Evidence-Based Surgical Hypothesis

Socioeconomic status and the assessment of operative risk

Vincent Chong, MD, MS, Laurel Imhoff, MD, *and* **Alden Harken, MD,** *Oakland, CA*

From the Department of Surgery, University of California, San Francisco-East Bay, Oakland, CA

Inequalities in health relative to socioeconomic status (SES), as measured by education, occupation, and income, have been documented and analyzed extensively. A relationship between SES and mortality has been confirmed in epidemiologic studies in the United Kingdom, Finland, and Russia. In a recent examination of SES relative to inequalities in health among 22 European countries, authors noted an increasing variability of magnitude in disparities of health between countries.[1] There is a 48-year difference in life expectancy among countries worldwide and a 20-year difference within some countries. The World Health Organization believes that social factors are at the root of these disparities, and they have launched a commission on the social determinants of health.[2] The purpose of this work is to promote the incorporation of SES in the preoperative assessment of operative risk.

Estimating operative risk is an essential aspect of perioperative decision-making. Surgeons have a multitude of tools designed to assist in this task. Despite an abundance of evidence for the independent effect of SES on operative morbidity and mortality, this variable is conspicuously absent from all standard calculations of operative risk.

The reasons for this omission are likely multifactorial. Data on SES are neither easily measurable nor readily available, and SES often is perceived to be immutable in the clinical encounter. Clinicians may therefore gravitate toward seemingly more quantifiable and modifiable risk factors, such as individual behavior, physiologic status, and accessible laboratory data.

We argue that clinician discomfort with SES should not preclude it from meaningful discussions of patient risk in the perioperative setting. Furthermore, we argue that evidence of plausible, reversible mechanisms explaining how SES influences health have come to light. The purposes of this work are: (1) to examine evidence linking SES to health outcomes, with a focus on perioperative morbidity and mortality; (2) to explore the influence of socioeconomically induced allostatic load on epigenetics; (3) to provide a plausible biologic mechanism through which SES may influence patient vulnerability to operative stress; and (4) to assert that SES should be incorporated into estimations of operative risk.

SES AND HEALTH OUTCOMES

Most contemporary studies of SES and health trace back to the Whitehall Studies, which demonstrated socioeconomic gradients in health outcomes for civil servants in the United Kingdom. Because health disparities related to SES are pervasive and persistent, their elimination has become the focus of national policy and research.[2] Increases in in-hospital mortality and cardiovascular complications caused by SES have been reported after coronary artery bypass grafting, total hip replacement, trauma, ventral hernia repair, and limb loss after revascularization. When the Carstairs Index is used, a summary measure of social deprivation based on neighborhood of residence, deprived patients undergoing curative resection for colorectal cancer were found 36% more likely to die from all causes, and 26% more likely to die from their cancer, than affluent patients. Defining SES via insurance status or median income based on zip code confirmed these disparities in operative outcome after each of 13 high-risk surgical procedures.[3]

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Reprint requests: Vincent Chong, MD, MS, Department of Surgery, University of California, San Francisco–East Bay, 1411 East 31st Street, QIC 22134, Oakland, CA 94602. E-mail: vchong@alamedahealthsystem.org.

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This significant association persisted after we adjusted for confounding variables such as race, age, patient comorbidities, hospital procedure volume, and hospital socioeconomic population (as measured by the incomes of patients served at that hospital).

After reviewing this literature, we contend that SES matters. We propose that its effect on postoperative outcomes withstands adjustment for clinical comorbidities that are highlighted already in surgical risk algorithms. We propose further that causal pathways between SES and postoperative outcomes are related plausibly to the embedding of social environments into patients' physiology and epigenome.

BIOLOGIC EMBEDDING

When social experiences and exposures alter human biologic processes, placing people at differential risk of disease, this phenomenon is termed biologic embedding, for which four conditions must be demonstrated[4]: (1) a biologic system (or related systems) sensitive to changing social experiences and exposures must be identified; (2) systematic differences in social exposure and environment (like SES differences) must lead to systematically different biodevelopmental states and/or functioning of the biologic system; (3) these biologic differences must be stable and clinically relevant; and (4) these biologic differences must confer the capacity to influence health, well-being, learning, and behavior through the life course. Neuroendocrine stress responses have attracted the majority of this investigative attention. These include the hypothalamic-pituitary-adrenal (HPA) axis, autonomic nervous system, prefrontal cortex, and social function of the locus coeruleus and amygdala, mediated by serotonin.

STRESS AND ALLOSTATIC LOAD

Stress is a common, unavoidable human experience with a strong link to disease. The hippocampus, amygdala, autonomic nervous system, and HPA axis play important roles in mediating biologic responses to stress. Allostasis is the organism's ability to cope with a changing environment and consists not only of the stimulation of physiologic systems in anticipation of and response to a threat, but also to de-escalation of systems when the threat resolves. Challenges to allostasis include anything that may stress an organism, including infection, malnutrition, low-resource neighborhoods, emotional neglect, violence, and operative procedures.

McEwen[5] postulated that environmental stress might have both protective and damaging effects on health. Allostatic changes may be acutely adaptive, but long-term dysregulation of cortisol can be damaging.[5] Chronic, persistent, repeated challenges lead to wear on the system, resulting in a pervasive, grinding allostatic load. Persistent allostatic load may provoke maladaptive responses to chronic, toxic stressors in the social environment. Both healthy and dysregulated responses to acute and chronic stress include: (1) repeated acute exposure to a stressor provokes adaptive de-escalation of the physiologic response; (2) chronic stress may deplete the capacity to adapt, leading to toxic neuroendocrine stimulation and damage; (3) a healthy organism (or patient) must be capable of decelerating the stress response appropriately; and, finally (4) chronic, smoldering stress can exhaust healthy responses to stress, leading to a "non-arousal" state.

Since its introduction, the concept of allostatic load has linked persuasively the social environment to health outcomes. As a biologic system, the HPA axis meets all the strict conditions for biologic embedding. Rat pups that receive a high level of maternal nurture in the early days of life exhibit low basal corticosterone levels and a proper response to acute stress; in contrast, abandoned rat pups exhibit greater baseline corticosterone levels and blunted responses to stress. These differences in corticosterone secretion persist beyond early development, as lifetime stress steroid levels are greater for undernurtured rat pups. These high levels of corticosterone also are associated with anatomic neuronal depletion in the undernurtured rat hippocampus. As a result, these rats exhibit deficits in memory, cognitive processing, and learning performance compared with nurtured rats. Low SES has long been understood to be associated with social isolation. Similarly, in humans, aspects of social deprivation, such as physical crowding, noise, family separation/turmoil, violence, single-parent status, lack of maternal high school diploma, and poor family finances, have been incorporated into an index of cumulative risk.[6] There is a compelling link between low SES and physiologic measures of increased allostatic load.

A robust and continuous inverse relationship between SES, educational level, and allostatic load was uncovered in all clinical age groups by Seeman et al.[7] This composite index of allostatic load includes blood pressure, blood hemoglobin A1c, waist-hip ratio, urinary cortisol, urinary norepinephrine and epinephrine, serum high-density lipoprotein cholesterol, total cholesterol/high-density lipoprotein ratio, albumin, interleukin-6, and C-reactive protein, forced expiratory volume in 1 second, dehydroepiandrosterone sulfate,