Psychosomatic Research in Hypertension: The Lack of Impact of Decades of Research and New Directions to Consider

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Hypertension has long been suspected of being, in some patients, a psychosomatic disorder, ie, a physical disorder caused by psychological factors. A half century of psychosomatic research has been funded in an effort to clarify the psychosomatic nature of hypertension in the hopes of improving our understanding and treatment of this disorder. A principle belief has been that recurring blood pressure (BP) elevation caused by emotional distress eventually leads to structural vascular changes and sustained hypertension. The corollary of this view is that stress reduction techniques could ameliorate hypertension.

It is time to step back and ask the obvious question: what has been the outcome of this huge and costly body of research? Specifically, what has this research taught us about the development of hypertension and, more importantly, what has been its impact on its treatment?

The rarely mentioned conclusion is that it has had very little impact. Thousands of studies and decades of research should have been enough to provide solid evidence to support psychosomatic views of hypertension and to translate those views into treatment advances. Thus, it is long past time to either abandon psychosomatic research in hypertension or seek new directions with greater promise of clinical relevance.

The purpose of this article is to briefly summarize where psychosomatic research in hypertension has taken us to date (part I), and to explore a different agenda that offers greater promise in understanding and managing hypertension (part II).

PART I: LACK OF IMPACT OF A HALF CENTURY OF STUDIES

Traditional Psychosomatic View of Hypertension

Psychosomatic research in hypertension has focused largely on three areas (Table I): (1) BP reactivity to acute stressors; (2) the relationship between BP and measures of stress or of perceived emotional distress, particularly anger and anxiety; and (3) the antihypertensive effects of stress-reducing interventions. The outcome of this enormous body of research has previously been reviewed and is briefly summarized here.

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BP Reactivity

Studies have consistently and incontrovertibly established that laboratory stressors, and emotional distress, transiently elevate BP. It was widely believed that the magnitude of the acute BP response to a standardized laboratory stressor is predictive of future development of hypertension. However, little can be concluded given the marked inconsistency of study results and the limitations of reactivity studies (Table II).[1–5]

The meaningfulness of interpreting results of studies of BP reactivity to any laboratory stressor is greatly compromised by many factors. First, an individual's reactivity can vary considerably from one laboratory stressor to another. Second, responses vary even in response to repeated testing with the very same stressor.[6] Third, even if reactivity did predict development of hypertension, it would be unclear as to whether the increased reactivity was a cause of future hypertension or instead just a marker of arterial stiffness that presages development of hypertension. Finally, BP reactivity to what are often trivial laboratory stressors, such as mental arithmetic, bears little relationship to reactivity to the much more severe stresses encountered in the real world.[6–8]

Relationship Between Hypertension and Measures of Stress or Perceived Emotional Distress

Innumerable studies have examined the widely suspected relationship between reported emotions, such as anger and anxiety, and hypertension. Meta-analyses have concluded that perceived anger is not linked to hypertension.[9,10] If anything, the tendency to express anger (''anger-out'') might be inversely related to BP.[9] Decades of research have similarly failed to confirm the widely held belief that chronic anxiety leads to hypertension.[9,11–19] Even in patients with severe hypertension, who are rarely included in psychosomatic studies, anxiety and anger scores did not differ from those of normotensive patients.[18]

Results have been highly inconsistent from study to study and support every point of view. Given the massive number of studies performed, no future study can be expected to alter the balance of study results.

Several factors may explain the widespread, albeit unconfirmed, belief that chronic anxiety leads to hypertension. Anxiety undoubtedly increases BP, but this effect is transient. Anxiety during BP measurement is a prominent component in the white-coat phenomenon, which contributes to overdiagnosis and overtreatment[20] and also contaminates studies that claim a link between anxiety and hypertension based on casual BP readings. Awareness of the diagnosis of hypertension is also associated with an increase in subsequent BP

TABLE I. Psychosomatic Research in Hypertension: Traditional Areas of Study

Blood pressure reactivity
The relationship between blood pressure and measures of stress or of perceived emotional distress, such as anxiety or anger
The antihypertensive effects of stress-reducing interventions

TABLE II. Blood Pressure Reactivity Studies: Limitations in Interpreting Results

Reactivity to laboratory stressors is not predictive of blood pressure variability in real life
Intra-individual variation in reactivity to different laboratory stressors
Intra-individual variation in reactivity to the same stressor
Conflicting results regarding correlation of reactivity to development of hypertension
Conflicting results regarding correlation of reactivity with future cardiovascular events
Uncertainty as to whether reactivity is causally related to future events or merely a marker for arterial stiffness

readings that is not seen in patients who are unaware of the diagnosis.[21]

Another commonly held and widely studied belief is that chronic stress leads to hypertension. The balance of evidence is also not convincing in this area. Surely if chronic stress causes hypertension, it should be evident in the many studies of job stress, given how much of our time and stress are job-related. However, here, as well, study results are inconsistent and unconvincing.[5,22–25] Certainly weight gain or alcohol abuse caused by job stress can contribute to development of hypertension. However, evidence of a link between job stress per se and development of hypertension is weak and inconsistent.[25] This is powerfully illustrated in a well-designed study that found no relationship whatsoever between job strain and ambulatory BP assessed after a 5-year follow-up, even among "hot reactors."[5]

Stress Reduction and BP

The final piece of the traditional paradigm was that if stress and emotional distress led to hypertension, stress reduction techniques could ameliorate it, providing the sought payoff for decades of psychosomatic research in hypertension. Unfortunately, study results have failed to support this expectation.[26,27]

There is ample evidence and wide agreement that just as emotional stress transiently increases BP, relaxation techniques and biofeedback transiently lower it. These interventions, however, have not been proven to achieve sustained BP lowering, particularly in controlled trials and in trials that assessed ambulatory rather than casual BP.[26–29] Clinical and research interest in the role of such interventions in the management of hypertension has subsequently waned.

One review did report a large effect when a combination of techniques was employed in an individualized fashion, but it did not take into account the placebo effect.[27] Transcendental meditation was reported to lower systolic BP, but only by 3 mm Hg.[30] Isolated studies reported that group psychotherapy lowered BP, and that churchgoers have lower BP readings.[31,32]

Inconsistency of Study Results in Psychosomatic Research in Hypertension

Psychosomatic research in hypertension has been seriously hampered by the inconsistency of results from study to study. Consequently, the results of any single study are suspect, and studies can be cited to support almost any opinion.

Several problems contribute to the inconsistency of study results:

Inaccuracy of the Diagnosis of Hypertension. Many studies are compromised by the unclear hypertension status of many of its participants. In most studies, nearly all hypertensive patients have borderline or mild hypertension, categories where differentiation of hypertension and normotension is often unclear. Also, in many studies, diagnosis was based on casual rather than ambulatory BP measurement and it is likely that many "hypertensive" subjects in fact had white-coat rather than sustained hypertension.

Self-Selection Bias. In many studies, there is a self-selection bias in terms of anxiety or psychological mindedness among volunteers for studies examining the role of psychological factors and interventions. In addition, hypertensive individuals who know they have hypertension report more emotional distress than hypertensive individuals who are unaware that they have hypertension, further confounding study results.[33]

Interpretation Bias. In many reports, weak positive correlations, sometimes limited to subgroups, are emphasized, while negative data are de-emphasized or even omitted. Similarly, significant correlations involving one or two variables are often emphasized, while the absence of a correlation involving a much larger number of variables that are at least as relevant, is not.

A half century of psychosomatic research has failed to confirm the traditional belief that BP elevation caused by day-to-day stress or emotional distress leads to the development of hypertension and has had little impact on our understanding and treatment of hypertension. Acceptance of these conclusions is long overdue.

One must therefore conclude either that hypertension is not related to psychological factors beyond transient changes in BP or that psychosomatic research needs to explore new perspectives. Part II of this article will discuss such perspectives, focusing on areas that have received much less research attention and that offer the potential of considerable and important clinical relevance.

TABLE III. Psychosomatic Research in Hypertension: Overlooked Areas of Potential Clinical Relevance

Role of repressed vs consciously experienced emotion
Relevance of psychological factors to choice of antihypertensive agents
Identification of clinical phenotypes of hypertension that are likely to be of psychosomatic origin

PART II: NEW DIRECTIONS THAT COULD ENHANCE CLINICAL RELEVANCE

Part I of this paper focused on the lack of impact of psychosomatic research on our understanding or treatment of hypertension. Part II explores new areas that have barely been studied that appear to offer much greater clinical relevance to the study of psychosomatic issues in hypertension (Table III). They include (1) the role of repressed vs consciously experienced emotion, (2) the important relationship between psychological factors and selection of antihypertensive medications, and (3) identification of phenotypes of hypertension that are the most likely to be psychosomatic in origin.

ROLE OF REPRESSED VS CONSCIOUSLY EXPERIENCED EMOTION

For decades, psychosomatic research in hypertension has focused largely on consciously experienced emotional distress, such as anger and anxiety, while paying minimal attention to emotion that is not consciously experienced. Major difficulties in studying the latter are the lack of conceptual clarity, uniform terminology, or psychometric instruments for measuring emotion that is not consciously experienced.

''Repression'' is one of several terms that have been used to connote an unconscious defense against awareness of distressful emotion. Such defenses are crucial in protecting against awareness of truly overwhelming emotion. They are also employed to a greater or lesser extent in dealing with day-to-day stress.

Existing evidence supports the need for more study of the role of such unconscious defenses in the development of hypertension. Aspects that might be studied include: (1) defensive patterns of coping in which negative emotion is routinely, automatically, and unknowingly kept from awareness, and (2) repression related to prior severe emotional trauma, including childhood trauma.

Defensiveness and Repressive Coping

A ''repressive coping style'' is variably described as a lifelong tendency to minimize, or not feel, distressful emotions.[34] Individuals with a repressive coping style tend to be very even-keeled and to insist that they feel fine even when confronted by considerable stress.

Existing evidence suggests that this style of ''defensive'' or ''repressive'' coping is associated with hypertension.[35–37] Studies in which defensiveness was assessed by questionnaires such as the Marlowe-Crowne Scale of Social Desirability have reported a consistent relationship between defensiveness and hypertension.[9,18,35,38] In addition, in a study that was unique in including patients with severe hypertension, the condition was associated with defensiveness and not with perceived anger or anxiety.[18]

Further evidence is gleaned from studies that have reported counterintuitively that hypertensive individuals report less emotional distress and are more defensive than normotensive individuals.[9,35] In response to laboratory stress, such individuals reported less emotional distress yet experienced a greater increase in BP than did others.[39–41] Studies also indicate an association between hypertension and alexithymia, which is characterized by the inability to label or report emotional experience, focusing instead on factual details.[19,42,43]

The results of other studies similarly suggest that hypertensive individuals are less aware of emotional distress than are normotensive individuals. In a study of 1428 bus drivers, hypertensive drivers reported less job-related distress than did normotensive drivers.[44] Meyer also reported less emotional distress among hypertensive patients.[45] In the Alameda County Blood Pressure Study, participants who subjectively rated a given stress as milder than objective measures of that stress had the highest BP.[46]

Traumatic Life Events and Childhood Abuse and Trauma

Stress research in hypertension has largely focused on current day-to-day stress, such as job stress, marital stress, laboratory stressors, or others. The role of prior emotional trauma has received little attention.

Emotional trauma is not uncommon.[47] More than 20% of people report a history of severe abuse or trauma, particularly during childhood.[47] Childhood abuse and trauma can have considerable effects on mood and behavior decades later.[48] It is not unreasonable to suspect that it can have autonomic effects as well, although this possibility has not been widely studied.

The psychological handling of severe trauma often involves unconscious defenses, which protect against awareness of potentially overwhelming emotion.[49] As a result, many trauma survivors who employ such defenses do not subsequently report emotional distress related to the trauma.

The potential link between hypertension and past trauma, including trauma encountered during childhood, is thus often concealed, both by the long-time interval and by the absence of perceived or reported emotional distress related to it. Recent reports, however, link past trauma to hypertensive disorders and suggest that it bears relevance to treatment, as discussed below.

RELEVANCE OF PSYCHOLOGICAL FACTORS TO CHOICE OF ANTIHYPERTENSIVE AGENTS

A major goal of psychosomatic research in hypertension is improvement in the management of hypertension. As discussed in part I, studies have not found interventions such as biofeedback, stress reduction techniques, or relaxation techniques to have a persisting effect on BP.[26,27]

The role of psychotherapy in the management of hypertension has not been well studied, but is also unlikely to be helpful. Aside from the considerable commitment in terms of time and cost, there is little evidence of its benefit. Further, patients who have defensive coping styles or have repressed emotion related to prior trauma, in the absence of emotional distress, are unlikely to seek or benefit from psychotherapy.

Given the paucity of evidence that psychologically based treatment is helpful in managing hypertension, it is time to consider other directions through which psychosomatic research could lead to advances in treating hypertension. One important area that has been virtually neglected is the relationship between psychological factors and the response to different classes of antihypertensive agents. Two aspects of such research would seem highly clinically relevant: (1) the relationship between psychological factors and physiologic mechanisms underlying hypertension, and (2) identification of the phenotypes of hypertension most likely to be driven by psychological factors and determination of whether patients who fit these phenotypes respond to different antihypertensive drugs than do other patients with hypertension.

Relationship Between Psychological Factors and Physiologic Mechanisms Underlying Hypertension

Three physiologic mechanisms have been implicated in the genesis of most cases of essential hypertension and are the targets of most of the antihypertensive drugs in current use: sodium/volume, the renin-angiotensin system (RAS), and the sympathetic nervous system (SNS) (Table IV).[50] In any individual, any or all of these mechanisms may be involved, and the hypertension is most likely to be controlled if drugs that target the operative mechanism(s) are selected.

A logical question can then be asked: in individuals whose hypertension is related to psychological factors, is the underlying mechanism of the hypertension different than in other patients? And would their hypertension then respond better to drugs targeting that mechanism? If so, then identifying individuals in whom psychological factors are contributory to hypertension would be highly relevant to the drug therapy. The relationship between psychological factors and selection of antihypertensive drugs has barely been studied, but existing evidence suggests that psychological factors are relevant to drug selection.

TABLE IV. Three Main Mechanisms of Hypertension and the Antihypertensive Drugs That Target Them

Mechanism	Effective Drug Classes
Volume	Diuretics, calcium channel blockers
Renin-angiotensin system	Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, direct renin inhibitors, β-blockers
Sympathetic nervous system	β-Blockers, α-blockers, central α-agonists

In most routine cases of essential hypertension, hypertension is attributable to one or both of two mechanisms, sodium/volume and the RAS. This is evident from the many studies that have consistently shown that a drug that targets sodium/volume (a diuretic or CCB) or a drug that targets the RAS (usually an angiotensin converting-enzyme [ACE] inhibitor or an angiotensin receptor blocker [ARB]), or a combination of the two, will control hypertension in $\geq$75% of cases.[51] Although studies consistently show that SNS tone is higher in hypertensive than in normotensive populations,[52] the success of drugs that target sodium/volume and the RAS in more than 75% of cases indicates that in most cases of essential hypertension, the SNS is not a driving force.

In a minority of cases, however, it is. Such patients would be regarded as having "neurogenic hypertension."[53] Identifying individuals with neurogenic hypertension is difficult because of the absence of clinically convenient methods to measure SNS tone. In some cases, neurogenic hypertension can readily be suspected in the setting of conditions known to be associated with increased SNS tone, such as sleep apnea, alcohol abuse, and acute stroke.[54–56] In most cases, however, the presence and origin of neurogenic hypertension is less readily apparent.

The undisputed fact that emotions stimulate SNS tone suggests the possibility that psychological factors are involved in the genesis of some, if not most, cases of neurogenic hypertension. If so, then clarification of the relevant psychological factors could be useful in identifying patients with neurogenic hypertension. In such patients, drug combinations that target sodium/volume and the RAS would be less likely and drugs that target the SNS would be more likely to be effective. The important questions would then be: are psychological factors the driving force of hypertension in most patients with neurogenic hypertension, and which psychological factors are involved: perceived emotional distress, as suggested for decades, or emotions that are not consciously experienced or reported, or perhaps both?

Combined α- and β-Blockade as Drug Therapy for Neurogenic Hypertension

Whereas most antihypertensive drugs target either sodium/volume or the RAS, several agents target effects of SNS-driven hypertension. There are drawbacks to many of these agents. Reserpine is difficult to

obtain, guanethidine is no longer available, and the imidazoline receptor agonists rilmenidine and moxonidine are not available in this country. Clonidine is very effective, but side effects, particularly fatigue, dry mouth, and sexual dysfunction are a problem in perhaps a majority of patients. Therefore, the remainder of this section focuses on two remaining drug classes whose use appears to merit greater attention in the management of psychologically related hypertensive disorders, β-blockers and α-blockers.

Few studies have explored the relationship between psychological factors and responses to different classes of antihypertensive drugs. Most of the studies that have been done have focused on the effect of drug therapy on BP reactivity to stressors. There is a common misconception that β-blocker monotherapy reduces BP reactivity. Yet studies consistently show that it doesn't.[57] Neither β-blocker monotherapy, nor α-blocker monotherapy, alters the BP response to stressors.[58,59] β-Blocker monotherapy blocks β-receptor–mediated increases in heart rate and cardiac output, but the BP response is maintained instead by α-receptor–mediated vasoconstriction.[59] Similarly, α-blocker monotherapy blocks α-receptor–mediated vasoconstriction, but the BP response is maintained instead by the β-receptor–mediated increase in cardiac output.[59] Monotherapy with other drugs, such as diuretics and ACE inhibitors, also fails to reduce BP reactivity.[60,61]

In contrast, the combination of an α-blocker with a β-blocker blocks the increase in both cardiac output and peripheral resistance and reduces BP reactivity.[53,58,59] Combined α- and β-blockade has also been shown to be very effective in treating sustained hypertension,[62–64] although its efficacy cannot be attributed solely to antagonism of effects of the SNS because β-blockers also antagonize the RAS.

The use of combined α- and β-blockade would seem particularly well-suited in patients with neurogenic hypertension. The study of the latter, however, has been hampered by difficulty in identifying individuals with neurogenic hypertension, as well as by the negative attitude toward α-blockers that followed the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), which found α-blockers to be inferior as first-step therapy for hypertension.[65]

Two studies have examined the interaction between psychological factors and response to antihypertensive drugs. In one study, nonresponders to diuretic treatment had higher levels of suppressed hostility.[66] In the other, sequential monotherapy with either an ACE inhibitor or a diuretic was reported to control hypertension in 75% of patients who did not report a childhood history of abuse or trauma but in only 25% of those who did.[67] The trauma survivors responded to combined α- + β-blockade. Ironically, the best response to ACE inhibitor monotherapy was seen in patients with high anger-out scores, indicating that perceived and expressed anger is not associated with reduced responsiveness to drugs that target the RAS.[67]

Thus, studies indicate the effectiveness of the combination of an α- and a β-blocker, and support the need for further consideration of its use in the treatment of hypertension and particularly hypertension driven by the SNS, ie, neurogenic hypertension. Clonidine and reserpine also merit consideration in treating neurogenic hypertension, although clonidine is a less attractive option because of side effects and reserpine is difficult to obtain in the United States.

TABLE V. Clinical Phenotypes of Neurogenic, Psychologically Linked Hypertension

Patients with comorbidities associated with increased sympathetic nervous system tone
Unexplained severe hypertension
Essential hypertension resistant to treatment targeting volume and the renin-angiotensin system
Paroxysmal hypertension
Labile hypertension

IDENTIFICATION OF CLINICAL PHENOTYPES OF PSYCHOSOMATIC HYPERTENSION

As stated above, the responsiveness of more than 75% of hypertensive patients to treatment with drugs that target sodium/volume and/or the RAS make it likely that fewer than 25% of hypertensive patients have neurogenic hypertension. Although identification of patients with neurogenic hypertension remains difficult, a neurogenic origin can be suspected in at least 4 subgroups of patients whose hypertension does not fit the usual pattern (Table V). These subgroups would seem to be better targets for psychosomatic research than the larger number of patients with routine essential hypertension, in whom neurogenic hypertension is unlikely.

Patients With Comorbidities Known to Be Associated With Increased SNS Tone

There is ample documentation that certain comorbidities are associated with increased SNS tone. Prominent examples include alcohol abuse, the acute aftermath of stroke, and sleep apnea.[53]

Unexplained Severe Hypertension

When patients have severe hypertension, physicians search for a cause of secondary hypertension, but usually fail to uncover one.[68] In such patients, a mechanism other than the usual sodium/volume and/or RAS might explain the uncommon severity of the hypertension. Supporting the contributory role of the SNS in such patients is documentation that SNS tone, measured by muscle sympathetic nerve activity, is greater in individuals with more severe hypertension than in those with milder hypertension.[52]

Resistant Hypertension: Hypertension Resistant to Treatment That Targets Sodium/Volume and the RAS

The failure of drug combinations that target both sodium/volume and the RAS to normalize BP provides strong yet widely ignored evidence that a mechanism other than sodium/volume and the RAS is likely involved. The SNS is a logical candidate. Consistent with this, a recent study found combined α- and β-blockade to be an effective alternative in the management of resistant hypertension.[69]

Labile Hypertension and Paroxysmal Hypertension

These two forms of hypertension are characterized by episodic rather than sustained BP elevation. Both appear to be neurogenic and to have a psychosomatic origin.

Although the terms labile and paroxysmal are often used interchangeably, important differences between the two are highly relevant to clinical management.[70] Several features distinguish these two disorders and their treatment.

Paroxysmal Hypertension. The previously unexplained and difficult-to-treat syndrome of paroxysmal hypertension (pseudopheochromocytoma) provides a vivid example of both the relationship between repressed emotion and hypertension and the crucial impact of such recognition on treatment. Patients with this disorder experience unprovoked episodes of severe BP elevation, accompanied by severe physical symptoms such as headache, sweating, flushing, and chest pain.[71,72] Patients almost uniformly insist that episodes are not triggered by stress, perceived emotional distress, or panic, obscuring its psychosomatic origin.[71,72]

Although this syndrome regularly suggests the diagnosis of pheochromocytoma, a catecholamine-secreting tumor, the tumor is found in only 1% to 2% of cases. In the remaining 98%, the cause and treatment had been a persisting mystery.[73] Clear increases in catecholamines during episodes, and the presence of elevated catecholamines at other times as well, are consistent with a neurogenic mechanism.[74,75]

A recent explanation for this disorder has linked it to repressed emotion, and treatment based on that understanding has offered a pioneering and successful approach to treatment in most patients with this frequently disabling condition.[71,72] Nearly all patients acknowledge either a history of unusually severe trauma, which they insist bears no lingering emotional impact, or manifest a prominent pattern of minimizing emotional distress.[71,72] Recognition of the link between this disorder and prior trauma had been hampered both by the long interval of time and by patients' insistence that there had been no persisting emotional impact. Ironically, that insistence is itself a clue of repressed emotion.

The strongest support for the understanding of this disorder is the success achieved in treating it. Paroxysms are managed acutely with anxiolytic agents, such as alprazolam, and/or antihypertensive agents such as the α-/β-blocker labetalol, given intravenously, or oral clonidine.[71,72] Ongoing treatment with combined α- and β-blockade may reduce the magnitude of BP elevation that occurs during attacks.[71,72]

More important, antidepressant agents are usually dramatically effective in preventing paroxysms and in enabling patients to resume a normal life.[71,72] Their effectiveness provides strong evidence that this is a psychosomatic disorder even though attacks do not originate from perceived emotional distress. Finally, in some cases, a shift resulting in awareness of trauma-related emotion cures the disorder.[71,72] This outcome is not the usual, however, given the resistance to awareness of the deeply painful emotions that are being defended against.

Labile Hypertension. Although "labile" hypertension is frequently encountered in clinical practice, specific diagnostic criteria to define it have not been established or even proposed. Instead, it is characterized mainly by the subjective clinical description of BP levels that frequently vary and can be quite elevated.

Labile hypertension differs from paroxysmal hypertension in that elevated readings typically occur at moments of acknowledged emotional distress. Also, the BP elevation is often asymptomatic, although palpitations and headache, possibly tension headache or hypertensive headache, is sometimes reported.

Labile BP elevation is likely to be neurogenic, as moment-to-moment BP changes are largely controlled by SNS tone. Further, the increases in BP are usually accompanied by elevation of heart rate, a clinical clue indicative of adrenergic stimulation. Finally, measures of BP lability such as reactivity and variability are also associated with increased sympathetic tone.[76,77]

It is unclear as to whether labile BP elevation is associated with target organ damage. It would seem that the more frequent or severe or persisting the BP elevation, the more likely that it would be associated with target organ damage and the more likely that the patient would benefit from antihypertensive therapy. In addition, in worried patients, the relief provided by attaining BP control may justify treatment, even in the absence of outcomes data.

In treating labile hypertension, use of agents that target the SNS rather than sodium/volume or the RAS would seem well-suited. Clinical experience indicates that combined α- + β-blockade is often dramatically effective, although, to date, the drug therapy of this commonly seen form of hypertension has received minimal attention.

CONCLUSIONS

It is time to acknowledge that decades of psychosomatic research have failed to establish a convincing link between perceived emotional distress and

hypertension, other than the widely recognized transient increase in BP, or to advance our understanding or treatment of hypertension. A new agenda is long overdue, one that gives greater consideration to the role of repressed emotion and that focuses on specific phenotypes of hypertension that are most likely to be psychologically and neurogenically driven. Clinical relevance also mandates that greater attention be paid to the relationship between psychological factors and response to different antihypertensive drug classes and, in particular, to the role of combined α- and β-blockade.

Modification of the traditional paradigm of psychosomatic research in hypertension is long overdue. A new agenda can restore the meaningfulness and clinical relevance of psychosomatic research in hypertension. The breakthrough in treating paroxysmal hypertension dramatically testifies to the relevance of such an agenda.

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