Exploring the Comorbidity, Pathophysiology, and Integrated Treatment Strategies of Hypertension and Depression

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Abstract
Hypertension and depression are widely prevalent conditions with substantial comorbidity. Hypertension, characterized by persistently elevated arterial pressure, is associated with significant cardiovascular morbidity and mortality. Depression, a multifactorial mood disorder, adversely affects mental and physical health through mechanisms involving neuroinflammation, neurotransmitter imbalances, and hormonal dysregulation. The association between these disorders has been recognized since the mid-20th century.

Historical medical literature from ancient Chinese and Indian systems identified symptoms indicative of hypertension, later classified as a distinct disease entity by Fredrick Akbar Mahomed in the 19th century. Notable...
epidemiological studies have elucidated the significant relationship between hypertension and depression, particularly among medical students and other high-risk populations. Research indicates that common pathophysiological mechanisms, including sympathetic nervous system overactivity, chronic inflammation, and endothelial dysfunction, underlie the comorbidity of hypertension and depression. Elevated levels of norepinephrine, proinflammatory cytokines, and oxidative stress contribute to the expression or worsening of both conditions. Shared genetic and environmental factors further compound this relationship, highlighting the need for integrated treatment strategies.

Pharmacological interventions, including angiotensin-converting enzyme inhibitors and angiotensin receptor blockers, have shown potential to improve mental health outcomes in patients with comorbid hypertension and depression. However, inconsistent findings regarding the impact of antihypertensive medications on depression necessitate further investigation. The comorbidity of these conditions complicates treatment adherence, leading to poorer health outcomes and increased healthcare costs.

The clinical implications of this unidirectional or bidirectional comorbidity are considerable, affecting quality of life, treatment adherence, and overall health outcomes. Integrated care approaches, incorporating lifestyle modifications and combined pharmacological treatments, have shown promise in improving compliance and outcomes. Despite extensive research, limitations persist, including difficulties in establishing causality, inconsistent diagnostic criteria, and the influence of confounding variables. Future research should focus on clarifying the bidirectional relationship between these conditions, the impact of social determinants, and the efficacy of various treatment modalities. Identifying high-risk populations and refining intervention strategies are crucial for improving clinical outcomes in patients with comorbid hypertension and depression.

This paper aims to review the current literature regarding the association and comorbidity of hypertension and depression.

**Abbreviations:** ACE: Angiotensin-Converting Enzyme; ARBs: Angiotensin Receptor Blockers; DBP: Diastolic Blood Pressure; mm Hg: Millimeters of Mercury; QoL: Quality of Life; SBP: Systolic Blood Pressure

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**Introduction**

Hypertension and depression have been recognized for centuries, although the scientific literature focusing on the comorbidity between the two conditions is relatively recent. In ancient times, the Chinese and Indian medical systems documented symptoms of hypertension such as headaches, dizziness, and flushed face [1,2].

Later, in the 19th century, Frederick Akbar Mahomed, a pioneer in the study of hypertension, first described essential hypertension as a distinct disease entity—differentiating it from similar vascular changes and hypertension secondary to chronic glomerulonephritis. The term "essential" was used because this type of hypertension was observed in otherwise healthy individuals, particularly as they aged, and was considered a part of the normal aging process [1,3].

The association between hypertension and depression was first observed in the 1950s in studies noting mood changes in patients being treated with medications for hypertension [4,5].

Notable research, such as those by Rubio-Guerra et al., suggest that individuals experiencing depression are at a higher risk for developing hypertension and stroke [6], while another by Schaare et al. indicates that hypertension may be linked to poorer mental health and increased depressive symptoms [7]—indicating a bidirectional relationship.

Further studies have also described the higher incidence of comorbid hypertension and depression in medical students, with one from a university in Jordan finding that medical students had a higher prevalence of both conditions than nonmedical students [8]. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) have been linked to improved mental health outcomes in patients with comorbid hypertension and depression, according to several studies. However, others have found no significant relationship between the use of antihypertensive drugs and depression [9,10].

The development of novel treatments, such as esketamine nasal spray derived from ketamine, for treating depression may have potential benefits for individuals with comorbid hypertension and depression (see Supplementary Note 1) [11].

**Discussion**

Hypertension, also known as high blood pressure, is a condition in which the force of the blood against the artery walls is too high. It is a common medical condition worldwide, with an estimated global prevalence of 26% [12]. Blood pressure is measured in millimeters of mercury (mm Hg), with a normal reading being less than 120/80 mm Hg. Blood pressure consistently exceeding 130/80 mm Hg is considered hypertension (see Supplementary Note 2). Persistent high blood pressure leads to serious
health consequences, such as heart disease, kidney disease, and stroke [13]. Depression is a mood disorder that affects a person’s overall mental health, behavior, and thoughts, often resulting in feelings of sadness, hopelessness, and loss of interest in activities that an individual previously enjoyed. It may be triggered by environmental, genetic, and lifestyle factors, affecting people of all ages, genders, races, and backgrounds [14]. The prevalence of depression is increasing globally, with more than 264 million people affected worldwide [15]. Studies have shown that there is a high comorbidity rate between hypertension and clinical depression, with findings suggesting individuals with hypertension are more likely to experience depression, and those with depression are more likely to develop hypertension compared to individuals without these conditions [6,16,17]. According to a meta-analysis, the prevalence of comorbid hypertension and depression was 26.8% [17]. A cross-sectional study found that 37.8% of 416 patients with hypertension had depression symptoms [18]. Comorbid hypertension and depression are higher in women than in men [19,20]. Although the pathogenesis of hypertension and depression is complex, shared etiological factors associated with elevated levels of inflammation, endothelial dysfunction, and autonomic dysfunction play a critical role in the link between depression and hypertension [21].

**Physiology of Hypertension**

There are two types of hypertension: primary or essential hypertension and secondary hypertension. Primary hypertension is the most common type of hypertension and has no identifiable cause except for genetic and lifestyle factors, whereas underlying medical conditions or medications cause secondary hypertension [22]. Chronic hypertension affects the cardiovascular system, leading to an increased risk of heart attack, heart failure, atrial fibrillation, and stroke [23]. Hypertension causes damage to the arterial walls, resulting in the thickening and narrowing of the arterial walls, increasing the work demand on the heart, and causing cardiac hypertrophy, which can lead to heart failure and arrhythmias [23,24]. Hypertension also affects the nervous system, particularly the sympathetic nervous system, which plays a crucial role in regulating blood pressure. Hypertension can cause overactivation of the sympathetic nervous system, leading to increased levels of circulating norepinephrine, reduced vasodilation, and increased vascular resistance—resulting in elevated blood pressure levels and adverse health effects [23,25–27]. Table 1 summarizes the physiology of hypertension.

<table>
<thead>
<tr>
<th>Table 1: Hypertension Types and Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category</strong></td>
</tr>
</tbody>
</table>
| **Types of Hypertension** | Primary (Essential) Hypertension  
Most common type; No identifiable cause except for genetic and lifestyle factors |
| | Secondary Hypertension  
Caused by underlying medical conditions or medications |
| **Chronic Hypertension Effects** | **Cardiovascular System**  
- Increased risk of heart attack, heart failure, atrial fibrillation, and stroke  
- Causes damage to arterial walls, leading to their thickening and narrowing  
- Increases work demand on the heart, leading to cardiac hypertrophy, heart failure, and arrhythmias |
| | **Nervous System**  
- Affects the sympathetic nervous system, which regulates blood pressure  
- Overactivation leads to increased levels of circulating norepinephrine  
- Causes reduced vasodilation and increased vascular resistance, elevating blood pressure levels |

**Physiology of Depression**

Depression can occur at any age but is more common in adults. The cause is often complex and multifactorial, involving environmental, genetic, and lifestyle factors. However, evidence suggests that depression is primarily caused by an imbalance in brain chemicals called neurotransmitters, particularly serotonin, dopamine, and norepinephrine. Other factors that contribute to depression include neuroinflammation, hormonal dysregulation, and structural changes in the brain [28,29]. Depression affects the brain in many ways, leading to a variety of cognitive and emotional symptoms. It impairs executive function, attention, memory, mood, and...
perception. It increases amygdala activity, the brain’s emotional center, and reduces prefrontal cortex engagement, associated with decision-making and self-control [28–30]. Depression also negatively impacts the cardiovascular system. It increases the risk of developing heart disease and stroke, and it may accelerate the progression of existing cardiovascular disease. Depression is associated with autonomic dysfunction, decreased heart rate variability, and increased systemic inflammation, all of which increase the risk of developing cardiovascular disease. Depression may also worsen cardiovascular disease outcomes by reducing medication adherence, increasing healthcare costs, and affecting quality of life (QoL) [21,31,32]. Table 2 summarizes the physiology of depression.

**Table 2: Occurrence, Causes, and Effects of Depression**

<table>
<thead>
<tr>
<th>Category</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Overview of Depression</strong></td>
<td></td>
</tr>
<tr>
<td>Occurrence</td>
<td>Can occur at any age; more common in adults</td>
</tr>
<tr>
<td>Causes</td>
<td></td>
</tr>
<tr>
<td>- Complex and multifactorial: environmental, genetic, and lifestyle factors</td>
<td></td>
</tr>
<tr>
<td>- Primarily caused by an imbalance in brain chemicals (neurotransmitters: serotonin, dopamine, and norepinephrine)</td>
<td></td>
</tr>
<tr>
<td>- Other factors: neuroinflammation, hormonal dysregulation, and structural changes in the brain</td>
<td></td>
</tr>
<tr>
<td><strong>Effects of Depression</strong></td>
<td></td>
</tr>
<tr>
<td>Brain</td>
<td></td>
</tr>
<tr>
<td>- Impairs executive function, attention, and memory</td>
<td></td>
</tr>
<tr>
<td>- Affects mood and perception</td>
<td></td>
</tr>
<tr>
<td>- Increases activity in the amygdala (emotional center)</td>
<td></td>
</tr>
<tr>
<td>- Reduces engagement of the prefrontal cortex (decision-making and self-control)</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular System</td>
<td></td>
</tr>
<tr>
<td>- Increases the risk of heart disease and stroke</td>
<td></td>
</tr>
<tr>
<td>- May accelerate the progression of existing cardiovascular disease</td>
<td></td>
</tr>
<tr>
<td>- Associated with autonomic dysfunction, decreased heart rate variability, and increased systemic inflammation</td>
<td></td>
</tr>
<tr>
<td>- Can worsen cardiovascular disease outcomes: reduced medication adherence, increased healthcare costs, and affected QoL</td>
<td></td>
</tr>
</tbody>
</table>

**Link Between Hypertension and Depression**

Previous studies suggest that there is a high comorbidity rate between hypertension and depression. According to Li et al. and Asmare et al., the prevalence of comorbid hypertension and depression is 26.8% and 37.8% in the adult population, respectively—depending on the survey instrument and diagnostic criteria used for diagnosing hypertension and depression [17,18]. Hypertension is associated with an increased risk of developing depression, and individuals with hypertension may be at higher risk of developing depression compared to those without hypertension [7,16]. In contrast, depression is also associated with an increased risk of developing hypertension [6]. The coexistence of depression and hypertension may increase the likelihood of adverse cardiovascular events, such as myocardial infarction, stroke, and congestive heart failure [21]. The association between hypertension and depression was particularly prominent in individuals who had a family history of hypertension and were overweight or obese [7,33].

**Mechanisms of Hypertension and Depression Comorbidity**

Studies suggest that there are various mechanisms underlying the association between hypertension and depression. Table 3 illustrates some of the proposed mechanisms.
Comorbid hypertension and depression affect

Clinical Implications of Hypertension and Depression Comorbidity

Hypertension and depression comorbidity have significant clinical implications. The coexistence of these two conditions can have a substantial effect on an individual's QoL, including negatively impacting their functional status, cognitive abilities, and occupational productivity, leading to an increased risk of suicide [36,37].

Comorbid hypertension and depression also affect treatment adherence, leading to worse overall health outcomes. Studies have shown that individuals with comorbid hypertension and depression are more likely to be nonadherent with their antihypertensive and antidepressant medications, resulting in poor symptom control and increased healthcare costs [21,31,32].

Management of comorbid hypertension and depression should include lifestyle modifications, such as exercise and dietary interventions, as well as psychological and pharmacological approaches targeting both conditions [38,39].

Various studies have demonstrated the effectiveness of integrating depression treatment into care for hypertension, which has been associated with improved adherence to both antidepressant and antihypertensive medications, leading to better symptom control, reduced healthcare costs, and improved QoL [40,41].

Antihypertensive medications, such as angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs), have been associated with improved mental health outcomes in patients with comorbid hypertension and depression [9]. However, the choice and dose of medications should be tailored to the individual patient's needs, with consideration given to potential drug interactions and side effects.

Plain Language Summary

Hypertension (high blood pressure) and depression have been known for a long time, but research linking the two is more recent. Ancient Chinese and Indian medical systems noted hypertension symptoms like headaches and dizziness. In the 19th century, Frederick Akbar Mahomed identified hypertension as a distinct disease [1-3].

The link between hypertension and depression was first observed in the 1950s [4,5]. Since then, further studies have explored this connection, with results noting a bidirectional relationship [6-8].

Some blood pressure medications, like ACE inhibitors and ARBs, may improve mental health in people with both conditions, though findings are mixed [9,10]. Recently, new treatments for depression, such as esketamine nasal spray, may benefit those with both hypertension and depression [11].

Hypertension occurs when the force of blood against artery walls is too high. Hypertension affects about 26% of the global population [12]. Normal blood pressure is below 120/80 mm Hg, while hypertension is above 130/80 mm Hg. Chronic high blood pressure can lead to serious health issues like heart disease, kidney disease, and stroke [13].

Depression is a mood disorder causing sadness, hopelessness, and loss of interest in activities. It can be triggered by environmental, genetic, and lifestyle factors and affects over 264 million people worldwide [14,15]. Studies show that people with hypertension are more likely to experience depression and vice versa [6,16,17]. Women are more affected by the comorbidity of these conditions than men, with the
varying prevalence of comorbid hypertension-depression of 26.8% and 37.8% [17-20].

There are two types of hypertension: primary or essential (common, with no apparent cause) and secondary (caused by other medical conditions) [22]. Chronic hypertension affects the heart and blood vessels, increasing the risk of heart attack, heart failure, and stroke. It damages artery walls and overworks the heart. It also affects the nervous system, particularly the sympathetic nervous system, which controls blood pressure [23-27].

Depression can happen at any age but is more common in adults. It involves an imbalance of brain chemicals like serotonin, dopamine, and norepinephrine. Depression impacts the brain, causing problems with thinking, memory, and mood [28-30]. It also affects the cardiovascular system, increasing the risk of heart disease and stroke and possibly worsening existing heart conditions [21,31,32].

Hypertension and depression often occur together. Hypertension increases the risk of depression, and depression increases the risk of hypertension [6,7,16]. This combination can lead to more severe health issues, such as heart attacks and strokes, especially in people with a family history of hypertension or who are overweight [7,21,33].

- Both conditions involve increased activity in the sympathetic nervous system, which can damage the heart and other organs [23,25-27].
- Chronic inflammation is shared in both conditions, causing oxidative stress and artery damage [34,35].
- Both conditions can damage the lining of blood vessels, reducing their ability to dilate and increasing blood pressure [23,35].
- Shared genetic and environmental factors, such as obesity and poor lifestyle habits, increase the risk of both conditions [22,28,29].

The combination of hypertension and depression significantly affects QoL, leading to poor health outcomes and increased healthcare costs [21,31,32]. Treating both conditions together, including lifestyle changes and medications, can improve adherence to treatment and overall health. Integrating depression treatment into hypertension care has shown benefits, such as better symptom control and reduced healthcare costs [38-41]. Antihypertensive medications may also help with mental health, but treatment should be personalized to avoid side effects and drug interactions [9].

Limitations of Research
The association and comorbidity between hypertension and depression have been extensively studied in recent years. Despite the growing body of literature on this topic, several research limitations should be noted.

One of the primary limitations is the difficulty in establishing a cause-and-effect relationship between hypertension and depression. Some studies have shown that hypertension may increase the risk of developing depression or worsen depressive symptoms, while others suggest that depression may lead to the development of hypertension [6,7]. It remains unclear whether the comorbidity of hypertension and depression is due to a common underlying pathophysiology or if the two conditions are independent but mutually interacting [9,16,24,42,43].

Another limitation is the lack of consistency in the diagnostic criteria used for hypertension and depression, making it difficult to compare results across studies. Also, most studies focused on individuals with hypertension or depression, which may not represent the general population [6,24].

These studies did not account for the potential confounding effects of other variables, such as age, race, sex, and body mass index, which may influence the association between hypertension and depression. Therefore, further research is needed to evaluate the directionality and underlying mechanisms of the association between hypertension and depression, both for diagnosis and interventions [7,9,42,43].

Future Research
Future research regarding the association and comorbidity of hypertension and depression should address some of the limitations of previous studies. One area of focus should be the elucidation of the bidirectional relationship between hypertension and depression, including the underlying mechanisms and causality [36,44,45].

Future research should also explore the impact of social determinants of health, such as education, income, and access to healthcare, on the association between hypertension and depression. Additionally, studies should examine the effectiveness of different treatments for the comorbidity of hypertension and depression, such as pharmacological, psychological, and lifestyle interventions [7,9,42].

Further investigation is also needed to determine whether the use of antihypertensive drugs contributes to the development of depression or whether they improve mental health outcomes, as the results are mixed [7,9,42].

Research should focus on identifying the populations most at risk for the comorbidity of hypertension and depression, such as medical students and geographically diverse populations, to improve intervention strategies and treatment outcomes [8,42].

Conclusion
The comorbidity between hypertension and depression presents significant clinical challenges due to their complex and interrelated pathophysiological mechanisms. Hypertension, characterized by elevated
blood pressure levels, leads to cardiovascular complications and autonomic dysfunction. Depression, a multifactorial mood disorder, is linked to neurotransmitter imbalances, neuroinflammation, and hormonal dysregulation, adversely affecting both mental and physical health. The high prevalence of concurrent hypertension and depression underscores the importance of recognizing their intertwined nature, driven by shared etiological factors such as sympathetic nervous system overactivity, inflammatory responses, endothelial dysfunction, and common genetic and environmental influences. The bidirectional relationship between these conditions exacerbates their impact on patient outcomes, including increased risks of myocardial infarction, stroke, and heart failure. Furthermore, the comorbidity impairs medication adherence and increases healthcare costs, necessitating integrated management approaches. Effective treatment—comprising lifestyle modifications and pharmacological interventions targeting both conditions and psychological support—improves adherence and overall health outcomes. Despite extensive research, limitations persist in understanding the precise causal pathways and interactions between hypertension and depression. Inconsistent diagnostic criteria and insufficient consideration of confounding variables complicate comparative analyses. Future research should focus on elucidating the bidirectional relationship, the role of social determinants, and the effectiveness of integrated treatment strategies. Addressing these gaps will enhance the ability to mitigate the impact of these comorbid conditions and improve the quality of life for individuals affected.

**Conflict of Interest Statement**

The authors declare that this paper was written without any commercial or financial relationship that could be construed as a potential conflict of interest.

**Supplementary Note 1**

**Esketamine and Ketamine Nasal Spray: Comparative Efficacy and Side Effect Profiles**

Esketamine nasal spray and ketamine nasal spray are both medications used for the treatment of depression, particularly treatment-resistant depression [46]. However, they contain different forms of the same compound, ketamine, and have some differences in their formulation, approval, and clinical use.

**Esketamine Nasal Spray** [11,47]:
- Composition: Esketamine is the S-enantiomer of ketamine, which is the more potent form responsible for the medication’s antidepressant effects.
- Formulation: Esketamine nasal spray contains only the S-enantiomer of ketamine, which is thought to have a more favorable side effect profile than the racemic mixture of ketamine.
- FDA Approval: Esketamine nasal spray, sold under the brand name Spravato, received FDA approval in 2019 specifically for the treatment of treatment-resistant depression in adults, stipulating concurrent use with an oral antidepressant as part of a comprehensive treatment regimen.
- Clinical Use: Esketamine nasal spray is administered under medical supervision in a healthcare setting due to its potential for side effects and abuse.
- Dosage: The dosage and administration schedule are determined by a healthcare professional and typically involve a gradual titration to find the optimal dose for each patient.

**Ketamine Nasal Spray** [48,49]:
- Composition: Ketamine nasal spray contains a racemic mixture of both the R- and S-enantiomers of ketamine.
- Formulation: Ketamine nasal spray contains both the R- and S-enantiomers of ketamine, which may lead to a different side effect profile than esketamine.
- Regulatory Status: Ketamine nasal spray is not typically available as a commercial product with specific FDA approval for the treatment of depression.
- Clinical Use: Ketamine nasal spray may be used off-label in some cases for the treatment of depression, often administered in specialized clinics or under the supervision of a psychiatrist.
- Dosage: The dosage and administration of ketamine nasal spray may vary and are typically determined based on clinical judgment and patient response.

In summary, esketamine nasal spray contains only the S-enantiomer of ketamine and has specific FDA approval for the treatment of treatment-resistant depression. Whereas ketamine nasal spray contains both the R- and S-enantiomers and is not typically approved for depression treatment but may be used off-label in some cases (Table 4).

<table>
<thead>
<tr>
<th>Table 4. Comparing Esketamine and Ketamine Nasal Spray</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aspect</strong></td>
</tr>
<tr>
<td>Composition</td>
</tr>
</tbody>
</table>
Both medications have shown promise in the treatment of depression, particularly in individuals who have not responded to traditional antidepressant medications. However, they are administered under medical supervision due to their potential for side effects and abuse.

**Supplementary Note 2**

**Relevance of Systolic and Diastolic Blood Pressure in Hypertension and Its Implications for Older and Younger Individuals**

Systolic and diastolic blood pressure are critical in diagnosing and managing hypertension; however, they represent different phases of the cardiac cycle and have distinct implications for health.

**Systolic Blood Pressure (SBP)** [13,22,50]:
- **Definition:** SBP is the pressure in the arteries when the heart contracts (beats) and pumps blood into the body’s arteries.
- **Measurement:** It is the higher of the two numbers in a blood pressure reading (e.g., in 120/80 mmHg, 120 is the systolic pressure).
- **Implications in Hypertension:**
  1. **Primary Indicator:** Elevated SBP is a primary hypertension indicator, particularly in older adults.
  2. **Isolated Systolic Hypertension (ISH):** Common in older adults, ISH occurs when only the systolic pressure is elevated (≥140 mmHg) while diastolic pressure remains normal (<90 mmHg).
  3. **Risk Factors:** High systolic pressure is strongly associated with increased risks of adverse cardiovascular events such as stroke, heart attack, and kidney damage.

**Diastolic Blood Pressure (DBP)** [13,22,51]:
- **Definition:** DBP is the pressure in the arteries when the heart rests between beats and refills with blood.
- **Measurement:** It is the lower of the two numbers in a blood pressure reading (e.g., in 120/80 mmHg, 80 is the diastolic pressure).
- **Implications in Hypertension:**
  1. **Secondary Indicator:** While diastolic pressure is also crucial, it is typically a more significant indicator of hypertension in younger individuals.
  2. **Isolated Diastolic Hypertension (IDH):** This occurs when only the diastolic pressure is elevated (≥90 mmHg), with normal systolic pressure.
  3. **Risk Factors:** High diastolic pressure can indicate a risk for certain cardiovascular conditions, although its impact on overall cardiovascular risk is generally less pronounced than that of systolic pressure, especially in older adults.

Table 5 summarizes the differences between systolic and diastolic blood pressure regarding hypertension.

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Systolic Blood Pressure</th>
<th>Diastolic Blood Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase of Cardiac Cycle</td>
<td>Heart contraction (systole)</td>
<td>Heart relaxation (diastole)</td>
</tr>
<tr>
<td>Position in BP Reading</td>
<td>Higher number (e.g., 120 in 120/80 mmHg)</td>
<td>Lower number (e.g., 80 in 120/80 mmHg)</td>
</tr>
<tr>
<td>Hypertension Indicator</td>
<td>Primary indicator, especially in older adults</td>
<td>Important, particularly in younger individuals</td>
</tr>
<tr>
<td>Common Conditions</td>
<td>Isolated systolic hypertension (ISH)</td>
<td>Isolated diastolic hypertension (IDH)</td>
</tr>
<tr>
<td>Risk Implications</td>
<td>Strongly associated with increased risk of stroke, heart attack, and kidney damage</td>
<td>Associated with risk for cardiovascular conditions, but less than systolic pressure in older adults</td>
</tr>
<tr>
<td>Age Relevance</td>
<td>More critical in older adults due to arterial stiffness</td>
<td>More critical in younger individuals</td>
</tr>
</tbody>
</table>

SBP is more critical in older adults, strongly associated with cardiovascular risks, and serves as a primary hypertension indicator. Whereas DBP is more critical in younger individuals, remains consequential for overall...
cardiovascular health, and acts as a secondary hypertension indicator [52]. Both systolic and diastolic pressures are essential for a comprehensive assessment of hypertension and its potential health impacts. 

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