HEART RATE AND BLOOD PRESSURE IN THE STUDY OF LABORATORY PAIN IN MAN UNDER NORMAL CONDITIONS AND AS INFLUENCED BY HYPNOSIS

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Professor Konorski’s interests ranged widely beyond the topics of classical and instrumental conditioning with which he was primarily identified. Although it was those topics that led to a long mutual understanding between him and the senior author, he welcomed a discussion of some of the earlier data from this research program in the Nencki Institute in Warsaw in 1970. His interest in anticipatory responses, hallucinations, and aphasia made the distortions within hypnosis relevant to his own thinking.

Pain continues to be a puzzling phenomenon (e.g., Melzack 1973). The subjective experience of pain in its many forms is too common to require comment, and the bodily concomitants of intense pain involve many neurophysiological systems. Although the search has been widely conducted, no satisfactory single indicator of felt pain in man has been found. Under special circumstances a selected indicator may correlate with the intensity of the noxious stimulus and with the subjectively reported pain, under other circumstances the same indicator may be responding to a nonpainful source of arousal. This chapter summarizes the conclusions reached from a number of studies conducted in our laboratory over a period of years bearing upon cardiovascular responses as concomitants of felt pain in the normal condition and in hypnosis. The results bear on the suitability of heart rate and blood pressure as indicators of pain in man, and upon the special problems of understanding the nature of hypnotic analgesia.
Experimental procedures and arrangements

The details of the several experiments can be found in the published sources, listed at the end of this paper. In this summary we shall avoid the tedious details of taped instructions, balanced orders of presentation, statistical tests, and the other evidences of care in experimentation, moving as rapidly as possible to those results that we consider firmly established or still in doubt.

The two procedures that we have used consistently in the study of laboratory pain with normal subjects, that is, with subjects whose pains we generated as well as relieved, were the cold pressor response and a tourniquet-exercise pain that we refer to as ischemic pain. The cold pressor response has a long history, the best introduction being that of Wolf and Hardy (1941). In the form in which we have used it, a hand and forearm have been placed in circulating ice water for not over 60 sec, with measures commonly recorded at 5 sec intervals. A common comparison point has been at 30 sec in the water, when pain is already near a tolerance level, and cardiovascular indicators are near their asymptotes. In one experiment other temperatures than 0°C were used (5°, 10°C) to provide validation for changes in pain with milder cold stress. The tourniquet pain has also had a considerable history. The form in which we use it was developed in Beecher's laboratory at Harvard (Smith et al. 1966). An arm is held up in the air to begin reducing the blood in it; the arm is then wrapped tightly with an elastic bandage to further reduce the blood, and a sphygmomanometer cuff is inflated above the elbow to 250 mm. Hg to cut off the circulation. The bandage is then removed, and the subject exercises the occluded muscle by squeezing a dynamometer at a 10 kg load, squeezing 20 times for 2 sec and releasing for 2 sec alternatively. The dynamometer is taken away, his arm rested on the arm of the chair, and a waiting period ensues in which pain mounts. The pain mounts more slowly than in the cold pressor test, and reaches a high level in a matter of minutes rather than of seconds. It becomes quite severe and annoying, and is reported to behave to placebo and morphine very much as post-surgical pain does.

We have found it quite satisfactory to obtain reports of felt pain on a numerical scale in which 0 represents no pain and 10 a pain so severe that the subject would wish to terminate it, although he is willing to report pains above 10 when stressed for a while longer. A scale open at the top is more satisfactory than one with a fixed ceiling. If, for example, 10 is defined as a maximum, then reports become 9.5, 9.7 and so on, producing a marked flexion in the rising curve of pain with time. With the open top, the rise is more uniform, approximating a straight line.
when plotted in log-log form, that is, the logarithm of reported pain against the logarithm of time, a power function. These pain reports are quite reliable on retest, and valid in the sense that they produce significant differences between pain at slightly different water temperatures, and are differentially reduced by hypnotic analgesia according to the measured hypnotizability of the subjects.

In the recording of the cardiovascular accompaniments of the painful stimulation we have relied on piezoelectric impedance plethysmographs attached to the fingers of the nonstressed hand. Heart rates have been counted directly from the polygraph records. To obtain a measure at any one time, the average rate has been computed for 5 sec preceding and 5 sec following this time. Systolic blood pressure was recorded from an occlusion cuff attached to the middle phalanx of the middle finger of the nonstressed hand. One impedance plethysmograph was attached to the tip of the finger, a second one below the cuff. The cuff was inflated and deflated by means of a servomechanism. A polygraph recorded the rising and falling pressure in the cuff, as well as heart beats, so that a measure of systolic blood pressure could be obtained when the circulation was shown to be cut off, both on the plethysmographic record and the cuff record. Because of the manner of operation, a measure of systolic blood pressure was obtained at approximately 5 or 6 sec intervals.

Four main conditions have been superimposed upon the exposure to these physical stresses. The first condition was normal waking, the usual nonhypnotic condition, with instructions to experience the pain without drifting into hypnosis, even though some relaxation was involved. The other conditions were: hypnotic nonanalgesia, in which the subject was hypnotized but the sensitivity of his arm to pain remained normal, testing the question whether the relaxation of hypnosis itself reduced the felt pain; hypnotic analgesia, in which the hypnotized subject received the suggestion that his arm would be insensitive to pain and other stimuli; and, in one experiment, hypnotically hallucinated pain, in which the arm, remaining on the cradle above the ice water, was hallucinated as having been dropped into the water and as having been painfully stimulated by it.

Hypnotic induction typically followed the procedures of the Stanford Hypnotic Susceptibility Scales, Form A (SHSS-A), although with highly susceptible and experienced subjects the induction was sometimes abbreviated. For them, a subjective report of hypnotic depth on a numerical scale sufficed to serve as a criterion of satisfactory hypnotic condition for the purposes of the experiment. SHSS-A provides a measure of hypnotic responsiveness on a scale of 0 to 12, without di-
rectly measuring response to analgesia suggestions; these score have been used in stratifying subjects according to measured hypnotic susceptibility to determine the relationship between hypnotic responsiveness and the effectiveness of analgesia suggestions.

After some preliminary training as necessary, the more formal sessions consisted of a relaxation period of a few minutes which in some cases included the hypnotic induction. This was followed by a specification of the conditions to follow in order to provide measures of anticipatory changes, especially at a time labelled as prestimulus when the warning was given that the stressful stimulation was about to begin. There was often a change in physiological responses between the relaxation measurements and prestimulus, so that responses subsequent to prestimulus and from the beginning of the experimentally imposed stress often were the preferred indicators of the changes owing to the physical stress.

**Cardiovascular concomitants of normal waking pain**

The heart rate rises with the mounting pain after the hand and forearm are submerged in circulating ice water. A maximum rate is reached in about 15 sec when the pain level has reached about 7 on the subjective scale, and may drop off slightly after that even though felt pain continues to rise to a mean level of around 12 at 40 sec. The amount of heart rate rise was found to be correlated with water temperature, hence a significantly greater rise in the coldest water, corresponding to the pain does, and the rather low (if significant) correlation with felt stress period (following the prestimulus level) was found to correlate 0.33 with the verbally reported pain, a significant correlation for the 23 subjects in that experiment. So far, heart rate would seem to be a reasonable indicator of felt pain, except for its reaching a maximum before the pain does, and the rather low (if significant) correlation with felt pain. Heart rate rises also in the ischemic pain experiments, but we do not have data from which to make a precise comparison with the amount of felt pain in a random sample. For more highly hypnotizable subjects, experiencing pain normally, the rise in heart rate within the stress period averaged much less than in the cold pressor test, in one experiment with eight subjects averaging only a rise of 5 BPM, even though the pain reached a scale report of 15.5, and no average rise at all in another experiment with eight subjects in which the pain level reached a mean of 13 on the pain scale. Hence the heart rate appears to be moderately satisfactory as a concomitant of pain in the cold pressor test, but not very satisfactory in the ischemic test.
The blood pressure measure, although initially showing some promise, turned out to be a less satisfactory measure than heart rate in the cold pressor test. The encouraging sign came from the significant difference between the blood pressure rise in 0°C water and water at warmer temperatures (5° and 10°C). However, the correlation with felt pain for those experiencing more pain and those experiencing less pain to a common stress was not significant, as it was with heart rate, the correlation turning out to be but 0.16, in the same direction to be sure, but too low to be of any significance. The correlation is disturbed by wide individual differences, because a fourth of the subjects, although experiencing considerable pain, showed either no rise in blood pressure or a trivial rise not exceeding 2 mm of Hg on our records.

The blood pressure measure appears to be more satisfactory than the heart rate measure in ischemia in the normal waking condition. Lenox (1970) reported a mean rise of 27.7 mm from the beginning of stress to its termination at a mean pain level of 15.5 on the subjective scale. This large blood pressure rise in waking is in part a consequence of the criteria used for what he called "maximum tolerable pain". He required that the subject show a blood pressure rise of at least 20 mm and a pain report of at least 10. When we repeated the experiment with less severe criteria, we found a corresponding total rise of 19.8 mm, with less severe ultimate pain that averaged 13 on the subjective scale. There is little doubt that blood pressure rises within the total period, but, as we shall note later, there are some unresolved questions about anticipatory responses.

If both heart rate and blood pressure were satisfactory indicators of pain, the two measures ought to be positively correlated when they are recorded simultaneously. This logical relationship is not found; that is, those who are in the upper half of changes in heart rate are not the same subjects as in the upper half of changes in blood pressure. In fact, in some of the experiments the correlation is negative, though not significantly so. This is not too surprising, in view of a number of experimental findings in which heart rate and blood pressure are differentially affected by experimental conditions (e.g., Schwartz 1972).

Because heart rate changes and blood pressure changes are uncorrelated, although each shows some relationship to painful physical stress, neither is alone a satisfactory indicator of felt pain.

**Concomitants of hypnotic suggestion**

In view of the uncertainties about heart rate and blood pressure as indicators of normal pain, it is not surprising that some of the results within hypnosis are also equivocal. In most cases the verbal reports of
pain are more orderly and dependable than the physiological measures that we have studied. Still, there are some generalizations that can be made with confidence.

The first of these is that hypnosis without analgesia suggestions does not alter to any appreciable extent either the verbally reported pain or the physiological concomitants, whether in the cold pressor test or the ischemic one. This bears on the common assertion of a relationship between anxiety and pain. If hypnotic relaxation were itself responsible for the reduction of anxiety, and secondarily the reduction of pain, then there should be a difference between the normal waking pain and the pain within 'hypnotic nonanalgesia. The failure to find this result is worth noting because of a false impression that sometimes results from a generalization from Shor's (1962) study. He reported in the following words: “When the incidental anxiety component of the total pain experience is high, physiological responses to pain are large. Hypnotic analgesia is a means of reducing these responses”. The point that is overlooked in this generalization derives from an examination of the actual data of his experiments, and some supplementary information in the original dissertation (Shor 1959). The two groups that he compared were one in which hypnotic analgesia suggestions were not successful in reducing pain, and another in which hypnotic analgesia suggestions entirely eliminated the pain. Yet the physiological responses were equally reduced in both groups. What this means is that general relaxation, in his experimental setting, was effective in reducing some physiological responses, but these responses were independent of the amount of felt pain reported. The felt pain was indeed reduced by hypnosis among the more susceptible in his dissertation; Shor reported a significant correlation (phi coefficient) of 0.46 between rated depth of trance achieved in the experimental situation and the adequacy of the analgesia, based on the responses of 23 subjects. Hence, while hypnotic procedures may indeed produce relaxation and reduce some physiological activation, even among the hypnotically unresponsive, it cannot thereby be asserted that felt pain has been reduced through such relaxation alone.

A number of investigators from other laboratories as well as ours have found what Shor reported, a correlation between the susceptibility to hypnosis and the amount by which pain is reduced through analgesia suggestions. The correlation tends to run about 0.50. A point at issue is whether or not, for susceptible subjects, a prior induction of hypnosis is necessary to obtain analgesia, or whether "waking suggestions" are equally effective. Evans and Paul (1970) found waking suggestion as effective as hypnotic suggestion, although the amount of pain reduction still correlated with measured susceptibility to hypnosis. With more
highly selected subjects, our experiments have shown that there is some additional pain reduction following hypnosis, a result that would not be expected from the design of the Evans and Paul experiment in which unselected subjects were used, so that many of them were not responsive to hypnotic induction in any case.

The amount by which pain is reduced is only probabilistically related to measured hypnotic susceptibility. For example, unselected subjects, inexperienced in hypnosis, may be tested in the cold pressor experiment in the normal waking condition and then following hypnotic induction with suggested analgesia. If the subjects are then classified according to their hypnotic susceptibilities into three groups (high, medium, and low) the results of Fig. 1 are found. While two-thirds of the high group re-

![Fig. 1. Reduction of pain through hypnotically suggested analgesia as related to susceptibility to hypnosis. The subjects were 54 university subjects with prior experience of hypnosis limited to the experience of the procedures for measuring hypnotic susceptibility.](image)

duced their pain by one-third or more, only one-sixth of the medium group were this successful, and one-eighth of the low group. The relative proportions were reversed for those unsuccessful in reducing pain. Even so, some of the highs failed to reduce pain, although more of the lows fell in this category.

It is of some interest to estimate the number in an unselected stu-
dent population who can eliminate pain entirely in laboratory experi-
ments of the kind reported. Our estimate, based on large samples of uni-
versity students, is that (at least without extended practice) the propor-
tion is about 5 in 100. For clinical uses of pain reduction, as in dentistry
or obstetrics, it is of course not necessary to eliminate pain entirely for
hypnotic pain reduction to be useful.

If the heart rate rise in the cold pressor response is a consequence
of pain, rather than a direct reflex action to the cold stimulation, it
should be reduced by hypnotic analgesia that clearly reduces felt pain.
The testimony of many prior experiments is that the heart rate increase
with pain remains unaltered as a consequence of hypnotic analgesia (for
cold pressor pain, Barber and Hahn 1962, Evans and Paul 1970; for re-
 sponses to electric shock, Shor 1962). Other physiological measures, such
as the GSR, have yielded comparable findings of response to shock (e.g.,
Shor 1962, Sutcliffe 1961). In view of Shor's finding that relaxation will
itself reduce physiological responses, it would be expected that careful
measurements made with a stress more severe than some of those pre-
viously used would show at least some effect of hypnotic analgesia, and
we have, in fact, found this to be the case. In three separate experiments
we have found slightly but significantly less rise in heart rate under
conditions of hypnotic analgesia than of waking, although the amount of
decrease does not appear to be correlated with the amount by which
subjective pain is reduced.

Hypnotic analgesia suggestions, whether or not they reduce pain,
produce a much more quiescent subject, as Shor also reported. The amount
of difference in heart rate between waking and analgesia, of the order
of 5 to 12 BPM, could be secondary to the amount of overt agitation in the
waking condition. The result is consistent enough to indicate that in the
cold pressor pain there is a slight change in heart reaction through hyp-
notic analgesia, and a comparable result has been reported in one of
the experiments with ischemic pain (Lenox 1970). It does not mean,
however, that the complete absence of subjectively felt pain means the
absence of any indication of stress, for some rise in heart rate was found
even for those able to eliminate felt pain entirely.

The corresponding changes were not found to the same extent for
blood pressure. In the experiment in which heart rate changes were re-
duced by hypnotic analgesia there was no difference whatever between
the blood pressure rise within stress for the waking and hypnotized sub-
jects. These negative findings have to be considered in the light of two
reports of positive findings, both obtained in our laboratory (Lenox 1970,
Sachs 1970). Lenox found essentially no rise of blood pressure within
analgesia in the ischemic pain experiment, after a substantial rise in wak-
ing, and Sachs found less rise in blood pressure in the cold pressor test when his subjects were analgesic, as compared to when they were feeling the pain. Both these studies used highly hypnotizable trained subjects.

In an attempt to replicate these findings, 8 highly hypnotizable subjects were selected for testing in both the ischemia and cold pressor tests. In the cold pressor test, the total blood pressure rise was slightly higher under conditions of hypnotic analgesia, as compared to waking. On further examination of the data, however, this rise was found to be greater during the anticipatory period in the hypnotic analgesia condition, and greater during the stress period itself in the waking condition. The difference within the stress period, while not striking ($p = 0.05$, one-tailed), is in line with the results reported by Sachs for his subjects who were also highly susceptible and trained.

In the ischemia part of the experiment we were able to replicate Lenox's findings in part. That is, if measured from a relaxation baseline, the total rise in blood pressure, in our experiment as in his, was significantly less than in waking.

However, further analysis led us to raise some questions about this finding. When the total course of blood pressure was followed, it was noted that there were consistent perturbations in the blood pressure, an initial rise (prior to the stress) that at prestimulus in some instances actually exceeded the final blood pressure. Within the ischemic period itself the blood pressure fell off for a time, probably because of the delay before any severe pain is felt, and then rose very slowly. Apart from initial blood pressure being consistently higher for these subjects when they faced the analgesic task, the course of the blood pressure rise within ischemia itself was not appreciably different between waking and analgesia, as shown in Fig. 2.

These results do not necessarily refute Lenox's findings, although the fact that we support his findings when we use his analysis, and do not support them when we bring into play additional anticipatory measures not registered by him, means that the matter remains ambiguous. There were some differences between our experiments. He used more experienced subjects whom he had trained himself often through many hours of practice. In addition, Lenox carried his pain beyond the pain at which we terminated our subjects, and their agitation in the final stages of waking could be responsible for some of the excessive blood pressure rise in waking. These waking responses produce the difference between our experiments and his; the rises within hypnotic analgesia were not significantly greater in our experiments than in his.

We are left about where we were with heart rate: poorer evidence of blood pressure reduction within the cold pressor response as a conse-
Fig. 2. The course of finger blood pressure over time in normal waking and in hypnotic analgesia in an experiment with tourniquet-exercise ischemia of the arm. The anticipatory rise occurs before the ischemic condition has been produced. The ischemia and exercise take place between the prestimulus announcement and the first measure of pain within stress. Mean values of 8 highly susceptible subjects, who reported no felt pain whatever during the stress period under hypnotic analgesia.

A consequence of analgesia than was found for heart rate; ambiguous evidence with respect to ischemia. The conservative conclusion is that no striking change in blood pressure response to a normally painful stress can be expected as a consequence of hypnotic analgesia.

**Effects of anticipated pain and hallucinated stress**

A confounding problem in all of the experiments with induced pain was that the physiological indicators associated with pain began to rise in the period prior to the onset of the physical stress, as already illus-
trated in Fig. 2. Interestingly enough, these anticipatory responses often occurred even when the subject, experienced in hypnosis, knew that no pain was going to be felt because of hypnotic analgesia. The question of the magnitude of these anticipatory reactions, as compared with those during stress, has implications for psychophysiology, but it also bears upon problems of measurement in the study of pain.

When no analgesia was involved (even though the subjects were hypnotized), there was very little, if any, anticipatory rise in heart rate in the cold pressor test. Hence the heart rate was not increased by the expectation of the sudden pain of immersion in the cold water, even though the stress itself caused a substantial rise. Curiously, the anticipatory rise in heart rate was greater when the subject knew that he was going to be required to reduce the pain through hypnotic analgesia. These responses, as shown in Fig. 3, can be interpreted to mean either

that there is something conflictual about the hypnotic demand for reducing the pain of the ice water, or that some cognitive effort is involved in maintaining the condition required by the hypnotic suggestions. The
effort may be reflected in the heart rate, following the kinds of hypotheses proposed by Lacey (1967).

While the anticipatory rises in heart rate do not approach in magnitude the responses within the stress itself, with blood pressure the situation is different. In several experiments the anticipatory rise and the stress rise did not differ significantly, whether in the cold pressor or the ischemic experiments; in only one study of the series was the stress rise greater than the anticipatory rise. Unless these anticipatory effects are carefully taken into account, misleading results could be found, especially when comparing hypnotic analgesia (in which the anticipatory rises are prominent) and waking pain (in which the anticipatory rises are smaller or absent).

As a control on these anticipatory effects, the changes in cardiovascular responses were also studied in a condition in which the ice water pain was hallucinated. This made it possible to distinguish between the effects related to subjectively felt pain and those owing to the physical stimulation by cold water. The first finding, holding for both heart rate and blood pressure, was that the anticipatory responses were those corresponding to the rises found in hypnotic analgesia. This appears to support the interpretation that something having to do with maintaining

![Heart rate acceleration in hypnotically hallucinated pain compared with normal pain (cold pressor test). Mean values of 12 highly susceptible subjects. The hallucinated pain reached a level only slightly below that of the normally experienced pain.](image)
the hypnotic condition is responsible, for it did not matter whether pain or no pain was expected. The second finding was that, for heart rate, the changes in the hallucinated stress followed the same course and at roughly the same magnitude, as in response to the actual stress (Fig. 4). The differences are not statistically significant, but it may be noted that the hallucinated pain was reported somewhat below the physical pain in intensity and the heart rate corresponded with this difference. That the consequences of hallucinated pain do in fact differ from those of physically induced pain was shown in the blood pressure concomitants. Here there was an initial rise in blood pressure as the hallucinated pain began, but as the subjective pain continued to mount the blood pressure fell to the initial baseline (Fig. 5). This is the clearest demonstration of all that heart rate and blood pressure are not equally responsive to subjectively felt pain.

This summarization completes the main findings from this series of experiments. The findings provide the background for some interpretive comments, and have led to further experiments in new directions.

Fig. 5. Blood pressure changes in hypnotically hallucinated pain compared with normal pain (cold pressor test). The records were obtained simultaneously with those of Fig. 4. Note that in hallucinated pain the blood pressure rises initially but then falls off as the pain mounts.
In the study of suprathreshold pain, with which these studies have been concerned, the most satisfactory measure has been the verbal report on a simple numerical scale. While in itself such a single value cannot completely describe the pain that is felt, it is more specifically related to the intensity and duration of stimulation than any other single measure that has been reported, either in our experiments or by those using other responses. To employ a numerical measure of this kind most successfully it is desirable both to have a scale open at the top and to have some reference values so that all subjects report on scales with similar units.

If physiological measures are to be used in the study of suprathreshold pain it is important that the stresses used are severe enough to affect these measures substantially. In some of the experiments on cold pressor pain, for example, the ice water has not been circulated. In such experiments a warm layer develops around the submerged bodily member, and pain is less intense; correspondingly, the heart rate is likely to rise very little. Barber and Hahn (1964) reported a rise of but 1.5 BPM over 1 min in water at 2°C, when there was no stirring. With that little change it is not surprising that different conditions produced nonsignificant changes.

Another kind of problem is created by the prominence of anticipatory responses. When the consequences of pain are estimated from waste products in the blood or urine, it will be extremely difficult to separate the anticipatory stresses from those in the pain itself, unless careful controls are run in which the experiment is unexpectedly terminated before the actual stress is presented. There are, of course, some awkwardnesses in making use of this kind of control.

When the modification of pain by hypnosis is under consideration, it is essential to stratify subjects according to their measured hypnotic responsiveness, if various conditions, such as waking vs. hypnosis, are to be compared. Otherwise those who do not respond differentially to hypnosis will be thrown in with those who do, reducing the likelihood of finding any small differences attributable to hypnosis. Some of the contradictions in the experimental literature are based on the failure to observe this obvious requirement. Another persistent problem, in view of the wide individual differences in autonomic lability, is the matter of using a subject as his own control. Again, this has obvious statistical advantages; although there are also disadvantages that may be countered by the use of randomly selected groups, the advantages of using the subject as his own control greatly outweigh the disadvantages, especially
if the nature of the experiments limits them to a small number of subjects.

Our results overall argue against the reliance on any one (or even a small number) of physiological indicators associated with pain. In several instances heart rate and blood pressure yielded different results. Similarly, it is desirable to use more than one kind of pain. The results with cold pressor pain and ischemia, although largely consistent, were not completely symmetrical, particularly in relation to anticipatory responses and the relative effects on heart rate and blood pressure.

**Resolving the paradox of the persistence of physiological indicators when subjective pain is not felt**

There were sufficient increases in both heart rate and blood pressure for them to indicate that the body was being stressed during the pain experiments, whether or not there was a precise correlation with felt pain. What requires explanation, however, is that even in those circumstances in which the subject reported no felt pain whatever, the indicators of stress persisted at approximately their normal levels. Related findings led Sutcliffe (1961) to propose that hypnotic pain reduction must be considered a delusion. That is in part a matter of semantics, for if pain is defined by the physiological indicators that have been selected, then it persists in analgesia; if, however, pain is defined as something that hurts, absence of pain is genuine, regardless of what happens to the physiological measures. Semantics aside, the paradox of absent pain and present physiological changes remains to be resolved.

The first thing to note is that there are, in fact, bodily changes that are modified by the analgesia suggestions. There is no agitation, no writhing or moaning, and little change in respiration as compared with normal waking pain. These are all responses that have some voluntary components, but they are part of the total "physiological response" to pain. If one accepts a two-component theory of pain, one being a sensory-informative component, the other a reactive or suffering component, it may be that the reduction in these overt responses should affect the second component more than the first. This would appear to have some plausibility in relation, for example, to the gate-control theory of pain (Melzack and Wall 1965).

One of the experiments in our laboratory was designed to test this possibility (Knox, Morgan and Hilgard 1974). Using ischemic pain, separate reports on numerical scales were called for of sensory pain and of suffering, a distinction that the subjects could make without difficulty. In normal waking pain both ratings rose as the stimulation conti-
nued, with the suffering lagging behind the sensory pain, but gradually becoming closer as the intensity of the pain rose. Under hypnotic analgesia, as tested in the usual way, the eight highly responsive subjects in this experiment reduced both pain and suffering to zero. At least at this superficial level the hypothesis that suffering was affected more than sensory pain was not supported.

The theoretical problem has been approached by another technique, however, that is available when experimenting with subjects highly responsive to hypnosis. This goes back to some nineteenth century studies with automatic writing, in which reports of experiences under hypnosis may differ from those reported by the hypnotized person when questioned in the usual way. This does not mean that the report of the hypnotized person is in any sense dishonest, but rather that something like amnesia has prevented access to some of the information available through automatic writing. We have found that a related technique that we refer to as automatic talking yields similar results. The hypnotized subject is told that some part of him knows more about what has been going on than the hypnotized part, and that this part can be contacted when the hypnotist places his hand on the subject's shoulder. Under these circumstances the hypnotized subject who has just experienced hypnotic analgesia will report that this "hidden part" experienced sensory pain, but commonly he will report also that it did not trouble him. Hence there is a genuine possibility that at this hidden level, available only through such special techniques, it will be shown that suffering is indeed affected more than sensory pain by hypnotic analgesia. In the experiment previously referred to, this method of inquiry was also used, with an alternation every two minutes for an inquiry of the hypnotized part (reporting no pain and suffering) and of the hidden part (reporting nearly normal pain and suffering). Although these results did not give clear support to the reduction of suffering over sensory pain, it may be that in the frequent alternation of conditions there was a "sampling" of the pain and suffering within the course of the experiment, that is, a temporary turning off of the analgesia before restoring it. Such rapid shifts are known to be possible within hypnosis (e.g., Blum 1972). In any case, that first experiment did not give any substantial support to the hypothesis that suffering and sensory pain were differentially affected by hypnotic analgesia.

Subsequent pilot studies, undertaken just as this report is being prepared, have modified the experiment so that the inquiry about pain and suffering within hypnotic analgesia by way of automatic talking is postponed until the experiment has been completed. In that way the "sampling" referred to is not possible, because the pain is no longer pre-
sent. The first subjects studied in this manner, whose pain and suffering reports in waking were like those in the previous experiment, reported sensory pain retrospectively at essentially waking level, but assigned a zero level to suffering. If these results are confirmed, the hypothesis that suffering is more affected than sensory pain will be supported, and the paradox may be resolved. The resolution would be that the sensory pain suffices to affect the selected physiological indicators (which are only mild physiological responses in any case), while the suffering has been controlled. There remains the problem why the sensory effect is also denied in hypnotic analgesia. This must have something to do with a process like posthypnotic amnesia that is very familiar within hypnosis; the difference is that the pain that is “forgotten” must have been deflected from consciousness, that is, made “amnestic”, before it was ever fully experienced.

Nineteenth century investigators, such as Pierre Janet, proposed that the separate functions of the “conscious” and “subconscious” parts of cognitive apparatus were “dissociated”. While this concept is more descriptive than explanatory, we have been examining the possibility that a revised form of dissociation theory may prove to be instructive not only in explaining the complex relationships between consciousness of pain and events in the nervous system, but other cognitive control processes as well. Called neodissociation theory, only its broad outlines are now available, but it is hoped that a fuller account may soon be provided (Hilgard 1973ab, 1974).

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