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Published in:

Psychology & Health: Official journal of the European Health Psychology Society (EHPS)

DOI:

[10.1080/08870449808406127](https://doi.org/10.1080/08870449808406127)

Publication date:

1998

[Link to publication in Tilburg University Research Portal](#)

Citation for published version (APA):

Nyklicek, I., Vingerhoets, A. J. J. M., & van Heck, G. L. (1998). The under-reporting tendency of hypertensives: An analysis of potential psychological and physiological mechanisms. *Psychology & Health: Official journal of the European Health Psychology Society (EHPS)*, 13, 1-21. <https://doi.org/10.1080/08870449808406127>

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The Under-Reporting Tendency of Hypertensives:
An Analysis of Potential Psychological and Physiological Mechanisms

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Abstract

Hypertensives have repeatedly been found to exhibit diminished self-report rates of stressor exposure and distress. In this article we discuss the evidence for two possible explanations for this phenomenon by means of reviews of the literatures on blood pressure in relation to defensive coping and pain perception. This approach provides support for the plausibility of two, not mutually exclusive, explanations: one based upon defensive coping and the other in terms of a physiologically based altered appraisal of aversive stimuli involving baroreceptor activity. Moreover, some indirect evidence has been obtained for a possible association between the two mechanisms. These findings have resulted in the formulation of a hypothetical model in which baroreceptor mediated central nervous system inhibition results in less negative appraisal of stressful situations and contributes to the development of hypertension via an operant conditioning mechanism. In addition, this mechanism may underlie the so-called defensive coping style. Finally, some directions for future research, aiming at testing the proposed model, are offered.

Key Words: blood pressure, stress, appraisal, defensive coping, baroreceptors, pain

Introduction

Generally, it has been acknowledged that essential hypertension¹ is a complex multifactorial disease, in which biological, behavioral, and psychosocial factors play a role in its etiology and course (Davies, 1971; Pickering, 1990; Sommers-Flanagan & Greenberg, 1989). In addition, there is accumulating evidence that hypertension--and being aware of having elevated blood pressure--in its turn, may influence several psychological functions, including perception, memory, and behaviour (e.g., Irvine, Garner, Olmstead, & Logan, 1989; Madden & Blumenthal, 1989), and physiological processes, such as the baroreflexes (Ditto & France, 1990; Harrell, 1980). These complex interactions imply a dynamic relationship between essential hypertension and various psychological and physiological factors, which considerably complicates the interpretation of the results of many studies on the role of those factors in the onset and course of hypertension.

One major research tradition in this area has been focusing on the association between stressor exposure and distress on the one hand and development of hypertension on the other. A considerable amount of literature has been devoted to this theme, but we would like to call attention to specifically one intriguing and counter-intuitive phenomenon that has been repeatedly found in this research field. When potential confounders, such as awareness of one's blood pressure level, have been taken into account, hypertensives and persons with elevated blood pressure have been found to have diminished self-report rates of negative life events (Linden & Feuerstein, 1983; Svensson & Theorell, 1983; Theorell, Svensson, Knox, Waller, & Alvarez, 1986), occupational stressors (Jenkins, Hurst, & Rose, 1985; Winkleby, Ragland, & Syme, 1988), physical symptoms, such as headaches and dizziness (Davies, 1970; Kidson, 1973; Tibblin & Lindström, 1972), negative psychological characteristics, such as neuroticism, anxiety, and depression (Davies, 1970; Santonastaso, Canton, Ambrosio, & Zamboni, 1984), and painfulness of laboratory aversive stimulation (Rau et al., 1994; Zamir & Shuber, 1980) in comparison with normotensives. In some studies, continuous inverse associations have been found between blood pressure level and stress(or) report rates, even in normotensive samples (Winkleby et al., 1988; Tibblin & Lindström, 1972; Bruehl, Carlson, & McCubbin, 1992; McCubbin & Bruehl, 1994). All these studies strongly suggest a non-complaining

attitude and a tendency to under-report problems in hypertensives, especially in the unaware, asymptomatic individuals (Theorell, 1990). An exhaustive discussion of this research is beyond the scope of this article (see for an overview, Nyklíček, Vingerhoets, & Van Heck, 1996). Given the fact that exposure to psychosocial stressors is believed to contribute to the onset of hypertension (e.g., Henry, 1988), mechanisms which could potentially explain these counter-intuitive findings should be examined.

In the present article, literature will be reviewed on the relationship between elevated blood pressure and two phenomena, which may provide an explanation for the diminished subjective stress(or) report rates of hypertensives. First, it is hypothesized that hypertensives have a defensive coping style which minimizes the (reported) aversiveness of events, either unconsciously or consciously (e.g., repression, denial, and non-disclosure). Although this concerns an old notion, recent studies on the topic warrant a reevaluation. The second hypothesis states that hypertensives have a physiologically based altered appraisal of aversive stimuli, as a consequence of which they become less threatening. Evidence supporting this hypothesis comes mainly from research on blood pressure and pain appraisal. Finally, it is suggested that these two explanations may not be independent of each other.

The literature searches on the relevant studies were carried out using electronic databases, such as PsychLit and Social Sciences Citation Index. Furthermore, existing reviews of the relevant literature (Davies, 1971; Randich & Maixner, 1984; Sommers-Flanagan & Greenberg, 1989) were consulted. When discrepancies between reported results are found, possible differences between the studies will be discussed with respect to: subject sample characteristics, operationalizations of the independent and dependent variables, ways of controlling for confounding variables, and the design and statistical methods used. In addition, when considering the relative importance of the results of the investigations discussed, several epidemiologic criteria will be applied, especially regarding the nature of the samples of subjects used. In this regard, particularly two mutually related issues are highly relevant as to their potential to moderate (Baron & Kenny, 1986) the targeted relationships: selection bias and labelling. Selection bias occurs when the hypertensive sample consists of patients treated for the disorder. Hypertensive patients may differ on a number of psychological

characteristics from persons which are unaware of their elevated blood pressure, such as neuroticism and anxiety (Irvine et al., 1989; Santonastaso et al., 1984). A relatively elevated level of these characteristics, which may have enhanced the probability of hypertension diagnosis in patient samples, may influence several other variables of interest, in particular those assessed by self-reports (Irvine et al., 1989; Monk, 1980; Soghikian, Fallick-Hunkeler, Ury, & Fisher, 1981). Therefore, in research on the relationship between psychosocial variables and blood pressure, results of studies based on samples from the general population should be regarded as more representative and more conclusive than research on hypertensive patient groups. Related to this issue is the notion that self-reports are also influenced by labelling a person as "hypertensive" (Berghlund, Ander, Lindström, & Tibblin, 1975; MacDonald, Sackett, Haynes, & Taylor, 1984; Rostrup & Ekeberg, 1992). This implies that research based on unlabelled subjects should be given more weight than studies involving labelled hypertensives. Finally, needless to say that prospective investigations usually yield more powerful conclusions than cross-sectional research, particularly with respect to causal relationships.

Blood Pressure and Defensive Behaviour

In the discussion of the relationship between blood pressure and defensiveness, those behaviours will be emphasized, which may have an influence on the self-report rates of stressor exposure in hypertensives. These behaviours include constructs such as repression, defensiveness, and non-disclosure.

There is much conceptual confusion concerning these constructs. Some researchers have used them as synonyms (Sommers-Flanagan & Greenberg, 1989), while others have attempted to make theoretical distinctions between them, which however appeared to be mutually incongruous (Paulhus & Reid, 1991; Sackheim, 1988; Tomaka, Blascovich, & Kelsey, 1992). Trying to disentangle the various constructs conceptually is beyond the scope of this article. Our interest here concerns a common feature that is shared by these defensive behaviours: deception of the self or the other in order to prevent problems or to put the self (in front of others) or the external world (in front of the

self) in a more favourable light, regardless of whether the subject is aware or unaware of this. More precisely, this implies that an individual using these coping styles tends to trivialize negative experiences and avoids the expression of negative emotions. In this way defensive behaviour might be responsible for distortions of self-reported distress and stressor exposure. Literature on alexithymia, a personality concept related to repression (Cumes-Rayner & Price, 1989), will not be reviewed here. The major reason is that alexithymics have been found to exhibit higher scores on both somatic and psychological symptoms scales (Cohen, Auld, & Brooker, 1994; Taylor, Parker, Bagby, & Acklin, 1992) and to report more hassles and perceived stress (Kohn, Gurevich, Pickering, & MacDonald, 1994) than individuals scoring low on alexithymia. These findings would contradict a potentially explanatory role of alexithymia in under-reporting distress and stressor exposure. Moreover, in recent studies no evidence for a relationship between alexithymia and blood pressure has been found (Newton & Contrada, 1994; Kauhanen, Julkunen, & Salonen, 1991). Although self-disclosure may be regarded as a reporting tendency rather than a coping style, a review of the literature on the relationship between blood pressure and self-disclosure will be included in this article. It has been suggested that frequently a false-positive diagnosis of repression was due to a mere tendency not to disclose threatening information (Cumes-Rayner & Price, 1989).

First, we will discuss studies concerning the relationship between blood pressure and constructs like repression, defensiveness, social desirability, denial, and self-deception. In the second subsection, the results of studies focusing on the relationship between blood pressure and self-disclosure will be reported.

Repression and Related Constructs

In the literature concerning emotional repression and hypertension, many studies have traditionally been dealing with repression of anger/hostility (Ewart, 1991). Occasionally, no relation between repressed hostility and hypertension was found (e.g., Cochrane, 1973). Other, more recent studies (Aritzi et al., 1989; Boutelle, Epstein, & Ruddy, 1987; Hafner & Miller, 1991; Netter & Neuhäuser-Metternich, 1991) demonstrated that hypertensive patients scored higher on repressed aggression than normotensives. These studies should be interpreted with caution, however, in view of

the danger of biases due to awareness of having the disorder, treatment, and self-selection in research based on patient samples (Irvine et al., 1989; Nyklíček et al., 1996).

In screening studies, conducted in general populations, hypertensive samples consist of subjects in which high blood pressure has just been diagnosed, instead of hypertensive patients. This implies that biases due to treatment or self-selection do not apply. In one such screening study, Mann (1984) found no evidence for repressed hostility in unaware hypertensives in comparison with normotensives. However, the subjects consisted predominantly of individuals who responded positively to the General Health Questionnaire (Goldberg, 1972), thus admitting having psychological problems. Therefore, the sample can hardly be viewed as being representative of the general population, which seriously obscures any conclusions regarding the targeted relationship. In another screening study, Thomas and Kirkcaldy (1988) found diminished scores on aggressiveness, dominance, and openness to expression in young individuals who were just diagnosed as hypertensives. The authors interpreted these findings as not being indicative of repression, but as being the result of inoculation. This concept, introduced by Eysenck (1983), refers to a diminished sensitivity to acute challenges as a result of frequent or chronic exposure to stressors. Given the fact that hypertensives have frequently been found to be cardiovascular hyperreactors to behavioral stressors, thus being physiologically sensitive to stressors (Anderson, 1989; Drummond, 1985; Pickering & Gerin, 1988; Steptoe, Melville, & Ross, 1984), the inoculation hypothesis does not seem very plausible. Moreover, Eysenck himself has suggested that inoculation may play a role in cancer rather than in hypertension (Eysenck, 1983). In addition, the presence of the moderating effect of labelling (MacDonald et al., 1984) on the self-reports can not be ruled out in both of these investigations. Finally, Cottington and coworkers (Cottington, Matthews, Talbott, & Kuller, 1986) found that among hourly workers, those who suppressed their anger and reported more uncertain job future and dissatisfaction with co-workers, were more likely to suffer from elevated blood pressure. This study is the most convincing one of the screening studies discussed so far, since in addition to have been sampled from the general population, the subjects were not labelled as hypertensives.

Cardiovascular hyperreactivity is suggested to be a risk factor for the development of hypertension in two recent prospective studies (Light, Dolan, Davis, & Sherwood, 1992; Menkes et

al., 1989). Jorgensen and Houston (1988) investigated whether hyperreactivity was associated with repression of aggressive feelings. They found that systolic blood pressure reactivity was associated with repressed hostility in subjects with a family history of hypertension. This result suggests that repression of aggression is associated with a risk factor for hypertension. A similar association has been suggested for repression or denial in general; in several studies, repressors and individuals with a family history of hypertension who were characterized by denial have been found to be cardiovascularly more reactive than non-repressors (Jorgensen & Houston, 1986; Linden, 1985; Weinberger, Schwartz, & Davidson, 1979). These studies, conducted on healthy, normotensive subjects, thus provide support for a relationship between defensiveness and risk for hypertension.

This brings us to studies concerning repression and related concepts in general (see Table 1).

Insert Table 1 about here

In his review of the literature, Davies (1971) concluded that the relationship between blood pressure and psychosocial factors has often been obscured by self-selection bias and inaccurate measurement methods. When not suffering from these methodological drawbacks, results from several studies have suggested that high blood pressure may indeed be related to emotional inhibition. Also in a more recent review (Sommers-Flanagan & Greenberg, 1989) support was reported for an association between hypertension and the use of psychological defense mechanisms--i.e., denial and repression--, both in patient samples and in samples of unselected unaware hypertensives. In the study by Sapira, Scheib, Moriarty, and Shapiro (1971), hospitalized patients with hypertension watched two movies about doctor-patient interactions. In one film the doctor was rough and not interested in the patient (the bad doctor), in the other one the doctor was gentle and considerate (the good doctor). Hypertensives showed enhanced pressor and pulse rate responses during the films and during the interview. The most important finding, however, was that the hypertensives tended to deny seeing any differences in behaviour between the two doctors, which were clearly noted by the normotensive controls. Thus, hypertensive patients seemed to perceive noxious information less

clearly. Relevant in this context is the view of Lacey and Lacey (1970), who have suggested that cardiovascular pressor responses--which are stronger in hypertensives--may go along with rejection of or non-responsiveness to the external environment.

Santonastaso et al. (1984) reported that recently diagnosed hypertensives scored significantly lower on neuroticism, depression, and inadequacy than normotensives. They also had lower scores for neuroticism, anxiety, somatization and inadequacy than aware hypertensives. It was concluded that "'new' hypertensives seem to deny life problems and psychological disturbances" (p. 11). Tibblin and Lindström (1972) found that untreated hypertensives reported less physical and psychological symptoms than normotensive controls. Moreover, for most symptoms a gradient was found: the higher the blood pressure the lower the reported frequency of symptoms. Therefore, these authors concluded that hypertensives "are denying or suppressing their feelings" (p. 139). Unfortunately, in these two studies, repression or denial was not measured directly.

In contrast with the studies discussed above, in several investigations on general population samples blood pressure level was the dependent variable. This procedure results in more valid outcomes by controlling for the undesirable moderating effects of selection-bias and labelling. Nevertheless, the direction of the associations found was the same as in the former studies. For instance, in an epidemiological study in Michigan (Cottington, Brock, House, & Hawthorne, 1985), suppressed emotion was associated with higher blood pressure in both males and females undiagnosed and untreated for hypertension. Our research group has recently investigated the relationship between blood pressure, defensive coping, and self-reported daily hassles in a sample of 141 male and female employees from a wide range of occupations (Nyklíček, Vingerhoets, Van Heck, & Van Limpt, 1995). Self-report questionnaires were filled out before the collection of the medical data in order to prevent that knowledge of blood pressure level would influence the self-reports. After controlling for eight potentially confounding variables, we found that repressors reported fewer daily hassles, while exhibiting higher resting systolic blood pressure levels than non-repressors. Finally, in both medical patients (Warrenburg et al., 1989) and in subjects from a general population sample (King, Taylor, Albright, & Haskell, 1990), it has recently been demonstrated that repressive individuals have higher resting systolic blood pressure levels and higher systolic blood pressure reactivity

in response to a mental challenge than non-repressive individuals.

In summary, evidence for a relationship between elevated blood pressure and defensive behaviour is accumulating. Support for this association has been obtained from patient- and from population-based studies and from both studies treating blood pressure level as an independent and as an outcome variable.

Self-Disclosure

According to Cumes-Rayner and Price (1989), one may wonder whether the diagnosis of repression and denial often not merely represents a preference not to disclose. In this section, we discuss research that examined the relationship between blood pressure and disclosure of negative events or emotions without reference to a repressive coping style.

Pennebaker (1988, 1989) has shown that, in general, disclosure of previously inhibited experiences positively affects autonomic nervous system (ANS) activity (lower baseline values) in the long run. Handkins and Munz (1978) reported that, in an interview situation, treated male hypertensives disclosed less about intimate topics than male normotensives matched on age and weight/height ratio. Unselected young males with elevated blood pressure have been found to express sorrow to significantly less people than their normotensive counterparts (Knox, Svensson, Waller, & Theorell, 1988). Cumes (1983) showed that subjects with high blood pressure reported less concerns than normotensives. They were also less likely to report feelings of being distressed after a cognitive task, even when blood pressure increases occurred. Finally, Cumes-Rayner and Price (1989) found that borderline hypertensive students preferred not to disclose information about their concerns in comparison to normotensives. When they felt that there was no other option than to disclose, this had a disregulatory effect on their blood pressure. In this study disclosure correlated highly with repression and anxiety in the hypertensive group.

Thus, results of this research, predominantly obtained from unbiased samples, suggest that hypertensives indeed may have a preference not to disclose information about experienced negative

events or emotions.

Conclusions

The results from the studies concerning the relationship between blood pressure and coping/reporting style reviewed here suggest that individuals with elevated blood pressure tend to be defensive and are inclined to minimize aversiveness, in particular concerning their negative emotions and experiences. The issue of causality cannot be resolved from the cross-sectional studies reviewed here. Prospective research would be needed to obtain more insight into causal relationships. However, apart from the question whether the association is a causal one--and if so, what the direction of this causality might be--the importance of the association between blood pressure and defensive behaviour is that it may explain the observed diminished stressor report rates by an altered appraisal of threatening stimuli or by hiding negative information. The mutual relationship between appraisal and coping has been extensively described by Lazarus and coworkers (Lazarus, 1966; Lazarus & Folkman, 1984). When a situation is first appraised as being threatening, using a repression-like coping style may immediately alter the (primary) appraisal into a more positive evaluation of the situation. Alternatively, if the mechanism would rather consist of just hiding negative information from others, primary appraisal would largely be unaffected, implying the need for another coping strategy to deal with the--still threatening--situation.

To what extent the under-reporting may be mediated by one or more of the defensive constructs mentioned in this review (repression, self-deception, non-disclosure, etc.) remains unclear, partly due to the definitional confusion. Therefore, elucidating the theoretical and statistical relationship between these constructs seems desirable in order to be able to design studies that could discriminate between the hypotheses regarding the role of defensive behaviour in the under-reporting tendency in hypertensives.

Blood Pressure and Appraisal of Painful Stimuli

Evidence for an association between blood pressure and altered appraisal of threatening

stimuli has also been obtained from pain research. In the past 15 years a solid body of evidence has been accumulated regarding links between high blood pressure and diminished sensitivity to painful stimulation (antinociception), presumably as a result of baroreceptor stimulation mediated central nervous system (CNS) inhibition. In their review on the association between cardiovascular and pain regulatory systems, Randich and Maixner (1984) have stated that pharmacological, neuroanatomical, electrophysiological, and behavioral data have provided evidence for a strong association between cardiovascular function and the perception of pain. It has been demonstrated that individuals with high blood pressure have a higher pain threshold and pain tolerance than individuals with normal blood pressure, indicating a tendency for hypertensives to perceive these stimuli as less aversive. This has been found for electrical (Elbert, Rockstroh, Lutzenberger, Kessler, & Pietrowsky, 1988; Zamir, Simantov, & Segal, 1980), thermal (Sheps et al., 1992), and finger pressure pain stimulation techniques (Bruehl, Carlson, & McCubbin, 1992). Moreover, also in normotensive samples an inverse relationship between blood pressure and perceived painfulness of physical stressors has been obtained (Bruehl et al., 1992). This inverse relationship has been found in both between-subjects (Zamir & Shuber, 1980) and within-subjects designs (Dworkin, Filewich, Miller, Craigmyle, & Pickering, 1979), in animal (Randich & Maixner, 1984) as well as in human studies (Sheps et al., 1992). The results of these studies are presented in Table 2.

Insert Table 2 about here

Animal Studies

The first evidence came from animal studies, when Dworkin et al. (1979) showed that artificially raising blood pressure in rats caused antinociception. When the baroreceptors of the rats were denervated, however, this effect was not seen. The authors concluded that "a rise of blood pressure could have motivational consequences significant for human hypertension" (p. 1299). This hypothesis is discussed in more detail below. In the same year, Zamir and Segal (1979) reported a similar effect: rats in which hypertension was induced by applying a solid silver clip on the left renal

artery showed elevated pain thresholds. In other experiments, direct baroreceptor stimulation produced antinociception (Randich, 1986; Randich & Hartunian, 1983). Moreover, Saavedra (1981) showed that in hypertensive rats antinociception (in that article called 'analgesia') to a thermal pain stimulus could be reversed by the opiate antagonist naloxone; in normotensive rats, however, no naloxone effect could be detected. This result suggests opioid involvement in this form of antinociception in hypertensive rats. Animal studies have also yielded evidence of opioid involvement in the development of hypertension; development of hypertension of different etiologies could be attenuated in dogs and rats by chronic opioid receptor blockade by naloxone (Szilagyi, 1989). Maixner and Randich (1984) showed, however, that when the cardiopulmonary baroreceptors were activated by volume expansion, this resulted in antinociception to heat in rats which did not appear to be mediated by endogenous opioids: naltrexone, an opiate antagonist, had no effect on it. Thus, in some forms of antinociception, a non-opioid system seems to be involved. It has been suggested that an opioid system may be involved in exposure to low-intensity short-duration aversive stimuli, whereas non-opioid mechanisms may predominate when the organism is exposed to high-intensity stressors of long-duration (Terman, Shavit, Lewis, Cannon, & Liebeskind, 1984).

Human Studies

From human studies, there is also a growing body of evidence supporting the association between elevated blood pressure and antinociception. For instance, Zamir and Shuber (1980) found that hypertensives have higher pain thresholds to electrical tooth pulp stimulation than normotensives. Sheps et al. (1992) showed that hypertensives have a higher pain tolerance and a higher pain threshold, when confronted with a thermal pain stimulus. In their study, hypertensives also had higher plasma β endorphin levels than normotensives; again pointing at the possible role of opioids. Bruehl et al. (1992) used an apparatus that exercised pressure on a finger as pain inducer. They found that, in normotensives, pain ratings were inversely related to resting systolic blood pressure, indicating a continuous negative relationship between blood pressure level and pain sensitivity. Similar results were reported in a study with normotensive subjects, in which the involvement of the opioid system was tested by opioid blockade by naloxone (McCubbin & Bruehl,

1994). Unfortunately, the results concerning the role of opioids remained inconclusive. After naloxone blockade the negative correlation between resting systolic blood pressure and pain ratings was no longer significant, but the difference between the magnitude of the two correlations also failed to reach significance. Moreover, in multiple regression analyses with pain ratings as the dependent variable, naloxone did not significantly modify the predicting power of resting systolic blood pressure. Rau et al. (1994) used both thermal and finger pressure pain inducers in a study in which hypertensives and normotensives were compared regarding their pain thresholds in a baroreceptor stimulation condition and in a control condition. Consistent with the study of Sheps et al., hypertensives exhibited higher pain thresholds for thermal pain than normotensives. However, baroreceptor stimulation had no effect on pain thresholds for thermal pain. In contrast, baroreceptor stimulation did heighten pain thresholds for pressure pain, but for this stimulus hypertensives and normotensives did not differ in terms of their absolute pain thresholds. The latter finding is inconsistent with the results obtained by Bruehl et al. This discrepancy may be due to the fact that all subjects in Bruehl et al.'s study had to endure the pain stimulus for one minute, thus containing a strong component of inescapability, whereas in the study of Rau et al. the subjects disengaged the pain inducer when pain threshold was reached. It could therefore be hypothesized that having control in a (pressure) pain design abolishes any differences in pain sensitivity between hypertensives and normotensives. An other explanation for this discrepancy may be the fact that the pressure pain experiment took place after the thermal pain session on another day, which could have eliminated differences between the groups due to habituation to the setting. With respect to the failure to find a baroreceptor stimulation effect for the thermal stimulus, the authors suggested that baroreceptor activity may not have a pain-inhibitory effect for all pain modalities, possibly due to, among other things, different anatomical pathways existing for different pain modalities.

Elbert et al. (1988) studied the effect of baroreceptor stimulation on sensitivity to pain induced by electric stimulation. They demonstrated that in students with elevated blood pressure, baroreceptor stimulation elevated their pain threshold, whereas the same procedure had the opposite effect in normotensives. These results suggest that baroreceptor mediated antinociception may not operate in all individuals; it may depend on the presence of an unknown moderator variable, for

instance a certain physiological predisposition. Additionally, the authors found a positive shift in negative cortical slow waves during baroreceptor stimulation, suggesting cortical inhibition. This positive shift was related to resting blood pressure, indicating differences in cortical activity during pain between normotensives and hypertensives. The authors concluded that these differences may be linked to a risk factor for hypertension development, by means of an operant conditioning mechanism, which is discussed below. The suggestion of a link between antinociception and risk for hypertension is in accordance with the findings of France and coworkers (France, Ditto, & Adler, 1991; France & Stewart, 1995). They compared healthy males at risk for hypertension with subjects without an increased risk for hypertension but with the same resting blood pressure values. The risk for hypertension was defined as either the presence of a parental history of the disorder or enhanced cardiovascular reactivity. In both studies, the experimental group showed reduced pain sensitivity, in the first one to a constrictive thigh-cuff pressure stimulus (France, Ditto, & Adler, 1991), in the latter one to an ischemic pain stimulus (France & Stewart, 1995).

In conclusion, although some inconsistencies have been found regarding results which involved different pain modalities, overall, a good body of evidence exists for an association between blood pressure and baroreceptor activity mediated (and at least in some cases opioid based) antinociception. Thus, hypertensives appear to have a physiologically based altered appraisal of physical aversive stimuli. The evidence is particularly convincing given the fact that most studies have been performed on unselected and unlabelled human samples or on animals; some of which were based on normotensive subjects only. It is tempting to speculate that the same mechanisms found for physical stressors are responsible for an altered appraisal of psychosocial stressors, which could be involved in the diminished stressor and symptom report rates by hypertensives. Future research should address these important issues, the more since the extrapolation of the diminished appraisal of painful stimuli in hypertensives to appraisal of psychosocial stressors has been an implicit assumption in the operant conditioning of hypertension hypothesis, discussed below.

Operant Conditioning of Blood Pressure Elevation

Given the frequently found association between blood pressure elevations and antinociception, a mechanism has been proposed that may have etiological implications for hypertension (Dworkin et al., 1979). As Randich and Maixner (1984) have suggested: "the inhibition of pain brought about by elevations in either arterial or venous blood pressure may provide a form of psychophysiological relief under situations of stress exposure and contribute to the development of essential hypertension in humans" (p. 343). This hypothesis proposes an operant conditioning mechanism operating under stressful situations, which would be--partially--responsible for the development of idiopathic hypertension. This mechanism would presumably involve baroreceptor stimulation mediated CNS-inhibition, which may be based on endogenous opioid activity. Indirect support for a part of this hypothesis is obtained in research on stress-induced analgesia, which is a well recognized phenomenon demonstrated in animals as well as in humans (Amit & Galina, 1988; Bandura, Cioffi, Taylor, & Brouillard, 1988). For instance, Bandura et al. (1988) found in human subjects that high distress, induced by a frustrating cognitive task, which decreased perceived self-efficacy, resulted in higher heart rate and activation of the endogenous opioid system, and hence, in diminished pain sensitivity to the cold pressor test. When the opioids were blocked by naloxone, no decrease in pain sensitivity was observed. This result demonstrates the association between perceived stressfulness of a situation, cardiovascular activation, and opioid mediated antinociception. Furthermore, in animal studies it has been demonstrated that stress-induced, opioid system mediated analgesia can be conditioned to environmental cues (Amit & Galina, 1988). However, no attempts were made to examine the involvement of the baroreceptors. More direct support for the plausibility of the proposed mechanism is provided by the findings that blood pressure elevations (a) can be learned instrumentally in both animals and in humans (Dworkin, 1988) and (b) can result in cortical inhibition, which has been found to be associated with diminished pain sensitivity (Dworkin et al., 1994; Elbert, Roberts, Lutzenberger, & Birbaumer, 1992; Elbert et al., 1988). Finally, in the first prospective study regarding the conditioning hypothesis of hypertension, Elbert et al. (1994) recently demonstrated that sensitivity to baroreceptor mediated antinociception predicted an increase in blood pressure 20 months later; the increase being proportional to self-assessed daily life stress.

Integration and Hypothetical Model

If elevated blood pressure is associated with both lower pain sensitivity and defensive coping, it would be of interest to investigate the possible links between these findings.

Davidson and Bobey (1970) found different responses to pain between repressors and sensitizers. Repressors showed initially to be less pain sensitive (higher pain tolerance), but they also showed a significant decrease in pain tolerance from trial 1 to 2, which was not exhibited by the sensitizers. Dubreuil, Endler, and Spanos (1988) reported that repressors had higher pain tolerance for acute focal pressure pain than sensitizers. In two other studies (Jamner & Schwartz, 1986; Jamner, Tursky, & Leigh, 1986) it has been found that repressors had higher pain thresholds and tolerance levels during electric stimulation than low self-deceptives, while no differences in sensory threshold were obtained. Support for possible opioid mediation has also been found. An association has been reported between repressive/defensive coping style and those immune function parameters, that are linked with the opioid system (Jamner, Schwartz, & Leigh, 1988). In addition, Jamner and Leigh (1994) reported that, although high self-deceptives had higher pain tolerance than low self-deceptives under both naloxone and saline conditions, a naloxone-induced hyperalgesia (enhanced pain sensitivity) was found in high self-deceptives, but not in low self-deceptives. The authors, therefore, suggested the existence of a relationship between repression and altered opioid function.

Thus, it could be hypothesized that the nervous system-inhibiting effects brought about by baroreceptor activation--and possibly involving the opioid system--, may both play a role in the development of hypertension and underlie the so-called "defensive or repressive" coping style. Diminished appraisal of stressors would play a central role in both relations. Additionally, repression has also been proposed to be involved in the development of cardiovascular disease (Alexander, 1939; McClelland, 1979; Niaura, Herbert, McMahon, & Sommerville, 1992; Schwartz, 1990), although until now not much direct support has been obtained for this link. Together, these relationships form our hypothetical model, which is graphically represented in Figure 1.

Insert Figure 1 about here

In the upper part of the illustration the conditioning mechanism is depicted. The link between this mechanism and both hypertension and "defensive coping" can only be established after prolonged periods of chronic or recurrent stressor exposure. Defensive coping is in quotes here, because in this context, defensiveness might just be reflecting a physiologically based, more or less permanent, altered appraisal of threatening stimuli, not defensiveness in the classical sense. It should also be noted that the mechanisms described here are not universal, they do not apply to all individuals. As suggested by Elbert et al. (1988), sensitivity to the cardiovascular dampening mechanism probably differs between individuals and may constitute a part of the hypertension-prone disposition. Given the fact that much of the evidence that has led to the formulation of this hypothetical model has been indirect, in the future the emphasis should be on the development of designs for testing directly (parts of) the model.

Conclusions and Future Research Directions

In this review, two explanations for the under-reporting tendency among hypertensives have been offered and discussed. This has been realized by means of reviews of the literatures on defensive coping and related constructs, and pain appraisal in relation to blood pressure.

For the role of defensive coping styles and for the tendency not to disclose intimate or threatening information to others support has been found. Numerous studies have demonstrated significant associations between elevated blood pressure and defensive coping style and a tendency not to disclose unfavourable information. This has been found in both samples of hypertensive patients and in samples of unselected and unlabelled subjects.

Evidence has also been found for the plausibility of a physiological mechanism responsible

for the under-reporting of distress and stressor exposure in hypertensives. An inverse association between blood pressure and pain perception has been reported in various studies performed with animals as well as with human subjects. Often this association has been found to be mediated by the activation of the baroreceptors and in several occasions the involvement of endogenous opioids has been shown. It might be hypothesized that baroreceptor activation, possibly enhancing central release of endogenous opioids, plays a role in the diminished perception and appraisal of both physical stressors (pain) and psychosocial stressors. Additionally, this mechanism may reinforce blood pressure elevations in potentially stressful situations, thus contributing to the development of some forms of essential hypertension. Finally, preliminary support has been obtained for a relationship between repression and diminished pain perception and, to a lesser extent, opioid activation. These findings have led us to construct a hypothetical model in which baroreceptor activity mediated CNS-inhibition, plays a role in both the development of hypertension and the exhibition of behaviour, which may be labelled as defensive. In order to test the proposed model, future studies need to address (parts of) the model directly and preferably also simultaneously. This implies the development of appropriate designs to test the relevant sub-hypotheses, of which some of the most crucial are listed below:

(a) Baroreceptor stimulation results in altered appraisal of both physical and psychosocial stressors. This hypothesis should be studied in a laboratory setting, in which the baroreceptors are stimulated experimentally, for instance, by the PRES neck-suction method, developed by Rau et al. (1994). As described earlier, some support for this hypothesis has been reported for several aversive physical stimuli. The crucial issue is to examine for which type of stressors this association holds. If this association can indeed be generalized to a range of psychosocial stressors, it would be desirable to examine the neurophysiological pathways involved, i.e., do the opioid systems play a role, what areas of the CNS, if any, are inhibited by the mechanism, etc.

(b) Blood pressure elevations can be conditioned to environmental cues. Although some support for this hypothesis has been obtained already (Dworkin, 1988), it should be demonstrated that this holds for cues related to both physical and psychosocial stressors.

(c) Altered appraisal of physical and psychosocial stressors is related to some defensive

coping styles in prolonged exposure to chronic stressors. Again, in order to be able to investigate this relationship, one first should disentangle conceptually and statistically the mutual relations between various defensive coping constructs. This hypothesis is suitable to be studied in both laboratory and in more ecologically valid settings, such as at the work place.

(d) Some defensive coping styles are related to enhanced sensitivity to baroreceptor mediated antinociception in prolonged periods of chronic stress. As will be discussed below, given its complicated nature, the assessment of chronic stress should be done very carefully.

(e) The combination of being exposed to chronic stressors and having an enhanced sensitivity to baroreceptor mediated antinociception is a risk factor for the development of hypertension. This hypothesis should be tested mainly by conducting prospective studies. The first direct evidence in favour of this hypothesis has recently been offered by the already discussed study by Elbert et al. (1994). Studies aiming at replicating their results should be encouraged.

When testing these hypotheses, the use of mixed experimental designs, including both within- and between-subject factors, should generally be recommended. The examination of a within-subject experimental factor gives opportunity to study mechanisms and to get a better insight into causal associations. The between-subjects factor, which is needed to investigate individual differences, would include variables such as risk for hypertension. This risk may be operationalized in terms of parental history of hypertension, sensitivity to baroreceptor mediated antinociception, or defensiveness. Groups at risk for hypertension, preferably with a high probability of experiencing stressful events in the near future or which just have been confronted with chronic exposure to stressors, would also be interesting to follow in prospective studies. This kind of research has been performed too rarely in the past and should receive much attention in the future in order to be able to draw conclusions about causal relationships between defensiveness, the baroreflex mechanisms and hypertension.

We would like to stress that many associations proposed in the model will not be found, unless the subjects are chronically exposed to stressors. Only when the individual is confronted with environmental threatening stimuli over and over again, these mechanisms will become apparent. This implies, that attempts to find associations between the proposed mechanisms and tonic blood pressure

level without taking into account stressor exposure, may reveal only weak relationships or may fail to show anything meaningful at all. If an individual hardly experiences any daily stressors, we would expect the risk of developing essential hypertension to be very low. According to the model, a complication regarding the measurement of distress and stressors arises: a higher blood pressure level will tend to be associated with a higher level of chronic stressor exposure, but at the same time a lower appraisal of the aversiveness of the stressors. These contrasting tendencies have also been demonstrated empirically (Fox, Dwyer, & Ganster, 1993). Self-reported environmental stressor exposure will always contain a mix of both entities, which will considerably complicate the interpretation of data based on this kind of measurements. For instance, despite the tendency to under-report experienced stressors in hypertensives, it is not inconceivable that one may find no relation or even a positive associations with blood pressure level (Schnall et al., 1990; Siegrist & Matschinger, 1989). This would be the result of an overriding effect of objective stressor exposure on blood pressure level. Therefore, in field research, it is recommended to simultaneously assess an objective index of stressor exposure, such as unemployment, and the subjectively perceived stressor exposure and obviously treat them as separate variables.

It has to be noted that attempts to find meaningful associations between altered appraisal and blood pressure reactivity will probably be obscured by the effect of distress and anxiety experienced in the trial, whether this takes place in the laboratory or in the field. As stated before, the sensitivity to baroreceptor mediated CNS-inhibition, as caused by blood pressure elevation, probably differs between individuals. In a person having a high sensitivity to this mechanism, exposure to a mild stressor would indeed tend to produce a negative association between experienced aversiveness and blood pressure reactivity. However, in a person having a low sensitivity to the baroreflex-CNS mechanism, a positive association between the same variables will emerge, as a result of the amplifying effect of distress and anxiety on blood pressure reactivity. Thus, when the sensitivity to the baroreflex-CNS mechanism of the subjects is unknown, it would be virtually impossible to predict the nature of the relationship between those variables. Therefore, it may be advisable to restrict oneself to relating appraisal to experimentally induced baroreceptor activity, instead of blood pressure reactivity.

Of course the objection may be raised that if the encounter with a stressor results in approximately the same amount of transient blood pressure elevations in individuals with a sensitivity to baroreceptor stimulation mediated antinociception and in those without such a sensitivity, then, why should the former group have a greater risk for hypertension in the first place? We should keep in mind that there is a qualitative difference in the nature of the blood pressure elevations between both groups: in the former group this elevation is a result of a reinforcement mechanism, whereas in the latter group the elevation is just an epiphenomenon of experiencing distress or anxiety. This implies that the former group may have acquired a general preference to react with a blood pressure elevation in a wide range of situations. Moreover, as Elbert et al. (1988) demonstrated, people with low tonic blood pressure levels even show a baroreceptor activation mediated hyperalgesia, suggesting that individuals without a risk for hypertension may even learn to try to avoid blood pressure elevations as much as possible. This implies that blood pressure elevations can be expected to take place in a much smaller range of situations in the latter than in the former group.

If the operant conditioning hypothesis of hypertension would be shown to be correct, this could have considerable consequences for hypertension treatment and even prevention. A behavioral therapy treatment of hypertension based on simultaneous extinction of the learned blood pressure elevation under stress, together with establishing other aversiveness reducing responses, may prove to be effective (Dworkin, 1988). For instance, certain forms of relaxation and stress management techniques may be suitable to establish alternative aversiveness reducing responses in hypertensives (Linden & Chambers, 1994). This does not imply that such a behavioral therapy treatment could not be supported pharmacologically. The treatment could also be applied for prevention purposes in persons at high risk for the development of hypertension, such as individuals having high sensitivity to the baroreceptor-CNS inhibition mechanism.

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The authors would like to thank James A. McCubbin for his comments on an earlier draft of this paper.

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Footnotes

¹ Frequently a distinction has been made between borderline hypertension (blood pressure level between 140/90 mmHg and 160/95 mmHg) and definite hypertension (blood pressure level of more than 160/95 mmHg). However, because blood pressure level is a continuous variable, every cut-off point on this continuum is an arbitrary one. For practical, especially diagnostic, purposes a distinction between hypertension and normotension can sometimes be useful, but the usefulness of a distinction between borderline and definite hypertension is equivocal (Pickering, 1990). Therefore, we only make this distinction occasionally for illustrative purposes. In general, when we discuss 'hypertension', we are referring to blood pressure of at least 140/90 mmHg (Lew, 1990) for which there is no known physical cause (Pickering, 1990).

Table 1

Blood Pressure and Repression

Studies	N ^a	Indep. variables ^b	Measurements ^c	Results ^d	
Blood pressure as independent variable					
Davies, 1971	review	HTs vs NTs	BP, self-report, interview	HTs: emotional inhibition	↑
Sapira et al., 1971	34 (m+f)	HT patients vs NT patients	BP, interview	HTs: emotional denial	↑
Tibblin & Lindström, 1972	622 (m)	untreated HTs vs NTs	BP, a 10-item scale	HTs: emotional denial	↑
Santonastaso et al., 1984	324 (m+f)	'new' aware HTs vs NTs	BP, SRT	HTs: emotional denial	↑
Sommers-Flanagan & Greenberg, 1989	review	HTs vs NTs	BP, self-report, interview	HTs: repression	↑
Blood pressure as dependent variable					
Cottington et al., 1985	402 (m+f)	emotional suppression	BP, a 8-item scale	DBP	↑
Jamner & Schwartz, 1986	534 (?)	repression	BP, MC-SDS, TMAS	BP-reactivity	↑
Jorgensen & Houston, 1986	122 (m+f)	denial, FH+	BP, MMPI-DS	BP-reactivity	↑
Warrenburg et al., 1989	45 (m)	defensiveness	BP, MC-SDS	SBP + SBP-reactivity	↑
King et al., 1990	120 (m+f)	repression	BP, MC-SDS, TMAS	SBP + SBP-reactivity	↑
Nyklíček et al., 1995	141 (m+f)	defensiveness	BP, WAI-SF (RD), EPCL	SBP	↑

table continues

table 1, continued

^a The number of subjects in the study: m = male, f = female, review = review article, based on studies with unknown number of subjects. ^b HTs = hypertensives (systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg), NTs = normotensives (systolic blood pressure < 140 mmHg and diastolic blood pressure < 90 mmHg), 'new' = individuals who just knew about their elevated blood pressure, FH+ = normotensives with a family history of hypertension. ^c BP = blood pressure, EPCL=Everyday Problem Checklist, MC-SDS = Marlowe-Crowne Social Desirability Scale, MMPI-DS = Denial Scale of the Minnesota Multiphasic Personality Inventory, SRT = Symptom Rating Test, TMAS = Taylor Manifest Anxiety Scale, WAI-SF (RD) = Repressive Defensiveness subscale of the Weinberger Adjustment Inventory (Short Form). ^d When groups were compared: scores of the first group (mentioned under 'Indep. variables') compared to the second group (HTs = hypertensives, SBP = systolic blood pressure, DBP = diastolic blood pressure, BP = blood pressure [systolic and diastolic], HR = heart rate, $\uparrow$ = higher).

Table 2

Blood Pressure and Pain Perception

Studies	N ^a	Design ^b	Stimulus	Ind. variables ^c	Results ^d	Opioids ^e
Animal studies						
Dworkin et al., 1979		16 (m) WS	electric shock	BP ↑	pain avoidance	↓ NT
Zamir & Segal, 1979		34 (m) WS	heat	BP ↑	pain sensitivity	↓ +
Saavedra, 1981	32 (m)	RMBS	heat	HTs vs NTs	HTs: pain sensitivity ↓	+
Randich & Hartunian, 1983	6 (m)	WS	heat	BS ↑	pain sensitivity	↓ NT
Maixner & Randich, 1984	15 (m) WS	heat	BS ↑	pain sensitivity	↓ -	
Randich, 1986	18 (f)	WS	heat	BS ↑	in HTs: pain sensitivity	↓ NT
Human studies						
Zamir & Shuber, 1980	55 (m) BS	electric shock	HTs vs NTs	HTs: pain sensitivity	↓ NT	
Elbert et al., 1988	20 (m) WS	electric shock	BS ↑	in HTs: pain sensitivity	↓ NT	
				in NTs: pain sensitivity	↑ NT	

table continues

table 2, continued

Studies	N ^a	Design ^b	Stimulus	Ind. variables ^c	Results ^d	Opioids ^e		
France et al., 1991		45 (m) BS	pressure	FH+ vs FH-	FH+: pain sensitivity ↓	NT		
Bruehl, et al., 1992		60 (m) BS	pressure	resting SBP ↑	rated pain ↓	NT		
Sheps, et al., 1992		20 (m) BS	heat	resting MAP ↑	pain sensitivity ↓	+/0		
Dworkin et al., 1994		139 (m+f)	WS	electric shock	BS ↑	rated pain ↓	NT	
McCubbin & Bruehl, 1994		16 (m) RMBS	cold	resting SBP ↑	rated pain ↓	0		
Rau et al., 1994	21 (m+f)	RMBSheat	HTs vs NTs	HTs: pain sensitivity ↓	NT			
				BS ↑	pain sensitivity 0	NT		
				pressure	HTs vs NTs	pain sensitivity 0	NT	
				BS ↑	pain sensitivity ↓	NT		

^a as in Table 1. ^b WS = within subjects design, RMBS = repeated measures between subjects design, BS = between subjects design. ^c BP = blood pressure, SBP = systolic blood pressure, MAP = mean arterial pressure, BS = baroreceptor stimulation, HTs = hypertensives (systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg), NTs = normotensives (systolic blood pressure < 140 mmHg and diastolic blood pressure < 90 mmHg), FH+ = family history of hypertension, FH- = no family history of hypertension. ^d ↓ = lower, ↑ = higher, 0 = no effect. ^e NT = role of the opioids not tested, +(/0) = role opioids (partially) confirmed, -(/0) = role opioids (partially) negated, 0 = results inconclusive.

FIGURE LEGEND

Fig. 1. Hypothetical model of an operant conditioning mechanism contributing to the development of essential hypertension.