

Vital Signs in Older Patients: Age-Related Changes

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Vital signs are objective measures of physiological function that are used to monitor acute and chronic disease and thus serve as a basic communication tool about patient status. The purpose of this analysis was to review age-related changes of traditional vital signs (blood pressure, pulse, respiratory rate, and temperature) with a focus on age-related molecular changes, organ system changes, systemic changes, and altered compensation to stressors. The review found that numerous physiological and pathological changes may occur with age and alter vital signs. These changes tend to reduce the ability of organ systems to adapt to physiological stressors, particularly in frail older patients. Because of the diversity of age-related physiological changes and comorbidities

in an individual, single-point measurements of vital signs have less sensitivity in detecting disease processes. However, serial vital sign assessments may have increased sensitivity, especially when viewed in the context of individualized reference ranges. Vital sign change with age may be subtle because of reduced physiological ranges. However, change from an individual reference range may indicate important warning signs and thus may require additional evaluation to understand potential underlying pathological processes. As a result, individualized reference ranges may provide improved sensitivity in frail, older patients. (*J Am Med Dir Assoc* 2011; 12: 337–343)

Keywords: Aged; vital sign; pulse; blood pressure

The 4 traditional vital signs—pulse, temperature, blood pressure, and respiratory rate—are objective measurements of vital function¹ and thus constitute a fundamental component of the physical examination and nursing assessment. Dysregulated organ system function as a result of age or age-associated pathophysiology, coupled with age-related loss of protective homeostatic mechanisms, suggests that among older patients vital sign response may not only deviate from normal ranges, but also remain confined to a range of values, unable to respond appropriately to stressors. Thus, health care professionals should pay special attention to vital signs in the elderly and perhaps expand the observation beyond the traditional vital signs in the frailest older patients.

Vital signs are noninvasively measured using simple equipment (ie, thermometer, sphygmomanometer, watch). Provided that the equipment is well calibrated and that the

assessor is well trained, vital signs are a reliable measurement (eg, 2 measurements should be highly correlated). Additionally, the vital sign produced is an objective measurement of physiologic function (ie, it is measured, not surmised). Combined with the consistent scales on which vital signs are measured and the well-established normative ranges, vital signs serve as a universal communication tool for patient status and severity of illness. Standardized methods of collecting vital signs enables information collected in one place and at one particular time to be useful in any other place, at any other time. Gaps in clinical information, including missing information about a patient's baseline and change from baseline, are common during the transfer of older adults from senior residences or nursing homes to emergency departments.² Improving continuity of care during transfer led to The Joint Commission including as one of its 2006 National Patient Safety Goals “the implementation of a “standardized approach to ‘handoff’ communications.”³ In this way, standardized tools, including vital signs, are important for longitudinal monitoring, continuity of care, and improved communication among health care professionals—factors particularly significant in the long-term care setting.

In an acute setting, vital signs are considered a marker of underlying pathology (sensitivity) and can alert the physician and physician extender to a disease process and severity, but they do not define which disease process is ongoing (specificity). For example, an abnormal temperature signifies an underlying process that needs additional investigation, but not

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the type of process, location, or cause. Using established normal ranges for each vital sign can help clinicians and health professionals quantify abnormal findings. When measured serially, vital signs are a monitoring tool that can signify both the progression of a disease process and the effectiveness of treatment. For example, a change in systolic blood pressure from 120 mm Hg to 90 mm Hg might mark increasing sepsis or the effect of a treatment. This article describes the effect of age, particularly in frail older patients, on the physiologic range of vital signs and the associated need for individualized vital sign ranges and serial measurement to capture abnormalities.

Because of the tendency to reduce homeostatic mechanisms with age, difficulty maintaining internal consistency prevents the optimal function of the body and hinders its ability to respond to specific homeostatic challenges.⁴ In nursing home patients, this trend may be especially pronounced because of advancing physiologic and functional decline. This can manifest itself as confinement of vital signs to individual ranges, thus reducing sensitivity (eg, elderly patients are less likely than the general population to mount a high fever response to infection), while successive vital sign assessments in an individual are more sensitive to change (eg, if an individual's blood pressure is consistently measured for years at 200/80, a sudden change to 120/80 could signify a serious adverse event). Thus, the clinician becomes less dependent on normative ranges for the overall population and more dependent on normal ranges for the specific older patient. As a result, successive vital sign measurements for an individual are *more sensitive* to change because a discrepant vital sign indicates that an insult is significant enough to exceed the threshold of the confined range for the patient.

The purpose of this article is to systematically review vital signs (blood pressure, pulse, respiratory rate, and temperature) in older patients, particularly nursing home patients, with a focus on the impact of (1) molecular changes, (2) organ system changes, (3) systemic changes, and (4) altered compensation to stressors. For each system, we investigated mechanisms of underlying age-related disease processes. We then explored how these aging-related changes in turn affect the vital signs.

METHODS

A systematic approach was used to select sources for this review. A literature search was conducted using PubMed and the Medline online encyclopedia. The Medical Subject Headings terms "blood pressure," "respiration," "pulse," and "body temperature" combined with "aged" were used. Additional keyword searches included the terms "vital signs," "temperature," "fever," "thermoregulation," "respiratory rate," and "heart rate" merged with "aged." Abstracts were reviewed if they were published in English and studied human subjects. Articles were reviewed if they pertained to the topic and had a study population composed of adults aged 60 years and older. Review articles were included if they were pertinent to the focus of this paper. Applicable articles were cross-referenced to find further relevant sources.

AGING OF SYSTEMS

The Cardiovascular System—Blood Pressure and Pulse

The cumulative effect of age-related molecular changes results in cardiovascular vital signs (blood pressure, pulse) with altered sensitivity, reliability, and normative ranges. On a cellular level, the endothelial aging process has been postulated to be a result of a combination of accumulating oxidative stress (ie, decreased nitric oxide and increased cyclooxygenase production)^{5,6} and increased production of elastases and metalloproteinases in vascular smooth muscle that degrade the endothelial basement membrane. These insults initiate a wound-injury response that releases growth factors and collagen. Ultimately, these factors contribute to increased arterial wall stiffness with advancing age,^{7,8} which accelerates the development of atherosclerosis and hypertension.⁹ The resultant damage initiates structural changes that further reduce the pliability of the arterial wall.¹⁰

Although the precise mechanism for arterial wall stiffness remains to be elucidated, the clinical significance on blood pressure is important. Advancing arterial stiffness requires higher systolic pressures to achieve forward flow, which increases the heart workload. Within the ventricle, there is an enlargement of myocyte size, a decrease in myocyte number, and an accumulation of collagen in the extracellular space that contributes to increasing left ventricular wall thickness with age.¹¹ The advancing cardiac wall thickness perpetuates diastolic dysfunction and is associated with decreased cardiac filling and increased myocardial oxygen demands. Although diastolic blood pressure is not necessarily affected, the result is a wider gap between systolic and diastolic blood pressures.

Although the prevalence of hypertension increases with age (Table 1), older patients are also at greater risk for experiencing the other extreme, hypotension, which is likely related to a reduction in the aging cardiovascular system's ability to respond appropriately and rapidly to stressors. A 2009 review on age-associated loss of cardioprotection mechanisms found that aside from structural changes to aging cells, changes in intracellular protein expression with age provided a partial explanation for the loss of cardioprotective molecules in animal models.¹² For example, older patients exhibit a decline of autonomic sensitivity. With aging, increasing levels of circulating catecholamines increase the number of sympathetic receptors, which is associated with a desensitization of the receptor and a disruption of intracellular signaling.¹³

The impact of this dysregulated signaling can lead to orthostatic hypotension, such that the body is unable to rapidly modulate blood pressure to compensate for postural changes. Orthostasis is common in older patients, occurring in 30% of older outpatients and in up to 50% of nursing home residents.^{14,15} Clinical manifestations include cognitive disturbances, dizziness, syncope, hospitalizations, and falls.^{16,17} In long-term care residents, the number of hypotension-related hospitalizations rises exponentially.¹⁸ Orthostasis can be caused by a variety of factors including

Table 1. *Increasing Blood Pressure with Age*

Age, y	% of Females with Hypertension	% of Males with Hypertension
55–64	54%	45%
65–74	73%	59%
≥75	83%	68%

(National Center for Health Statistics. Health, United States, 2004 With Chartbook on Trends in the Health of Americans. Hyattsville, Maryland: 2004.)

medications, diseases, and autonomic dysregulation¹⁹; thus, an individualized assessment for orthostasis is necessary in the older patient.

In most patients, pulse can be measured via palpation, although it can be limited by vascular stiffness and atherosclerosis in older patients. An important point is that pulse is a reflection of ventricular contraction, but may not always reflect this (for example, patients with tachyarrhythmia). Heart rate is a reflection of both sympathetic and parasympathetic control. In general, maximal heart rate falls with increasing age because of increased interstitial sympathetic neurotransmitter and resultant down-regulation of beta-1 receptor activity, which decreases sympathetic nervous system intracellular signaling and responsiveness.²⁰ Resting heart rate, in contrast, is often observed to increase with age owing to deconditioning and autonomic dysregulation.²¹ Heart rate variability, ie, the ability to modulate heart rate as a compensatory mechanism to outside stressors such as exercise, is negatively correlated with age.²² Thus there is a constriction of physiological range that may mask underlying systemic disease.²³ Disrupted heart rate variability is associated with adverse outcomes and poor prognosis for many diseases, including myocardial infarction,²⁴ sepsis,²⁵ and congestive heart failure.²⁶

Both pulse and blood pressure can be influenced by common age-related pathologies, including atherosclerosis, hypertension, and arrhythmias. Atherosclerotic disease can further increase an individual's pulse pressure, which in concert with an elevated resting heart rate results in mechanical stress and damage to the endothelium. Ultimately, the stress response further stimulates atherosclerosis. Damage as a result of atherosclerotic change can reduce the pliability of the arteries, contributing to the development of hypertension and the observation of increasing blood pressure with age. An overview of the impact of age-related changes on the vital signs is presented in Table 2.

The Respiratory System—Respiratory Rate

Age-related physiological changes to the respiratory system affect the chest wall, the shape of the diaphragm, and the lung parenchyma itself. At the molecular level, accumulation of reactive free radicals with age exerts similar damage to the pulmonary and cardiovascular systems, with resultant damage to DNA and eventually cellular senescence. Increased release of proteinases causes degradation of collagen and remodeling of vasculature, which can appear in normal aging to resemble a constant state of inflammation.²⁷ Functional consequences of the aged lung include decreased responsiveness to chemoreceptors and mechanoreceptors,²⁸ with one study determining

that older patients had a 50% to 60% decline in response to hypoxia and hypercapnia.²⁹ Oxidative damage and the stress response also contribute to increased production of elastases, which degrade elastic tissue within the lung, resulting in impaired elastic recoil and dilation of the airspaces.³⁰

As aging progresses, body shape undergoes changes that can compress the thorax. Muscle stiffness, osteoporosis, and calcification of costal cartilage increase kyphosis severity and ultimately result in a loss of chest wall compliance and reduced diaphragmatic efficiency.³¹

The decline in elastic recoil, coupled with the decline in chest wall compliance, have functional implications: increased work of breathing and decreased physiologic reserve. This largely results from decreased tidal volumes and increased residual volumes. Because minute ventilations are similar in the young and old, older adults may have an increased respiratory rate to compensate for the decrease in tidal volume.³² Although this may not affect day-to-day activities, it does reduce physiological reserve. For example, patients may be able to walk on a level surface, but inclines or stairs increase the oxygen needs and the body is unable to compensate.

Respiratory rate is easy to measure, requiring only observation and a watch with a second hand. Nevertheless, recent studies report that among the 4 standard vital signs, respiratory rate is least often recorded by health care professionals.^{33–35} A study using focus groups to understand why nurses frequently neglect to measure and document respiratory rate found a variety of explanations: increased nursing workload, lack of emphasis on measuring respiratory rate during nursing training, difficulty counting breaths without the patient being aware and changing breathing, lack of understanding about the importance of respiratory rate, and lack of electronic equipment for measuring respiratory rate.³⁶ Because high respiratory rates (>27 breaths per minute) have been shown to have a high predictive value for serious adverse events, including cardiac arrest in hospital patients,³⁷ respiratory rates may be more sensitive than pulse or blood pressure in determining critically ill patients.^{38,39} Thus, respiratory rate measurement signifies an important component of vital sign assessment in older patients.

The Thermoregulatory System—Temperature

Temperature is a vital sign influenced by the thermoregulatory and immune systems, both of which undergo changes with aging. Older adults commonly have lower core body temperature⁴⁰ and altered thermoregulatory responses.⁴¹ Although the mechanisms for these findings are not fully elucidated, various explanations have been presented, including reduced subcutaneous fat acting as insulation,⁴² loss of peripheral vasoconstriction capacity,^{43,44} possible decreased cardiac output with resultant decrease in blood flow to the extremities, and decreased muscle mass resulting in reduced heat production capacity.⁴⁰

Circadian fluctuations affecting body temperature are less predictable in older patients. Circadian rhythm is largely regulated by the hypothalamic-pituitary axis. Age-related structural changes in hypothalamic mineralocorticoid receptors

Table 2. Summary of Vital Sign Changes with Aging

Age-related Mechanisms of VS Change	Blood Pressure	Pulse	Respiratory Rate	Temperature
Molecular	<ul style="list-style-type: none"> • Oxidative and mechanical damage to vascular endothelium • Heightened inflammatory response: <ul style="list-style-type: none"> ◦ cytokines ◦ growth factors ◦ collagen ◦ elastases and metalloproteinases • Decreased arterial wall pliability 	<ul style="list-style-type: none"> • Desensitization of sympathetic receptors disrupts intracellular signaling 	<ul style="list-style-type: none"> • Increased elastases <ul style="list-style-type: none"> ◦ degrade elastic tissue ◦ reduce compliance ◦ cause dilation of airspaces 	<ul style="list-style-type: none"> • Decreased T-cell function owing to prolonged antigenic exposure • Less apoptosis in aged-resistant T cells • Structural changes in hypothalamic mineralocorticoid receptors cause hypothalamic-pituitary axis hyperactivity
Structural/ Organ	<ul style="list-style-type: none"> • Increased left ventricular wall thickness • Diastolic dysfunction owing to: <ul style="list-style-type: none"> ◦ increased wall thickness ◦ less cardiac filling 	<ul style="list-style-type: none"> • Temporarily decreased perfusion owing to: <ul style="list-style-type: none"> ◦ decreased baroreflex sensitivity ◦ delayed reaction 	<ul style="list-style-type: none"> • Altered chest wall shape owing to: <ul style="list-style-type: none"> ◦ kyphosis ◦ osteoporosis ◦ costal cartilage calcification • Increased work of breathing: <ul style="list-style-type: none"> ◦ altered diaphragm shape ◦ decreased compliance 	<ul style="list-style-type: none"> • Dysfunctional/deficient hypothalamic mineralocorticoid receptors increase nighttime cortisol levels • Hypothalamic suprachiasmatic nucleus hyperactivity
Systemic	<ul style="list-style-type: none"> • Arterial stiffness requires higher systolic pressures to pump blood • Increased pulse pressure 	<ul style="list-style-type: none"> • Altered cardiac output and increased resting heart rate • Maximum heart rate is more limited with age 	<ul style="list-style-type: none"> • Less elastic recoil & chest wall compliance results in air trapping, thus increased residual volumes & decreased tidal volumes • Increased respiratory rate compensates for less tidal volume 	<ul style="list-style-type: none"> • Reduced ability to maintain body heat owing to: <ul style="list-style-type: none"> ◦ less subcutaneous fat ◦ reduced peripheral vasoconstriction ◦ decreased cardiac output ◦ dysregulated circadian rhythm ◦ loss of muscle mass
Compensation to stress	<ul style="list-style-type: none"> • Reduction in endogenous cellular repair capability owing to damaged cardiomyocytes & vascular endothelium • Altered intracellular protein expression • Mitochondrial aging & changes in signal transduction cascades • Loss of responsiveness to sympathetic stimuli 	<ul style="list-style-type: none"> • Less sympathetic responsiveness hinders ability of cardiovascular system to adjust when stimulated • Less adaptability in heart rate is associated with falls, frailty 	<ul style="list-style-type: none"> • Weakened respiratory muscles, less compliant chest wall, & increased work of breathing diminish ability to adapt to stress • Less sensitivity of chemoreceptors & mechanoreceptors causes decreased response to hypoxia & hypercapnia 	<ul style="list-style-type: none"> • Loss of heat maintenance & thermogenesis mechanisms • Heightened vulnerability to hot and cold stressors • Lower core body temperature hinders ability to regulate body temperature

have been implicated as a cause for hypothalamic-pituitary axis hyperactivity and increased nighttime cortisol levels in normal aging.⁴⁵ Additionally, the depletion of melatonin may add to circadian temperature disruption.⁴⁶ These factors contribute to the dysregulated circadian rhythm often observed in the elderly. Studies have found that healthy older individuals experience altered nocturnal temperatures.^{47,48} Dementia has been shown to disrupt circadian rhythms, which can interfere with normal thermoregulation.^{49,50} Pathophysiology in any of the regulatory mechanisms further restricts thermogenesis and heat dissipation mechanisms, rendering the elderly more vulnerable to hot and cold stressors.⁵¹

Body temperature elevation represents an important immunologic tool in combating microbial infection. Advancing age leads to changes in the immune system, eg, blunting of adaptive immunity.⁵² In particular, T-cell function is observed to deteriorate with age, perhaps because of prolonged antigenic exposure throughout a lifetime, which results in a population of aged T cells resistant to apoptosis. Fever has been postulated to interfere with microbial survival and more significantly may support host defense mechanisms in combating infection.⁵³ Reduction in the capability to mount a fever response, owing to impaired temperature regulation and age-associated changes to the immune system, can thus have deleterious results in older patients.

Limitation in body temperature regulation in the geriatric population means that a subtle change in temperature may become more significant. Because older patients are less able to mount a fever response, a slight increase from their baseline temperature may signify an underlying disorder. One study advocates the importance of establishing an older individual's basal body temperature so that elevations to this temperature are noted as a fever, because among nursing home residents, body temperature rarely exceeds 101°F.⁵⁴ For the oldest-old, mean oral temperature has been observed to be even lower than for the general geriatric population, further reinforcing the association between age and lower body temperature.^{55,56} Therefore, taking into account age and establishing individual reference ranges can help health care professionals determine the significance of an elevated temperature in a particular older patient. In elderly patients, temperature has been observed to preempt other vitals as a preliminary sign of a problem.⁵⁷ Even subtle variation from the core body temperature can be a significant finding, as fever in an older patient often indicates a more serious

infection and is associated with increased rates of life-threatening consequences.⁵⁸

Beyond the Traditional 4: Other Proposed Vital Signs

A literature search of vital signs illustrates that there have been recent calls to adopt various additional vital signs, including for example, smoking status, health literacy, body mass index, and oxygen saturation, among many other proposals. Of these, pain and mental status represent important future directions in the adoption of new vital signs.

Pain

In recent years, pain assessment using a numeric rating scale from 0 to 10 has gained popularity as an important screening tool in a variety of health care settings, and has been widely referred to as the “fifth vital sign.”^{59–61} Between 25% and 50% of community-dwelling seniors and 45% and 80% of long-term care residents report that they commonly experience significant, undertreated, and often disabling daily pain.^{62,63} These data have encouraged the adoption of self-reported pain assessment as a vital sign, which will encourage systematic observation and hopefully better treatment of a common, costly, and debilitating ailment. Critics maintain that pain is problematic to include as a vital sign because it cannot be observed objectively.⁶⁴

Mental Status

Although the established vital signs provide important information about the cardiovascular, immune, and respiratory systems, the current vital signs fail to provide insight into a patient's cognitive function and mental status. Such an omission results in a significant lack of knowledge about a patient's overall state of health. Particularly in an elderly population, acute mental status changes are routinely missed by health care professionals, with deleterious results.^{65,66} A vital sign that can quickly and objectively assess mental status has been recently proposed.⁶⁷ Such a sign should be able to establish a patient's baseline, detect acute changes that can result from delirium or trauma, and monitor chronic cognitive impairments such as dementia. A mental status vital sign could have great utility in evaluating mental status change in a systematic way.

STUDY STRENGTHS/LIMITATIONS

Although there is a large body of literature describing the aging of systems, the number of studies that address the

Table 3. Examples of Common Pathologies and How They Can Affect Vital Signs

Pathology	Prevalence among Older Adults	Vital Signs Affected
Cardiovascular disease	71%–75%	BP, pulse
Hypertension	60%–80%	BP
Atrial fibrillation	2%–4%	BP, pulse
Diabetes mellitus	18%–23%	BP, pulse, RR
Orthostatic hypotension	11%–50%	BP
Malnutrition	Community dwelling elders: 2%–10%; hospitalized/institutionalized elders: 30%–60%	Temperature

BP, blood pressure; RR, respiratory rate.

change in vital signs with age is limited. Thus, a major limitation is the ability to filter the literature for important articles that are on topic. A second limitation is that most of the literature describes disease-specific changes. Our objective for this review was to describe changes that occur in normal aging and to evaluate how these changes impact vital signs. In some cases, the line between pathology and aging becomes blurred for disease processes that occur in a large proportion of the population, eg, atherosclerosis. This is amplified when multiple comorbidities are superimposed on a frail, older patient. Table 3 summarizes common pathologies in older adults and the vital signs that can be affected. To remain within our objective to describe changes in normal aging, we needed to be selective about the mechanisms and disease processes presented.

CONCLUSIONS

In the older patient, vital signs are increasingly reflective of age and pathological changes in organ systems. The resultant constriction of homeostatic capacity leads to a loss of regulatory and adaptive mechanisms such that insults are often not always met with an appropriate and timely response (eg, aging-related sympathetic dysregulation leading to postural hypotension). Clinically, this reduced capacity has 2 outcomes: (1) a constriction in the range of the vital signs (reduced variability) and (2) a reduction in the ability to compensate when stressed. Thus, in the older patient, clinicians should use a personalized reference range and consider values outside of the individualized range as a marker for underlying disease. Constriction in homeostatic capacity with age and pathological decline in function, as is seen in nursing home patients, renders successive vital sign measurements, such as those collected in the long-term care setting, especially sensitive and useful for detecting a potentially deleterious change.

Vital signs have developed as a fundamental tool for diagnosis, disease severity, and communication. In older patients, more studies are needed to validate that vital signs actually represent vital function. Additionally, consideration needs to be given to newer vital signs that may better measure disease in the older population (ie, mental status, function, pain, and so forth). Even in these patients, change from an individual reference range may be the most sensitive marker of vital function.

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