NIH Public Access
Author Manuscript
J Am Med Dir Assoc. Author manuscript; available in PMC 2012 June 1.

Published in final edited form as:
J Am Med Dir Assoc. 2011 June ; 12(5): 337–343. doi:10.1016/j.jamda.2010.04.009.

Vital Signs in Older Patients: Age-Related Changes

Jennifer Gonik Chester, M.D. Candidate[Class of 2012] and
Albert Einstein College of Medicine, Bronx, NY

James L. Rudolph, M.D., S.M.
Harvard Medical School, Boston, MA

Abstract

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: a) molecular changes; b) organ system changes; c) systemic changes; d) 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.

Keywords

Aged; Vital Sign; Pulse; Blood Pressure

Introduction

The four 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 exam 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, healthcare 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.

Current Address: 8A Mount Auburn Street, #22, Cambridge, MA 02138, Phone: 248-790-9211, Jennifer.chester@med.einstein.yu.edu
Current Address: VA Boston Healthcare System, Jamaica Plain Division, 150 S. Huntington Avenue, Boston, MA 02130, Phone: 857-364-6812, Email: jrudolph@partners.org

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Vital signs are non-invasively measured using simple equipment (i.e. thermometer, sphygmomanometer, watch). Provided that the equipment is well-calibrated and that the assessor is well-trained, vital signs are a reliable measurement (e.g. two measurements should be highly correlated). Additionally, the vital sign produced is an objective measurement of physiologic function (i.e. 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 between healthcare 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 which needs additional investigation, but not 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 which can signify both the progression of a disease process and the effectiveness of treatment. For example, a change in systolic blood pressure from 120 mmHg to 90 mmHg might mark increasing sepsis or the effect of a treatment. This paper 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 due to advancing physiologic and functional decline. This can manifest itself as confinement of vital signs to individual ranges, thus reducing sensitivity (e.g. 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 (e.g. 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 since 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: a) molecular changes; b) organ system changes; c) systemic changes; d) 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.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Methods

A systematic approach was used to select sources for this review. A literature search was conducted using Pubmed and Medline online encyclopedia. The Medical Subject Headings terms 'blood pressure,' 'respiration,' 'pulse,' 'body temperature' combined with 'aged' were used. Additional keyword searches included the terms: 'vital signs,' 'temperature,' 'fever,' 'thermoregulation,' 'respiratory rate,' 'heart rate,' and 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 comprised 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

1. 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 due to a combination of accumulating oxidative stress (i.e. decreased nitric oxide and increased cyclooxygenase production)[5,6] and increased production of elastases and metaloproteinases in vascular smooth muscle which degrade the endothelial basement membrane. These insults initiate a wound-injury response which 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

While 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 which 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.

While 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 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

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

variety of factors including medications, diseases, and autonomic dysregulation,19 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 due to 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 due to deconditioning and autonomic dysregulation.21 Heart rate variability, i.e. 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 which 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.

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 50–60% 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. Since 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 While this may not affect day-today 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.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Respiratory rate is easy to measure, requiring only observation and a watch with a second hand. Nevertheless, recent studies report that among the four standard vital signs, respiratory rate is least often recorded by health care professionals.[33,34,35] 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 not being aware and changing his breathing, lack of understanding about the importance of respiratory rate, and lack of electronic equipment for measuring respiratory rate.[36] Since 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.

3. 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 temperature[40] and altered thermoregulatory responses.[41] While 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 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, e.g. blunting of adaptive immunity.[52] In particular, T-cell function is observed to deteriorate with age perhaps due to 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, due 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 since among nursing home residents, body temperature rarely exceeds 101 degrees Fahrenheit.[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

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

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 0–10 has gained popularity as an important screening tool in a variety of healthcare settings, and has been widely referred to as the "fifth vital sign".59,60,61 Between 25–50% of community-dwelling seniors and 45–80% of long-term care residents report that they commonly experience significant, undertreated, and often disabling daily pain.62,63 This data has 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

While 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

While there is a large body of literature describing the aging of systems, the number of studies that address the change in vital signs with age is limited. Thus a major limitation is the ability to filter the literature for important articles which are on topic. A second limitation is that the majority 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, e.g., 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. In order to remain within our objective to describe changes in normal aging, we needed to be selective about the mechanisms and disease processes presented.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

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 (e.g. aging-related sympathetic dysregulation leading to postural hypotension.) Clinically, this reduced capacity has two outcomes: a) a constriction in the range of the vital signs (reduced variability) and b) 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 which may better measure disease in the older population (i.e. mental status, function, pain, etc.) Even in these patients, change from an individual reference range may be the most sensitive marker of vital function.

Acknowledgments

Declaration of Sources of Funding

This project was supported by funding from the American Federation for Aging Research [5-T35- AG026781-05]; the Einstein Research Scholarship; and the VA Rehabilitation Research and Development Career Development Award. The financial sponsors played no role in the design, execution, analysis or interpretation of data, or in the writing of the study.

References

1. Stedman’s Medical Dictionary for the Health Professions and Nursing. 5. Baltimore, MD: Lippincott Williams & Wilkins; 2005.
2. Cwinn MA, Forster AJ, Cwinn AA, et al. Prevalence of information gaps for seniors transferred from nursing homes to the emergency department. CJEM. 2009; 11:462–71. [PubMed: 19788791]
3. Joint Commission. 2006 Critical access hospital and hospital national patient safety goals #2E. [Accessed April 15, 2010]. http://www.jointcommission.org/PatientSafety/NationalPatientSafetyGoals/06_npsg_cah.htm
4. Kuchel, GA. Hazzard’s Geriatric Medicine and Gerontology. 6. New York: The McGraw-Hill Companies, Inc; 2009. Chapter 51: Aging and Homeostatic Regulation.
5. Csiszar A, Ungvari Z, Edwards JG, et al. Aging-induced phenotypic changes and oxidative stress impair coronary arteriolar function. Circ Res. 2002; 90(11):1159–1166. [PubMed: 12065318]
6. Herrera MD, Mingorance C, Rodríguez-Rodríguez R, Alvarez de Sotomayor M. Endothelial dysfunction and aging: An update. Ageing Res Rev. 2010; 9(2):142–52. [PubMed: 19619671]
7. Pauly RR, Passaniti A, Bilato C, et al. Migration of cultured vascular smooth muscle cells through a basement membrane barrier requires type IV collagenase activity and is inhibited by cellular differentiation. Circ Res. 1994; 75(1):41–54. [PubMed: 8013081]
8. Hariri RJ, Alonso DR, Hajjar DP, et al. Aging and arteriosclerosis. I. Development of myointimal hyperplasia after endothelial injury. J Exp Med. 1986; 164(4):1171–1178. [PubMed: 3760777]
9. Thorin E, Thorin-Trescases N. Vascular endothelial ageing, heartbeat after heartbeat. Cardiovasc Res. 2009; 84(1):24–32. [PubMed: 19586943]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

10. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a “set up” for vascular disease. Circulation. 2003; 107(1):139–146. [PubMed: 12515756]
11. Lakatta EG. Age-associated cardiovascular changes in health: impact on cardiovascular disease in older persons. Heart Fail Rev. 2002; 7(1):29–49. [PubMed: 11790921]
12. Boengler K, Schulz R, Heusch G. Loss of cardioprotection with ageing. Cardiovasc Res. 2009; 83(2):247–261. [PubMed: 19176601]
13. Lakatta EG. Deficient neuroendocrine regulation of the cardiovascular system with advancing age in healthy humans. Circulation. 1993; 87(2):631–636. [PubMed: 8425306]
14. Lipsitz LA. Orthostatic hypotension in the elderly. N Engl J Med. 1989; 321(14):952–957. [PubMed: 2674714]
15. Ooi WL, Hossain M, Lipsitz LA. The association between orthostatic hypotension and recurrent falls in nursing home residents. Am J Med. 2000; 108:106–111. [PubMed: 11126303]
16. Gupta V, Lipsitz LA. Orthostatic hypotension in the elderly: diagnosis and treatment. Am J Med. 2007 Oct; 120(10):841–7. [PubMed: 17904451]
17. Le Couteur DG, Fisher AA, Davis MW, McLean AJ. Postprandial systolic blood pressure responses of older people in residential care: association with risk of falling. Gerontology. 2003; 49:260–4. [PubMed: 12792163]
18. Shibao C, Grijalva CG, Raj SR, et al. Orthostatic hypotension-related hospitalizations in the United States. Am J Med. 2007; 120 (11):975–80. [PubMed: 17976425]
19. Mosnaim AD, Abiola R, Wolf ME, Perlmuter LC. Etiology and risk factors for developing orthostatic hypotension. Am J Ther. 2010 Jan–Feb; 17(1):86–91. [PubMed: 19433976]
20. Lakatta EG. Cardiovascular aging in health. Clin Geriatr Med. 2000; 16(3):419–444. [PubMed: 10918640]
21. Coupe M, Fortrat JO, Larina I, et al. Cardiovascular deconditioning: From autonomic nervous system to microvascular dysfunctions. Respir Physiol Neurobiol. 2009; 169 (Suppl 1):S10–12. [PubMed: 19379845]
22. Agelink MW, Malessa R, Baumann B, et al. Standardized tests of heart rate variability: normal ranges obtained from 309 healthy humans, and effects of age, gender, and heart rate. Clin Auton Res. 2001 Apr; 11(2):99–108. [PubMed: 11570610]
23. Chaves PH, Varadhan R, Lipsitz LA, et al. Physiological complexity underlying heart rate dynamics and frailty status in community-dwelling older women. J Am Geriatr Soc. 2008; 56(9): 1698–1703. [PubMed: 19166446]
24. Buccelletti E, Gilardi E, Scaini E, et al. Heart rate variability and myocardial infarction: systematic literature review and metanalysis. Eur Rev Med Pharmacol Sci. 2009 Jul–Aug; 13(4):299–307. [PubMed: 19694345]
25. Ahmad S, Ramsay T, Huebsch L, et al. Continuous multi-parameter heart rate variability analysis heralds onset of sepsis in adults. PLoS One. 2009 Aug 14.4(8):e6642. [PubMed: 19680545]
26. Jiang W, Hathaway WR, McNulty S, et al. Ability of heart rate variability to predict prognosis in patients with advanced congestive heart failure. Am J Cardiol. 1997 Sep 15; 80(6):808–11. [PubMed: 9315600]
27. MacNee W. Accelerated lung aging: a novel pathogenic mechanism of chronic obstructive pulmonary disease (COPD). Biochem Soc Trans. 2009; 37(Pt 4):819–823. [PubMed: 19614601]
28. Janssens JP, Pache JC, Nicod LP. Physiological changes in respiratory function associated with ageing. Eur Respir J. 1999; 13(1):197–205. [PubMed: 10836348]
29. Peterson DD, Pack AI, Silage DA, Fishman AP. Effects of aging on ventilatory and occlusion pressure responses to hypoxia and hypercapnia. Am Rev Respir Dis. 1981; 124(4):387–391. [PubMed: 7294501]
30. Verbeken EK, Cauberghs M, Mertens I, et al. The senile lung. Comparison with normal and emphysematous lungs. 1. Structural aspects. Chest. 1992; 101(3):793–799. [PubMed: 1541148]
31. Bonomo L, Larici AR, Maggi F, et al. Aging and the respiratory system. Radiol Clin North Am. 2008; 46(4):685–702. v–vi. [PubMed: 18922288]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

32. Krumpe PE, Knudson RJ, Parsons G, Reiser K. The aging respiratory system. Clin Geriatr Med. 1985; 1(1):143–175. [PubMed: 3913497]
33. Cretikos MA, Bellomo R, Hillman K, et al. Respiratory rate: the neglected vital sign. Med J Aust. 2008; 188(11):657–659. [PubMed: 18513176]
34. Chen J, Hillman K, Bellomo R, et al. The impact of introducing medical emergency team system on the documentations of vital signs. Resuscitation. 2009; 80(1):35–43. [PubMed: 19010579]
35. Leuvan CH, Mitchell I. Missed opportunities? An observational study of vital sign measurements. Crit Care Resusc. 2008; 10(2):111–15. [PubMed: 18522524]
36. Hogan J. Why don't nurses monitor the respiratory rates of patients? Br J Nurs. 2006; 15(9):489–492. [PubMed: 16723921]
37. Fieselmann JF, Hendryx MS, Helms CM, Wakefield DS. Respiratory rate predicts cardiopulmonary arrest for internal medicine inpatients. J Gen Intern Med. 1993; 8(7):354–360. [PubMed: 8410395]
38. Subbe CP, Davies RG, Williams E, et al. Effect of introducing the Modified Early Warning score on clinical outcomes, cardio-pulmonary arrests and intensive care utilisation in acute medical admissions. Anaesthesia. 2003; 58(8):797–802. [PubMed: 12859475]
39. Ridley S. The recognition and early management of critical illness. Ann R Coll Surg Engl. 2005 Sep; 87(5):315–22. [PubMed: 16176687]
40. Kenney WL, Munce TA. Invited review: aging and human temperature regulation. J Appl Physiol. 2003; 95(6):2598–2603. [PubMed: 14600165]
41. Sund-Levander M, Grodzinsky E. Time for a change to assess and evaluate body temperature in clinical practice. Int J Nurs Pract. 2009; 15(4):241–249. [PubMed: 19703039]
42. Daniels F, Baker PT. Relationship between body fat and shivering in air at 15 C. J Appl Physiol. 1961; 16:421–425. [PubMed: 13719620]
43. Collins KJ, Dore C, Exton-Smith AN, et al. Accidental hypothermia and impaired temperature homoeostasis in the elderly. Br Med J. 1977; 1(6057):353–356. [PubMed: 837095]
44. Falk B, Bar-Or O, Smolander J, Frost G. Response to rest and exercise in the cold: effects of age and aerobic fitness. J Appl Physiol. 1994; 76(1):72–78. [PubMed: 8175550]
45. Ferrari E, Cravello L, Muzzoni B, et al. Age-related changes of the hypothalamic-pituitary-adrenal axis: pathophysiological correlates. Eur J Endocrinol. 2001; 144(4):319–329. [PubMed: 11275940]
46. Reiter RJ. The pineal gland and melatonin in relation to aging: a summary of the theories and of the data. Exp Gerontol. 1995; 30(3–4):199–212. [PubMed: 7556503]
47. Richardson GS, Carskadon MA, Orav EJ, Dement WC. Circadian variation of sleep tendency in elderly and young adult subjects. Sleep. 1982; 5 (Suppl 2):S82–94. [PubMed: 7156658]
48. Weitzman ED, Moline ML, Czeisler CA, Zimmerman JC. Chronobiology of aging: temperature, sleep-wake rhythms and entrainment. Neurobiol Aging. 1982; 3(4):299–309. [PubMed: 7170047]
49. Anderson KN, Hatfield C, Kipps C, et al. Disrupted sleep and circadian patterns in frontotemporal dementia. Eur J Neurol. 2009; 16(3):317–323. [PubMed: 19170747]
50. Harper DG, Stopa EG, McKee AC, et al. Differential circadian rhythm disturbances in men with Alzheimer disease and frontotemporal degeneration. Arch Gen Psychiatry. 2001; 58(4):353–360. [PubMed: 11296096]
51. Weinert D, Waterhouse J. The circadian rhythm of core temperature: effects of physical activity and aging. Physiol Behav. 2007; 90(2–3):246–256. [PubMed: 17069866]
52. Sansoni P, Vescovini R, Fagnoni F, et al. The immune system in extreme longevity. Exp Gerontol. 2008; 43(2):61–65. [PubMed: 17870272]
53. Hasday JD, Fairchild KD, Shanholtz C. The role of fever in the infected host. Microbes Infect. 2000; 2(15):1891–1904. [PubMed: 11165933]
54. Castle SC, Norman DC, Yeh M, et al. Fever response in elderly nursing home residents: are the older truly colder? J Am Geriatr Soc. 1991; 39(9):853–857. [PubMed: 1885858]
55. Gomolin IH, Aung MM, Wolf-Klein G, Auerbach C. Older is colder: temperature range and variation in older people. J Am Geriatr Soc. 2005; 53:2170–2. [PubMed: 16398904]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

56. Gunes UY, Zaybak A. Does the body temperature change in older people? J Clin Nurs. 2008; 17:2284–2287. [PubMed: 18705705]
57. Langan, M.; Zieve, D. Aging changes in vital signs. Medline Plus Encyclopedia, US National Library of Medicine and the National Institutes of Health; [Accessed April 15, 2010]. (10-27-08)http://www.nlm.nih.gov/medlineplus/ency/article/004019.htm
58. Keating HJ 3rd, Klimek JJ, Levine DS, Kiernan FJ. Effect of aging on the clinical significance of fever in ambulatory adult patients. J Am Geriatr Soc. 1984; 32(4):282–287. [PubMed: 6707408]
59. McCaffery M, Pasero CL. Pain ratings: the fifth vital sign. Am J Nurs. 1997; 97:15–16. [PubMed: 9025664]
60. Molony SL, Kobayashi M, Holleran EA, Mezey M. Assessing pain as a fifth vital sign in long-term care facilities: Recommendations from the field. J Gerontol Nurs. 2005 Mar; 31(3):16–24. [PubMed: 15799633]
61. Lynch M. Pain: the fifth vital sign. Comprehensive assessment leads to proper treatment. Adv Nurse Pract. 2001 Nov; 9(11):28–36. [PubMed: 12420497]
62. Mobily PR, Herr KA, Clark K, Wallace RB. An epidemiologic analysis of pain in the elderly. J Aging Health. 1994; 6:139–55.
63. Ferrell BA, Ferrell BR, Osterweil D. Pain in the nursing home. J Am Geriatr Soc. 1990; 38:409–14. [PubMed: 2109765]
64. Livingston EH. Misinterpretation of the Fifth Vital Sign—Invited Critique. Arch Surg. 2007; 142(5):419–420.
65. Inouye SK. Delirium in hospitalized older patients: Recognition and risk factors. J Geriatr Psychiatry Neurol. 1998; 11:118–125. [PubMed: 9894730]
66. Lyness JM. Delirium: Masquerades and misdiagnosis in elderly inpatients. J Am Geriatr Soc. 1990; 38:1235–1238. [PubMed: 2246460]
67. Flaherty JH, Rudolph J, Shay K, et al. Delirium is a serious and under-recognized problem: why assessment of mental status should be the sixth vital sign. J Am Med Dir Assoc. 2007; 8(5):273–275. [PubMed: 17570303]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Table 1

Increasing Blood Pressure with Age

Age (years)	% of Females with Hypertension	% of Males with Hypertension
55–64	56%	47%
65–74	74%	61%
≥75	83%	69%

(NHANES [1999–2002], CDC/NCHS)

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Table 2

Summary of Vital Sign Changes with Aging

Age-related mechanisms of VS change	Blood Pressure	Pulse	Respiratory Rate	Temperature
Molecular	• oxidative and mechanical damage to vascular endothelium • heightened inflammatory response: – cytokines – growth factors – collagen – elastases and metaloproteinases • decreased arterial wall pliability	• desensitization of sympathetic receptors disrupts intracellular signaling	• ncreased elastases – degrade elastic tissue – reduce compliance – cause dilation of airspaces	• decreased T-cell function due to prolonged antigenic exposure • less apoptosis in aged resistant T-cells • Structural changes in hypothalamic mineralocorticoid receptors cause Hypothalamic-Pituitary Axis hyperactivity
Structural/Organ	• increased left ventricular wall thickness • diastolic dysfunction due to: – increased wall thickness – less cardiac filling	• temporarily decreased perfusion due to: – decreased baroreflex sensitivity – delayed reaction	• altered chest wall shape due to: – kyphosis – osteoporosis – costal cartilage calcification • increased work of breathing: – altered diaphragm shape – decreased compliance	• Dysfunctional/deficient hypothalamic mineralocorticoid receptors ↑ nighttime cortisol levels • Hypothalamic suprachiasmatic nucleus hyperactivity
Systemic	• arterial stiffness requires higher systolic pressures to pump blood • increased pulse pressure	• decreased cardiac output and increased resting heart rate • maximum heart rate is more limited with age	• 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	• reduced ability to maintain body heat due to: – less subcutaneous fat – reduced peripheral vasoconstriction – decreased cardiac output

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

Age-related mechanisms of VS change	Blood Pressure	Pulse	Respiratory Rate	Temperature
				– dysregulated circadian rhythm – loss of muscle mass
Compensation to Stress	• reduction in endogenous cellular repair capability due to damaged cardiomyocytes & vascular endothelium • altered intracellular protein expression • mitochondrial aging & changes in signal transduction cascades • loss of responsiveness to sympathetic stimuli	• less sympathetic responsiveness hinders ability of CV system to adjust when stimulated • less adaptability in heart rate is associated with falls, frailty	• 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	• loss of heat maintenance & thermogenesis mechanisms • heightened vulnerability to hot and cold stressors • lower core body temperature hinders ability to regulate body temperature

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

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%; hospital/institutionalized elders: 30–60%	Temp