procedure with a phobic object (spider) and were instructed to disregard the pain stimulus as much as possible. A similar experiment has been done by Weisenberg et al. (1984), but this study suffers from some methodological inadequacies, such as too small cell sizes, unclear tests of the hypothesis, and failure to demonstrate that Ss in the pain-attention conditions attended more to the pain than Ss in the pain-distraction conditions. Moreover, this study failed to demonstrate clear effects of attention, possibly due to the weak manipulation of attention.

Four hypotheses were tested:

(1) the hypothesis that anxiety, irrespective of the focus of attention, increases pain impact,
(2) the Bolles–Fanselow hypothesis, which would be supported if, irrespective of the focus of attention, anxiety would decrease pain impact,
(3) the hypothesis that attention, irrespective of anxiety, is related to pain impact, and
(4) the possibility of an interaction was also considered. When pain impact is larger only when the S is anxious and attention is directed towards the pain, this view would be supported.

Two types of pain measures were employed: subjective pain experience and physiological responses to pain. In many studies, threshold and tolerance/endurance levels are measured, which have a clear behavioural component (especially tolerance and endurance, which are related to escape from the pain stimulation). It should be noted, however, that the correlation between subjective and physiological pain impact responses on the one hand, and pain behaviour on the other hand, is generally low. In the present context, the influence of anxiety and attention on pain impact seemed to be most relevant.

METHOD

Subjects

Because of the anxiety manipulation used in the high anxiety/distraction condition (exposure to spiders), it was decided to use spider phobics as Ss not only in this condition, but, to secure comparability, in all four conditions. They were recruited by means of announcements at publicity boards in several university buildings, in the local hospital, and in the local library. Before starting a session, procedural information was given and Ss signed statements of informed consent. Ss with pain complaints or with serious cardiac trouble were excluded, as were pregnant Ss. A small remuneration was given for participating. There were 52 women and 3 men in the final sample (see below). Mean age was 26 yr (range 17–56). The mean score on the Spider Phobia Questionnaire (SPQ, Klorman, Weerts, Hastings, Melamed & Lang, 1974) was 16.3, which indicates that this sample was definitively phobic when American and Swedish norms are considered (Klorman et al., 1974; Fredrikson, 1983).

Materials

Anxiety as experienced during the shock and experienced pain were measured by means of 10 cm. Visual Analogue Scales (VASs). Fear of spiders was assessed by means of the Spider Phobia Questionnaire (SPQ, Klorman et al., 1974; Fredrikson, 1983). After the last trial, Ss rated degree of attention given to the pain during stimulation on a 10 cm VAS. An open question requested to make a note of the main focus of attention during the experiment. In the low anxiety/distraction condition, Ss were given a video tape as distractor. The Ss could choose between a nature movie (without spiders and insects), an episode of L. A. Law, and a performance by a Dutch cabaret artist.

Apparatus and physiological recording

Apparatus was placed in an adjacent room. Skin Conductance Responses (SCRs) and Skin Conductance Level (SCL) were measured by a Beckman Skin Conductance Coupler (type 9844), using the method of constant voltage (0.5 V). The coupler allowed for a maximum sensitivity of 0.05 µSiemens. Electrodes were filled with isotonic paste based on Unibase (as recommended by Fowles, Christie, Edelberg, Grings, Lykken & Venables, 1981) and were attached to the medial phalanges of the second and third fingers of the non-dominant hand. The highest deflection within the 10 sec stimulation period and the 10 sec post-stimulus period was measured. SCRs were square root transformed (cf. Levey, 1980) and range corrected (Lykken, 1972).

Heart rate was measured by a Beckman Voltage/Pressure/Volume Coupler (type 9853A). Heart rate was measured via electrodes placed below the sternum and at the left edge of the chest. The signal was first filtered through a low-pass filter (set at 40 Hz), in order to reduce noise due to the shock, before being led to a microprocessor for heartbeat detection. Interbeat intervals were transformed to heartbeats (per min) on a second by second basis, employing weights corresponding to the amount of the second occupied by the interbeat interval (Graham, 1978).

The pain stimulation was produced by means of a Siemens Neuroton 627, passed for use with patients. The painful shock was delivered at the ankle of the S via a concentric shock electrode (Tursky, 1974). The stimulation was raised during 2 sec to the preselected level, remaining at that level for 6 sec, and was returned to zero during 2 sec. This stimulation produces a painful, stinging sensation, which is unlike electric shocks as known from barbed wire, electric mains, etc. The intertrial intervals varied randomly from 21 to 59 sec (mean 40 sec).
A microcomputer (PDP Minc-11) controlled the pain stimulation, intertrial intervals, and registration.

Procedure

Ss were randomly allocated to one of the four conditions. After obtaining informed consent, the electrodes were fastened. In case of the high anxiety/distraction condition (see below), a hierarchy of spider anxiety-related items was constructed. Next, shock level was established by increasing the amperage from zero up in steps of 0.2 mA, until the S indicated that the shock was painful and rated it approx. 60 on a 0–100 VAS. This level was kept constant over trials. Following the selection of the shock level, Ss were asked to relax for 5 min, at the end of which a 15-sec baseline measurement was done. After this measurement, the second author entered the room and announced that the series would start (without mentioning the number of trials or the duration of the series), and instructed the S to fill in the VASs after each pain stimulation. A loose-leaf file contained VASs for more than 50 trials.

In the low anxiety/attention condition, Ss were instructed to concentrate as much as possible on the pain stimulation, and to try and find descriptions of the local sensations evoked by it. Ss were instructed that they would be interviewed after the series about their experiences. It was also made clear to the S that the stimulus was entirely safe, produced by medical apparatus used by neurologists and physiotherapists. After these instructions, the experimenter left the room and the S started with the task.

In the low anxiety/distraction condition, Ss were instructed to concentrate as much as possible on the video, and try not to let themselves be distracted by the pain stimulus. The Ss were told that they already were familiar with the stimulus, suggesting that it was unnecessary to give any attention to it. As in the other low anxiety condition, it was attempted to make clear to the S that the pain stimulus was safe, being produced by medical apparatus, etc. After these instructions, the experimenter left the room and the S started with the task.

Pilot studies on different ways to induce anxiety for the pain stimulus indicated that none of them was reliably effective for all Ss. Consequently, the two most effective methods were chosen (described below as ‘first version’ and ‘second version’ of the high anxiety/attention condition) and both were run. More Ss were run than in the other conditions, because it was anticipated that we would fail to induce anxiety in approximately half of the Ss. Note that anxiety is not a dependent measure in the present experiment, but a necessary condition for the high anxiety conditions.

In the first version of the high anxiety/attention condition, Ss (n = 13) were given exactly the same information and instructions as in the low anxiety/attention condition. Via the intercom, the start of the series was announced. The intercom was not switched off, and several personal remarks about the S were made by the experimenters in order to enhance credibility of the manipulation. It was intended to suggest that the experimenters had forgotten to switch off the intercom. After these personal remarks, a tape was played via loudspeakers in the control room. The S could hear the conversation on the tape via the intercom. The conversation contained various non-verbal alarming topics, such as technical troubles with the shocker; disturbances of the heart rate caused by the shocker and wrong connection of the apparatus; troubles with the capacitor of the shocker, which might discharge itself on the S, leading a technician, who dropped in, to insist on immediate discontinuation of the experiment; the application of the wrong type of paste in the shock electrode, possibly leading the shock to burn the skin, etc.

In the second high anxiety/attention version, Ss (n = 14) were given the same attention instruction as in the low anxiety/attention condition. After baseline measurement, the second author told the S that some shocks might become suddenly very strong at the moment that the standard shock would decrease to null. To increase credibility and anxiety, an example of such a shock was given. The S was told that a random generator in the computer would decide whether this would occur, that every shock could suddenly become stronger, and that if there would be several shocks without a sudden increase, this would not mean that this would decrease the possibility of a sudden increase (the analogy of throwing a dice was made).

In the high anxiety/distraction condition, the second author started the exposure after having given the instructions about the VASs. There were two or three types of spiders available in order to control the level of exposure. It was attempted to dose the exposure at a subjective level of
anxiety of approx. 50 on the 0–100 VAS. Ss were instructed to attend to the spider, and to try
not to let themselves be distracted by the pain stimulus.

Following the series of 20 pain stimuli, Ss in all four conditions rated degree of attention paid
to the stimulus during stimulation, and indicated main topics of attention during the series. After
carefully debriefing the S, a 15 sec baseline measurement was obtained. Finally, the S filled out
the SPQ. Electrodes were then removed, the S was paid and thanked for participating.

Data reduction and analysis

Inspection of the heart rate patterns in the four conditions revealed that a HR acceleration took
place after shock offset. Heart rate responses of each trial were therefore computed as the difference
between the mean HR during 9 sec after shock and the mean HR during 9 sec before the shock.
Subjective pain ratings, SCRs and HRRs were averaged in blocks of 4 trials, and subsequently
analyzed by means of a MANOVA trend analysis. Two trends were inspected: the mean trend
(reflecting the average response) and the linear trend (as a measure of habituation). In case of the
SCRs, the linear trend with the log of the trial number was used to test for differences in exponential
decrease. Two covariates were employed: pretest subjective pain as indicated on the VAS and
objective pain level (in mA). In case of SCRs, SCL served as additional covariate. The covariates
were deleted if their contribution was NS (P > 0.10). The SPSSX MANOVA output yields t-tests,
which allow for directional interpretation (these t-tests of course result in the same levels of
significance as F-tests when interpreted two-tailed). The anxiety effect was tested two-tailed,
because two opposite hypotheses were formulated (anxiety increases pain; anxiety decreases pain).
The attention effect was tested one-tailed, because a directional hypothesis was formulated for the
effect of attention.

RESULTS

Manipulation check and selection of Ss

Since the anxiety induction in the high anxiety/attention condition had rather different effects
on the various Ss, the median of the anxiety ratings over all Ss was used as a criterion for S
selection in this condition. Ss who had a mean anxiety rating that was smaller than the median
(16.35 on the 0–100 VAS) were excluded from the high anxiety condition (when this criterion is
used, none of the Ss who participated in the spider exposure has to be excluded). Similarly,
distraction as measured by a post-test VAS appeared to have been very modest in some Ss in the
low anxiety/distraction condition. Therefore, the median of this VAS over all Ss (62.50) was used
as a criterion for S selection. Of the 72 original Ss, 17 were excluded, 5 from the low
anxiety/distraction condition because they attended too much to the pain stimulus and 12 from
the high anxiety/attention condition because of failure to induce anxiety in them. Table 1 displays
the final Ns.

An ANOVA on the post-test attention ratings (Table 1) revealed that Ss in the distraction
conditions rated a smaller amount of attention paid to the pain stimulation than Ss in the attention
conditions (t = 10.94, P < 10^{-2}). The anxiety main effect was NS (t = 0.66, P = 0.51), as was the
anxiety × attention interaction (t = −0.10, P = 0.92). At the open question, all Ss in the
distraction conditions named topics not related to the pain stimulus, whereas all but one Ss in the
attention conditions named stimulus-related topics.

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<tr>
<th>Table 1. Mean attention and anxiety ratings in the four conditions</th>
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<td><strong>Condition</strong></td>
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<td>Low anxiety/distraction</td>
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Attention to pain was assessed by means of a posttest VAS
(0–100), anxiety by means of VASs (0–100) filled in after
each trial (the averaged score is presented here).

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<th>Table 2. Trend analysis on subjective pain ratings</th>
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The anxiety effect was tested two-tailed, the attention effect one-tailed.
An ANOVA on the averaged anxiety ratings (Table 1) revealed a significant effect of anxiety induction ($t = 8.13, P < 10^{-2}$). The main effect of attention vs distraction was NS ($t = 1.06, P = 0.29$), as was the interaction ($t = 1.91, P = 0.06$).

**Subjective pain ratings**

A MANOVA trend analysis revealed that there was a significant attention effect on the mean pain rating as well as on the linear trend (Table 2). Distraction was related to lower pain ratings and to habituation (Fig. 1), whereas attention was related to higher pain ratings and absence of habituation. The anxiety effect was NS, as was the anxiety × attention interaction (Table 2). Thus, these results support the hypothesis that only attention is related to the experience of pain, whereas anxiety has no effect.

**Skin conductance**

An ANOVA on the SCL during the series, with pretest baseline SCL as covariate, revealed a significant main effect of anxiety: in both high anxiety conditions, SCL was significantly higher than in the low anxiety conditions ($t = 2.21, P = 0.03$). The attention main effect was NS ($t = -0.78, P = 0.44$), as was the anxiety × attention interaction ($t = 1.15, P = 0.26$). The anxiety effect should, of course, be attributed to the anxiety manipulations.

A MANOVA trend analysis on the SCR revealed a significant attention effect on the mean SCR: attention to pain was related to stronger SCRs ($t = 2.34; P = 0.01$) than distraction from pain. The anxiety and interaction effects were NS (Fig. 2, Table 3). There were no significant differences with regard to SCR habituation. To summarize, there was clear evidence that attention was related to stronger SCRs, whereas anxiety appeared to have no effect at all on SCRs to pain.*

**Heart rate**

Inspection of the heart rate patterns of the four groups showed that acceleration took place after the shock. According to Turpin (1986), this long latency acceleration reflects a defensive response, and must be distinguished from early acceleration, reflecting a startle response. A MANOVA trend analysis on the heart rate accelerations revealed marginally significant effects of anxiety (anxious Ss showed on the average less HR acceleration than non anxious Ss, Fig. 3 and Table 4) and attention (attention to pain was related to stronger HR acceleration than distraction). The interaction was NS. There were no significant anxiety or attention effects on HRR habituation (Table 4).

The anxiety effect on HRRs could be attributed to increased pretrial HR in the high anxiety conditions, caused by the anxiety manipulations. If the difference between baseline HR and mean

*The first SCR to the pain stimulus was also analyzed. Results were similar to those of the maximum deflection: attention was related to stronger SCRs ($t = 2.22, P = 0.015$), anxiety did not relate to SCRs ($t = -0.91, P = 0.34$), and the interaction effect was also NS ($t = 0.21, P = 0.64$).
Table 3. Trend analysis on SCR

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<th>Experimental factor</th>
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The anxiety effect was tested two-tailed; the attention effect one-tailed.

retrial HR is employed as a covariate, the anxiety effect shrinks to a NS level \((t = -1.28, P = 0.21)\), whereas the attention effect remains essentially the same \((t = 1.31, P = 0.10)\). Thus, the anxiety effect on HRRs may have been caused by the effect of the anxiety manipulation on tonic HR.

Are the results dependent on the selection of Ss?

It could be argued that the observed distraction effects result from selecting Ss with low attention scores in the low anxiety/distraction condition: the high attention scores of the removed Ss may have been caused by a strong pain experience. The remaining Ss, likewise, may have had low attention scores because of low pain. Thus, instead of distraction causing lower pain impact, lower pain impact may have led to distraction. To control for this possible artefact, all analyses were repeated without selecting Ss on the basis of their attention VAS scores \((n = 60)\). However, the results appeared to be very robust: distraction from pain was still related to a smaller experience of pain \((t = 2.25, P = 0.01)\), to faster subjective habituation \((t = 2.03, P = 0.02)\), to smaller SRRs \((t = 2.03; P = 0.02)\) and (marginally significant) to smaller HRRs \((t = 1.60, P = 0.06)\). The anxiety and interaction effects also remained the same. Thus, the effect of attention vs distraction appeared to be no artefact of the S selection procedure, but to depend on the availability of a distractor. Since only anxious Ss were selected in the high conditions, it is unlikely that the observed lack of effect of anxiety can be attributed to S selection. The analyses over the whole sample \((n = 72)\) indeed revealed that distraction from pain was related to smaller subjective pain responses \((t = -1.94; P = 0.028)\), faster habituation \((t = -1.47; P = 0.072)\), and smaller SRRs \((t = -2.10, P = 0.02)\). The effects of anxiety were NS (mean subjective pain rating: \(t = -0.42, P = 0.68\); subjective pain habituation: \(t = -0.17, P = 0.87\); mean SCR: \(t = -0.01, P = 0.99\)), as were the interaction effects (mean subjective pain rating: \(t = 0.64, P = 0.53\); subjective pain habituation: \(t = 0.35, P = 0.73\); mean SCR: \(t = -0.49, P = 0.62\)).

The lack of effect of anxiety cannot be attributed to S selection either. The conclusion that anxiety has no influence on pain as derived from analyzing the whole sample can however be criticized because of failure to induce anxiety in many Ss in the high anxiety/attention condition. Therefore, the selection of Ss who were successfully made anxious seems to give a more valid base for testing the effects of anxiety.

DISCUSSION

Three unambiguous conclusions can be drawn from the data presented above. First, attention to pain is clearly related to stronger pain responses. Second, results do not support the hypothesis that anxiety is related to stronger pain responses. Third, the opposite theory, that anxiety is related to less responding to pain, did not receive much support either. Only heart rate responses appeared to be slightly less in the anxiety conditions. This effect may, however, have been caused by the influence of the anxiety manipulation on the tonic HR level (law of initial values). It seems difficult to attribute the lack of influence of anxiety on pain to too low levels of anxiety: especially in the high anxiety/distraction conditions, the anxiety was relatively high and ‘real’ because it was induced by means of exposure to a phobic object. There were, however, no indications whatsoever that pain responses were more strongly influenced in this condition.

*The heart rate data of the Ss who failed to get anxious were not further processed. Because of the relatively weak effects of the experimental factors on the HRRs, this seemed to be not very relevant. An additional check revealed that the removed Ss of the high anxiety/attention condition did not differ from the selected Ss of this condition with respect to mean experienced pain \((F(1,19) = 1.69, \text{ NS})\) and SRRs \((F(1,25) < 1.00)\). Therefore, the selected high anxiety/attention Ss were not anxious because their pain responses were stronger.
Attention appears to be the critical factor: subjective pain, SCRs and (marginally significant) HRRs were less when the S directed his/her attention away from the pain stimulation. The importance of attention is perhaps most clearly shown by the subjective pain ratings: when attention is focused upon the pain, there appears to be virtually no subjective habituation at all (Fig. 1). Distraction, on the other hand, appears to be related to ongoing habituation. The present data suggest that it might be fruitful and clinically relevant to examine the effect of distraction on long term habituation of pain responses.

The present study opens the possibility that the contradicting findings found in previous research on the influence of anxiety on pain results from the operation of the attention as a third factor. For instance, in the Malow (1981) study, experimentally induced anxiety was found to be related to lower pain reports and reduced pain discrimination ability. These effects may have been caused by the attention diverting effect of the anxiety induction manipulation, which was accomplished by warning the S with a red light on a shock apparatus: the red light may have diverted the S's attention from the pain, and therefore reduced pain report and pain discrimination ability. Similarly, the anxiety induction in the Bobey and Davidson (1970) study (letting the Ss listen to a tape with cries, screams and moans) may have diverted the S's attention away from the pain and therefore caused higher pain tolerance levels. With respect to correlational studies which measured state or trait anxiety before the pain application, the present findings suggest that positive anxiety-pain correlations might have been caused either by response tendency (high anxious Ss showing a negative tendency in rating any stimulus), or to the different use of attention diverting strategies in high vs low anxious Ss. The hypothesis that anxious Ss attend more to pain, or find it more difficult to avert their attention from pain, as long as there is no other powerful distracting stimulus, might be worthwhile to investigate. A study by Cornwall and Donderi (1988) seems to give some support for this hypothesis: Ss who were made anxious for a stressful interview, to be conducted after the pain test, showed elevated heart rate and elevated pain ratings during stimulation, compared to a control group. However, the majority of measures (pain threshold, pain tolerance, EMG, facial grimaces, and pain rated after the pain stimulation) was NS different from the control group. Moreover, attention effects were not controlled for. Thus, a more direct test of the hypothesis that anxious Ss have more difficulties to distract from pain as long as there is no powerful distracting stimulus, would be welcome.

The lack of influence of anxiety does not mean that anxiety is never relevant with respect to clinical pain. When the S is anxious about the causes or the course of pain, he/she may pay a lot of attention to the pain, thereby increasing the pain. However, the present study indicates that in such instances the critical pain increasing factor is attention, rather than anxiety or anxiety-related arousal. Pain-related anxiety merely motivates the S to direct attention to the pain. Anxiety might

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<th>Table 4. Trend analysis on heart rate responses</th>
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<td>The anxiety effect was tested two-tailed, the attention effect one-tailed.</td>
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also produce pain via anxiety-induced muscle tension, which can produce muscle aches. Clearly, this mechanism was not addressed in the present study. However, muscle tension will play no part at all in many cases of pain.

It is interesting to note that the present findings seem to contradict Groves and Thompson’s dual-process theory (1970) of habituation. Groves and Thompson state that two processes play a part in habituation: firstly, a decremental one, taking place in the direct connection between stimulus receptor and response effector (causing habituation); and secondly, an incremental process (causing sensitization), which reflects the general ‘state’ of the organism. More specifically, Groves and Thompson state that arousal (‘excitation’, ‘activation’, etc.) is the key concept in this second process; high arousal is related to stronger responding (and can therefore cause dishabituation, sensitization and delayed habituation). The present study indicates, however, that anxiety induced arousal is not related to stronger subjective and autonomic responses to pain, nor to delayed habituation. Instead of anxiety or arousal, attention seems to be the important state variable. It should be noted, however, the Groves and Thompson’s work is based primarily on the study of motor responses, and therefore should not necessarily apply to autonomic or subjective responses. It might be interesting to investigate anxiety and attention effects on human motor responses to pain.

Although of considerable theoretical importance, the relationships between anxiety, attention and pain are not without practical implications. The present findings strongly indicate that strategies to distract attention from pain (as already widely practiced by clinicians e.g. the G. P. who distracts the child’s attention form a painful intervention by telling a nonsense story, asking the child to correct the story*) seem to be more indicated than techniques or medicines for anxiety reduction. Nevertheless, when anxiety is the motivating factor in attending to pain, anxiety reduction might be helpful. In any case, anxiety reduction seems not to be contraindicated, as the Bolles–Fanselow hypothesis leads one to believe. On the contrary, the present findings suggest that anxiety reduction does not lead to an increased pain experience, as long as attention is not focused more on the pain. Moreover, reduction of suffering due to anxiety might be a legitimate goal in itself, which will not necessarily be in conflict with the goal of reducing suffering due to pain. With regard to chronic pain, the present findings suggest that attention-diverting techniques might be a powerful ingredient of psychological treatment, because distraction immediately decreases the experienced pain, and seems to promote subjective habituation. In addition, the experience to be able to master the attention-attracting properties of pain might be very important for chronic pain patients and may serve to reduce feelings of helplessness and increase a sense of control over the pain and life in general.

REFERENCES


*See also: Beers and Karoly (1979); Grimm and Kanfer (1976); Kanfer and Goldfoot (1966); Miller and Grant (1979); Tan (1982); Turk et al. (1983) for spontaneous and instructed use of distraction as a pain coping strategy.


