

Review Article

Psychiatric and Functional Physical Symptoms: The More Telling “Fifth” Vital Sign

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Abstract: Historically, psychiatric symptomatology has been looked upon with a mixture of bewilderment and derision—the evidence of some kind of psychological or spiritual problem—but not of a medical or biological one. However, an explosion of new research suggests that psychiatric symptomatology may actually reveal as much about the physiological functioning of the body as heart rate, respiratory rate, temperature, and blood pressure. For example, there is growing evidence that psychiatric symptoms are associated with autonomic dysregulation and the early development of chronic debilitating diseases, such as diabetes, cardiovascular, hypertension, and cancer. These diseases then take the lives of the mentally ill at a much earlier age than the general population. In addition, an association has been found between upper-end-of-normal resting vital signs and the later development of major psychiatric illnesses, such as generalized anxiety disorder, obsessive-compulsive disorder, and schizophrenia. Notably, a similar association has been found between upper-end-of-normal resting vital signs and the early development of the same illnesses that shorten the lives of the mentally ill. These associations raise the possibility that subtle elevations in resting vital signs and psychiatric symptomatology are different manifestations of a shared physiological abnormality. The identification of a core abnormality that influences such diverse emotional, psychological, and biological conditions could completely reshape the way we think about mental illness. It could unify mental disorders and medical disorders and raise psychiatric symptoms to the level of a “fifth” vital sign. This article will explore the links between mental illness, physical illness, and resting vital signs in an effort to utilize, if appropriate, psychiatric symptomatology as a barometer of physiological function. The unique value of psychiatric symptomatology in this regard is that it could reduce the need to distinguish psychiatric symptoms from medical symptoms while at the same time more clearly revealing, without any physical instrumentation, what is happening inside the body.

Keywords: Psychiatric Symptoms, Chronic Pain, Neuronal Hyperexcitability, Vital Signs, Biomarkers of Disease, Preventive Medicine, Anticonvulsants, Neuroregulators

1. Introduction

The measurement of the four basic vital signs—heart rate, respiratory rate, blood pressure, and temperature—have traditionally been an essential part of medical assessment because vital signs measure the body’s basic functions; they display a snapshot of what is going on inside the body. However, there is growing evidence of a bidirectional link between resting vital signs and psychiatric symptomatology. Specifically, persons with upper-end-of-normal resting vital

signs are more likely to develop mental illness and, conversely, persons with mental illness are more likely to have upper-end-of-normal resting vital signs [1, 2]. That raises the possibility that subtle elevations in resting vital signs and psychiatric symptomatology are different manifestations of a shared physiological trait. This article will explore the relationship between resting vital signs and psychiatric symptomatology in an effort to re-examine the way mental illness is thought about and managed in clinical practice.

2. Perceptions About Mental Illness

Since antiquity, mental illness has been viewed as more of a behavioral phenomenon than a medical phenomenon. Because persons with mental illness typically look normal and generally fail to develop any acute medical abnormalities, their psychological, emotional, and behavioral abnormalities have variably been attributed to sicknesses of the mind, the emotions, the will, or the spirit. Still today, mental illness is separated from mainstream medicine via differences in location of treatment (e.g., free-standing mental health centers and behavioral medicine clinics), specialized hospital consultation services (e.g., psychiatric consult-liaison), and carve-out payor contracts for mental health services.

However, a rapidly growing body of evidence suggests that mental illness may not be physiologically distinct from other illness processes. The binding tie is their shared relationship with resting vital-sign measurements. For example, in a longitudinal study involving more than one million men in Sweden, Latvala et al. [1] found that subtle elevations in resting heart rate (RHR) were predictive of the later development of generalized anxiety disorder, obsessive-compulsive disorder, and schizophrenia. Similarly, Blom et al. [2] found that adolescent girls with emotional disorders had increased resting respiratory rates (RRR) in comparison to healthy controls. Strikingly, the same kinds of associations have been found between resting heart and respiratory rates and general medical illnesses. For example, Kannel et al. [3] found that resting heart rates predicted cardiovascular disease mortality, and Baumert et al. [4] found that nocturnal respiratory rate predicted both cardiovascular and all-cause mortality in community-dwelling older men and women. Other measures that have been linked to an increased vulnerability to illness include resting blood pressure (RBP) [1, 5, 6], blood pressure variability [5, 7], heart rate variability [7-10], and heart rate recovery [11, 12]. Notably, the increased risk of morbidity and mortality associated with these indices can be more than two-fold [4, 9, 11, 13].

Taken together, these associations suggest that mental illness and physical illness may be different manifestations of a shared biological abnormality that also causes subtle elevations in RHR, RRR, and other indices of autonomic function. Although identifying such an abnormality has been the focus of intensive research, little has been offered in the way of what the abnormality might be. However, an emerging hypothesis contends that psychiatric symptoms are driven by pathological hyperactivity in symptom-related circuits in the brain. According to the Multi-Circuit Neuronal Hyperexcitability (MCNH) Hypothesis of Psychiatric Disorders [14], persistent firing in anxiety circuits causes persistent feelings of anxiety; persistent firing in depressive circuits causes persistent feelings of depression; persistent firing in cognitive circuits causes ruminative and obsessive thoughts; etc... This pathological circuit-specific hyperactivity is believed to be the consequence of an inherent failure of the neurological system to self-regulate when perturbed by a psychological, emotional, or biological stressor [15, 16].

Clinically, persons with psychiatric disorders are quick to react [17] and slow to recover [18] from stressful events. Even in the absence of specific stressors, they tend to be stressed by abstract worries and fears [18, 19]. Hypothetically, the same physiological abnormality that would cause this constitutional state of cognitive and emotional hyper-arousal, namely, neuronal hyperexcitability, would also cause a constitutional hyper-activation of the autonomic nervous system, the hypothalamic-pituitary-adrenal system, the immunologic system, the metabolic system, the muscular system, and various other systems of the body. This could explain the link between resting vital-sign elevations, chronic or recurrent psychiatric symptoms, and the plethora of chronic diseases of which they are predictive, such as diabetes, high blood pressure, heart disease, autoimmune disease, cancer, dementia, and many other health conditions [20]. The hypothetical reason that psychiatric symptoms tend to precede the development of the aforementioned disease processes is that the cognitive-emotional system is more acutely and overtly expressive of neuronal excitation than the physical organs and tissues of the body [20]. The only exceptions are “functional” physical symptoms and standard vital-sign measurements, as they, like psychiatric symptoms, express moment-to-moment changes in the physiological state of the body. Thus, psychiatric and functional physical symptoms, like heart rate, respiratory rate, blood pressure, and temperature, could appropriately be viewed as vital signs.

The idea that a subjective symptom could be a window into the body has already been accepted with the recent adoption of pain as a so-called “fifth” vital sign [21]. However, like pain, psychiatric and functional physical symptoms may reflect the sensitivity or “excitability” of the neurological system. That raises the question of whether psychiatric and functional physical symptoms should be combined with physical pain as a fifth vital sign. The answer would depend upon how vital signs should best be used.

3. The Use of Vital Signs

Currently, vital signs are used to get a sense of how the body is functioning. As one of those clinical signs, pain can be an indicator of tissue irritation or injury. However, as a clinical symptom, pain describes how the patient is feeling; hence, it does not necessarily relate anything about the state of tissues or organs [22]. That creates a clinical dilemma when the patient’s pain level exceeds that which would be expected under the circumstances.

By far the most common cause of excessive or persistent pain is an abnormal amplification of sensory signals by the neurological system. This, together with the plethora of other negative thoughts and emotions that can be driven by pathologically-elevated neurological activity, is the physiological basis of the *suffering* that is rolled into the perception of pain [22, 23]. Other possible causes of medically-unexplained pain include “conversion disorder” (the unconscious transfer of unacceptable psychological or physical symptoms to a more acceptable symptom, such as

pain); “factitious disorder” (the conscious feigning of a medical or psychiatric symptom for the emotional gratification of being in the sick role); and malingering (the conscious pretense of medical or psychiatric symptoms for the purpose of secondary gain) [24]. According to the MCHN hypothesis, any of these medically confounding factors could be, and most likely would be, reflective of an inherent hyperexcitability of the neurological system. That’s because excessive emotion, which itself is driven by neuronal hyperexcitability, is what most commonly causes people to say and do things that are socially and medically inappropriate.

As previously stated, neuronal hyperexcitability is also believed to be at the root of psychiatric symptoms and various somatic complaints for which no evidence of pathology can be found. Therefore, these so-called “functional” symptoms, which can include pain, could convey important information about the physiological functioning of the neurological system. Objective indicators of neuronal hyperexcitability are an RHR above 75 beats/min or an RRR above 15 breaths/min [20, 25].

Practically speaking then, psychiatric and functional physical symptoms could be grouped together with unusually severe or persistent pain and resting vital-sign measurements as markers of neuronal hyperexcitability. Appropriate treatment would involve any intervention that would have a calming effect on the nervous system. In the acute setting, the most expeditious intervention would be the administration of Neuroregulators (anticonvulsants and other brain-calming drugs). This idea is supported by the growing use of gabapentin and other non-benzodiazepine anticonvulsants in the treatment of chronic pain [26]. What deserves special attention, however, is that by quieting the nervous system, the treatment could be addressing the root cause of all of the patient’s symptoms, whether medical or psychiatric. The recognition of this would help circumvent the clinical dilemma of having to distinguish pain as a sign of tissue trauma from pain as a symptom of suffering.

Although resting heart and respiratory rates are, hypothetically, independent barometers of neuronal excitability, there is great value in adding psychiatric and functional physical symptoms to pain as a fifth vital sign. The advantage of these *subjective* markers of physiological function is that traditional vital-sign measurements, though *objective*, can be confounded by such factors as cardiorespiratory disease, cardiorespiratory medications, stimulant or depressant-type drugs, acute emotional states, levels of fitness, and recent food intake. This, together with the profound importance of being able to detect and treat neuronal hyperexcitability, would justify including psychiatric and functional physical symptoms in the “fifth” vital sign category.

Another problem that this new conceptualization of psychiatric and functional physical symptoms would help overcome is the challenge of disentangling psychiatric symptoms from medical symptoms. This common dilemma is the consequence of dichotomizing psychiatric and medical symptoms. However, when psychiatric symptoms are viewed

as subjective markers of a vulnerability trait that is shared by nearly all disease processes, the dichotomization of mental illness and physical illness can be seen for what it really is: a failure to understand the pathophysiology of mental illness. Recognizing this would give clinicians the satisfaction of knowing that in treating neuronal hyperexcitability they would be addressing the underlying physiological abnormality without having to distinguish psychiatric and functional physical symptoms from demonstrable organic pathology. Not only would this save valuable time, but it would also circumvent the stigma of mental illness as a potential barrier to appropriate treatment.

Practically speaking, the expanded fifth vital sign could be recorded alongside other vital signs as either “FLASH positive” or “FLASH negative.” FLASH is the user-friendly, non-stigmatizing, pathophysiologically-descriptive acronym that stands for Familial Limbic Autonomic System Hyperexcitability [20]. It describes what is hypothesized to be the fundamental abnormality in psychiatric disorders; namely, an inherent hyperexcitability of the neurological system that over-activates the limbic and autonomic nervous systems, thus elevating the affected person’s temperament, vital signs, and emotional responses to any form of stress—be it psychological, emotional, or biological. In recognition of its pervasive effects on bodily functions, FLASH would not be specific to mental illness but would be applicable to any pathological process that could be triggered or exacerbated by a hyperexcitability of the neurological system. Such broad applicability would work to prevent the term from becoming stigmatized, and it would dispel the many myths and erroneous assumptions that, throughout the ages, have been held about mental illness. The determination of FLASH status would ideally be made by nursing staff at the time of intake along with the patient’s standard vital signs. As previously stated, an RHR above 75 beats/min or an RRR above 15 breaths/min would be highly suggestive of the neuronal hyperexcitability trait [20, 25]. Any psychiatric or functional physical symptoms, which could include chronic pain, would provide additional support for a FLASH positive determination, although a positive determination could hypothetically be made based solely on either objective signs (i.e., RHR and RRR) or subjective symptoms (i.e., psychiatric and functional symptoms).

4. Discussion

Historically, mental illness has been viewed as an abnormality (or group of abnormalities) that is somehow distinct from other illnesses. This, together with the lack of a biological basis for psychiatric symptoms, has resulted in a dichotomization of mental illness and physical illness that frequently complicates clinical practice. However, an emerging hypothesis contends that psychiatric symptoms are the consequence of a pathological hyperactivity of symptom-related circuits in the brain. The underlying driver of this activity is thought to be a failure of neurons to self-regulate, thus resulting in the persistence of normal

thoughts and emotions that characterizes “psychiatric symptoms.” It can also result in a heightened sensitivity to pain, thus supporting the recent addition of pain as a fifth vital sign [21]. However, limiting this fifth vital sign to pain, though highly valuable in helping to reduce human suffering and comorbidity, fails to recognize that pain could be one of many symptoms that is revealing what a vital sign should reveal: important information about what is happening in the body. According to the MCNH hypothesis, pain, as well as psychiatric and functional physical symptoms, could be indicating that the neurological system is inherently hyperexcitable. In so-doing, these symptoms would not only sit alongside blood pressure, pulse, respiratory rate, and temperature as barometers of physiological function, but they could actually be quite telling about what is driving the abnormalities in these other vital signs. Because neuronal hyperexcitability heightens the reactivity of virtually every system of the body, it tends to increase heart rate, respiratory rate, blood pressure, and temperature. It also tends to dysregulate physiological systems, which is actually what abnormalities in standard vital signs are thought to reflect. The failure to recognize this could be one of the reasons that current initiatives to promote better pain management have failed to solve the problem of under-treated pain.

Although the MCNH hypothesis has yet to be validated through rigorous scientific studies, there is biological, observational, pharmacological, neuropsychiatric, behavioral, medical, psychophysiological, experimental, radiological, explanatory, and genetic evidence that nearly all psychiatric disorders and their functional comorbidities are rooted in this single, shared, neurophysiological abnormality [14]. Lending further support to the MCNH hypothesis is a rapidly growing body of evidence that links an increased vulnerability to developing psychiatric symptoms to subtle elevations in resting vital signs. From the perspective of the MCNH hypothesis, the physiological link between resting vital signs and the later development of mental illness is a failure of neurons throughout the central nervous system to maintain a normal resting state even in the absence of an acute stressor. This chronic, low-grade revving is thought to explain both the persistence of subclinical symptoms that characterizes most psychiatric disorders and the subtle vital-sign elevations that have been found to predict the development of more clinically apparent psychiatric symptoms when the neurological system is perturbed by stress.

The recognition of this has enormous implications for healthcare because 1) it identifies the core physiological abnormality in psychiatric disorders, thereby streamlining treatment; 2) it allows all clinicians to target the root cause of psychiatric symptoms without having to rely on psychiatric assessments that can potentially delay treatment, offend patients, and sometimes even be refused by patients; 4) it reconceptualizes psychiatric and functional physical symptoms as a “fifth” vital sign, thereby reducing the stigma of mental illness and increasing the accuracy of vital-sign measurements in identifying the core abnormality in chronic disease; 5) it unifies seemingly diverse streams of pathology

under one highly treatable neurophysiological abnormality; 6) it incentivizes patients to implement natural interventional strategies rather than relying solely of medical therapy; 7) it gives doctors a pathophysiologically-based way to target pain, thus reducing the risk of either under-medicating or over-medicating this common complaint; 8) it opens the door to illness prevention because the vulnerability trait, once identified, can be readily modified through the use of Neuroregulators and healthy lifestyle changes; and 9) it gives payers an evidence-based reason to end discriminatory practices that limit insurance coverage for mental health services.

5. Suggestions for Future Research

The value of applying the MCNH hypothesis in the form of adding psychiatric and functional physical symptoms to pain as a fifth vital sign could be tested by comparing anticonvulsant therapy to standard care in patients who, based on resting vital-sign measurements and the recommended fifth vital sign, were deemed to have a hyperexcitable neurological system. Also recommended are prospective studies looking at the accuracy of using standard vital-sign measurements both with and without the new “fifth” vital sign to predict which patients will have more complicated treatment courses and longer hospital stays for various illnesses.

6. Conclusion

Under the common assumption that mental illness is somehow different from organic disease processes, clinicians often minimize the complaints of patients who either present with psychiatric symptoms or have a history of psychiatric symptoms. Redefining these common symptoms as VITAL SIGNS has the potential to completely reshape the way health care professionals think about and treat mental illness. Completely antithetical to thinking about psychiatric symptoms as vague complaints without any organic basis, it has the potential utilize them as a means of identifying the core abnormality in most illnesses, whether mental or physical. It also has the potential to focus treatment directly at the target without having to disentangle psychiatric symptoms from other symptoms of disease. At the same time, the destigmatizing effect that this would have on mental illness would encourage patients to be more forthcoming about their psychiatric symptoms and more willing to accept treatment for them. This, in turn, would increase the usefulness of psychiatric symptoms as a “fifth” vital sign and facilitate the successful administration of Neuroregulators and other brain-calming interventions for the treatment of psychiatric and related physical symptoms and the prevention of all forms of chronic illness.

Conflicts of Interest

The author declares that he has no competing interests.

References

- [1] Latvala A, Kuja-Halkola R, Rick C, et al. Association of resting heart rate and blood pressure in late adolescence with subsequent mental disorders: A longitudinal population study of more than 1 million men in Sweden. *JAMA Psychiatry* 2016; 73 (12): 1268-1275.
- [2] Blom EH, Serlachius E, Chesney MA, Olsson EMG. Adolescent girls with emotional disorders have a lower end-tidal CO₂ and increased respiratory rate compared with healthy controls. *Psychophysiology* 2014; 51 (5): 412-418.
- [3] Kannel W, Kannel C, Paffenbarger R, Cupples A. Heart rate and cardiovascular mortality: The Framingham study. *Am Heart J* 1987; 113: 1489-1494.
- [4] Baumert M, Linz D, Stone K, et al. Mean nocturnal respiratory rate predicts cardiovascular and all-cause mortality in community-dwelling older men and women. *European Respiratory Journal* 2019; DOI: 10.1183/13993003.02175-2018.
- [5] Yano Y, Kario K. Nocturnal blood pressure and cardiovascular disease: a review of recent advances. *Hypertension Research* 2012; 35: 695-701.
- [6] Gottesman RF, Schneider ALC, Albert M. Midlife hypertension and 20-year cognitive change: the atherosclerosis risk in communities neurocognitive study. *JAMA Neurol* 2014; 71 (10): 1218-1227.
- [7] Barletta G-M, Flynn J, [...], Furth S, et al. Heart rate and blood pressure variability in children with chronic kidney disease: a report from the CKiD study. *Pediatr Nephrol* 2014; 29 (6): 1059-1065.
- [8] Dalal J, Dasbiswas A, Sathyamurthy I, et al. Heart Rate in Hypertension: Review and Expert Opinion. *International Journal of Hypertension* 2019; 2019.
- [9] Tsuji H, Larson MG, Venditti FJ, et al. Impact of reduced heart rate variability on risk for cardiac events. The Framingham Heart Study. *Circulation* 1996; 94: 2850-2855.
- [10] Thanou A, Stavrakis S, Dyer JW, et al. Impact of heart rate variability, a marker for cardiac health, on lupus disease activity. *Arthritis Res Ther* 2016; 18 (1): 197.
- [11] Jouven X, Empana J-P, Schwartz PJ, et al. Heart-rate profile during exercise as a predictor of sudden death. *N Engl J Med* 2005; 352: 1951-1958.
- [12] Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med* 1999; 341: 1351-1357.
- [13] Ó Hartaigh B, Gill TM, [...], Hardy R, et al. Association between resting heart rate across the life course and all-cause mortality: longitudinal findings from the Medical Research Council (MRC) National Survey of Health and Development (NSHD). *J Epidemiol Community Health* 2014; 68 (9): 883-889.
- [14] Binder MR. The multi-circuit neuronal hyperexcitability hypothesis of psychiatric disorders. *AJCEM* 2019; 7 (1): 12-30.
- [15] Johnstone T, van Reekum CM, Urry HL, Kalin NH, Davidson, RJ. Failure to regulate: counterproductive recruitment of top-down prefrontal-subcortical circuitry in major depression. *J. Neuroscience* 2007; 27 (33): 8877-8884.
- [16] Leuchter AF, Cook IA, Hunter AM, Cai C, and Horvath S. Resting-state quantitative electroencephalography reveals increased neurophysiologic connectivity in depression. *PLoS One* 2012; 7 (2): 1-13. e32508.
- [17] Herr RM, Barrech A, Reidel N, et al. Long-term effectiveness of stress management at work: Effects of the changes in perceived stress reactivity on mental health and sleep problems seven years later. *Int J Environ Res Public Health* 2018; 15 (2): 255.
- [18] Larsen BA, Christenfeld NJS. Cardiovascular disease and psychiatric comorbidity: the potential role of preservative cognition. *Cardiovascular Psychiatry and Neurology* 2009; Article ID: 791017.
- [19] Brainstorm Consortium; Anttila V, Bulik-Sullivan B, Finucane HK, et al. Analysis of shared heritability in common disorders of the brain. *Science* 2018; 360 (6395): eaap8757.
- [20] Binder MR. FLASH Syndrome: tapping into the root of chronic illness. *AJCEM* 2020; 8 (6): 101-109.
- [21] Scher C, Meador L, Van Cleave JH, Reid MC. Moving beyond pain as the fifth vital sign and patient satisfaction scores to improve pain care in the 21st century. *Pain Manag Nurs* 2018; 19 (2): 125-129.
- [22] Crofford LJ. Chronic Pain: Where the Body Meets the Brain. *Trans Am Clin Climatol Assoc* 2015; 126: 167-183.
- [23] Cousins N. Anatomy of an illness - as perceived by the patient. W. W. Norton Company, 2005.
- [24] Gallo S, Tatu L, Bogousslavsky J, Aybek S. Conversion, Factitious Disorder and Malingering: A Distinct Pattern or a Continuum? *Front Neurol Neurosci*. Basel, Karger 2018; 42: 72-80.
- [25] The Neuronal Excitability Spectrum: A New Paradigm in the Assessment, Treatment, and Prevention of Mental Illness and its Relation to Chronic Disease. *AJCEM* 2021; 9 (6): 193-209.
- [26] Binder MR. Gabapentin—the popular but controversial anticonvulsant drug may be zeroing in on the pathophysiology of disease. *AJCEM* 2021; 9 (4): 122-134.