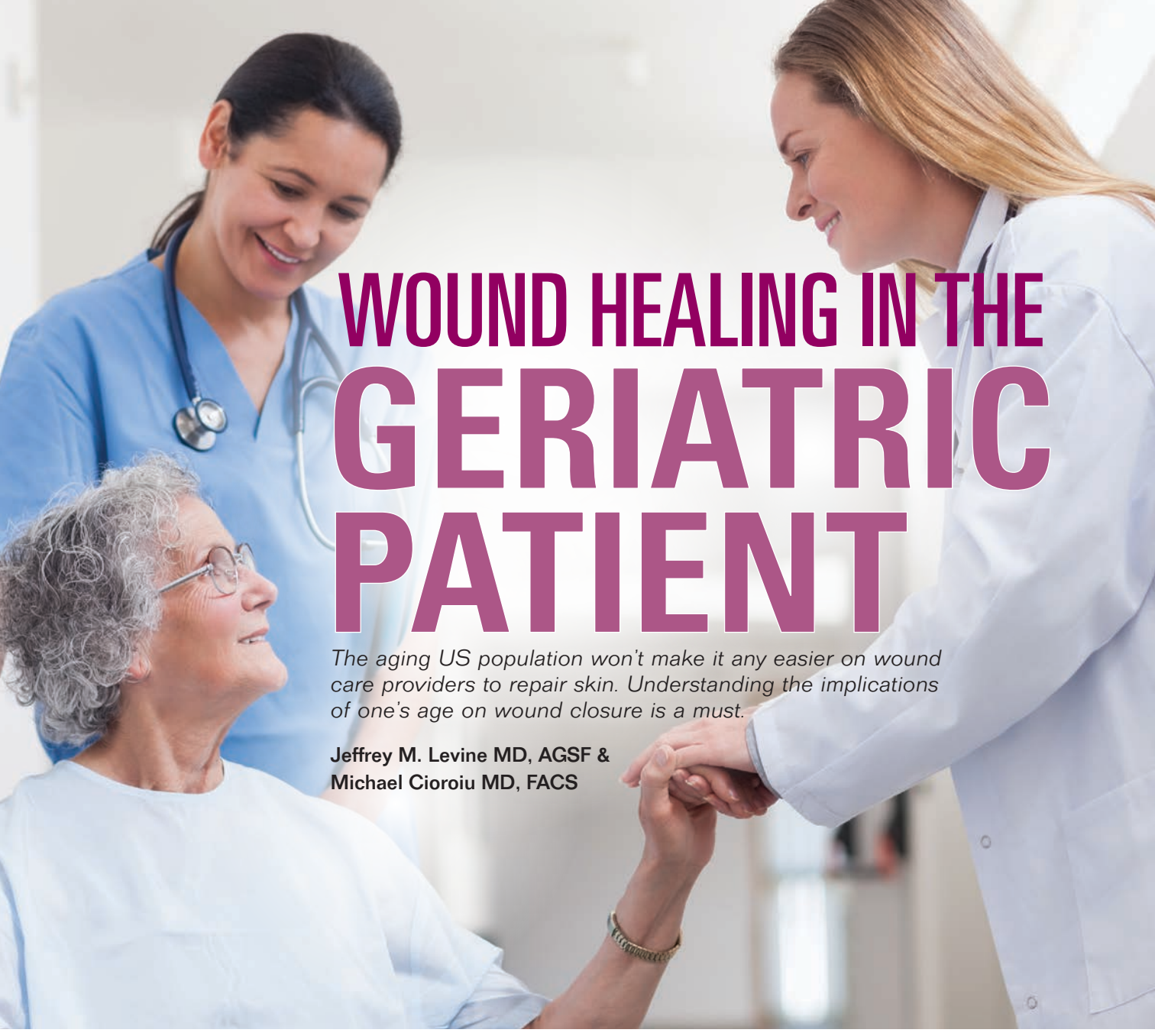

WOUND HEALING IN THE GERIATRIC PATIENT

The aging US population won't make it any easier on wound care providers to repair skin. Understanding the implications of one's age on wound closure is a must.

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The US is in the midst of a profound demographic change with the rapid aging of its population — a steep increase reflected by the estimated 75 million baby boomers who will move into the ranks of the geriatric healthcare population during the next 20 years. This demographic change is having a major impact on the organization and delivery of healthcare itself, particularly in the shift from acute to chronic illnesses that require healthcare providers to have an increased understanding of basic principles related to aging. Among the most impacted arenas of healthcare as the population gets older and medicine allows people to live longer with comorbid conditions will be outpatient wound centers. Chronic wounds have great impact on quality of life, and experts have estimated the cost of their care to be in excess of $25 billion.[1] In order to provide the highest quality of care and cost-effective treatment with this patient population, wound care clinicians must consider the biologic and psychosocial complexities of aging. Because many nonhealing wounds are the consequence of functional changes and diseases that accompany aging, it is of paramount importance that outpatient wound centers be equipped with the knowledge and infrastructure to meet this challenge and take a comprehensive, multidisciplinary approach to care. This article will provide guidance related to changes associated with aging skin as well as altered physiology and comorbidities that impact wound healing.

INTRINSIC & EXTRINSIC CAUSES OF AGING SKIN

Normal skin serves the human body with several critical functions, most notably providing barrier protection against physical and chemical insults. Adipose cells in subcutaneous tissue in concert with capillaries, arterioles, and vasodilatation

controlled by autonomic nerves provide thermoregulation. Skin and specialized sweat glands provide regulation of water loss while Langerhans cells, mast cells, and T and B cells provide protection from microorganisms. Specialized nerve cells provide sensation and give us signals about our environment. Vitamin D3 is produced in the skin, and sebaceous glands are important in testosterone metabolism.

Changes that occur to human skin as it ages are classified as intrinsic and extrinsic. Intrinsic refers to physiologic changes taking place in the aging process and are most evident in sun-protected areas, while extrinsic refers to environmental influences. It is sometimes difficult to separate intrinsic from extrinsic factors due to the pervasive impact of diet and lifestyle, but there are profound genetic and ethnic differences in the body's response to both.

One major factor impacting both intrinsic and extrinsic aging is oxidative stress leading to macromolecular damage and cell senescence. Changes of aging depend largely upon homeostasis between free radical production and the proper working of repair systems. The term reactive oxygen species (ROS) refers to free radicals and non-radicals that contain an oxygen atom. ROS are byproducts of cell respiration that takes place in mitochondria during oxidative phosphorylation, but are generated by other cellular structures such as peroxisomes and endoplasmic reticulum.[2] Not only does ROS increase with age, but aging is accompanied by reduced antioxidant activity and decreased DNA repair capability.

Extrinsic causes of aging skin are also mediated by oxidative stress and ROS. The most important cause of aging skin is ultraviolet (UV) light from the sun, which is divided into UVA and UVB. UVA is considered more damaging because of its deeper penetration into the dermis. Other environmental factors recognized as extrinsic causes of aging include cigarette smoke, ozone, and airborne particulate matter with adsorbed polycyclic aromatic hydrocarbons.[3] These extrinsic factors generate free radicals and ROS that overwhelm the body's natural antioxidant defenses and stimulate lipid proxidation reaction cascade, which in turn releases pro-inflammatory mediators that include matrix metalloproteinases (MMPs). Mitochondrial DNA mutations resulting from ROS leads to defective electron transfer activity and oxidative phosphorylation.

Telomere shortening is a known result of oxidative insults resulting from environmental factors and is associated with psychological stress.[4] Telomeres are repetitive DNA sequences at the end of linear DNA that shorten each time a cell divides and ultimately leads to cessation of cell division and apoptosis, or programmed cell death.

Polycyclic aromatic hydrocarbons adsorbed to airborne particulate matter induce xenobiotic metabolism that releases ROS and MMPs that accelerate aging. Xenobiotic metabolism refers to the metabolic pathways the body uses to eliminate environmental toxins such as polycyclic aromatic hydrocarbons that are adsorbed to airborne particulate matter.

CHANGES IN AGING SKIN

There are numerous changes in aging skin that impair the adaptive and homeostatic capacity and leads to increased susceptibility to environmental and internal stresses that lead to impaired wound healing and chronic wounds. These are discussed as follows:

In the epidermis there is reduced keratinocyte proliferation and turnover time, and surface pH is less acidic. Desquamation is less effective and lipid biosynthesis in the stratum corneum is impaired. There are decreased melanocytes that protect from UV radiation and decreased Langerhans cells that process microbial antigens and present them to other immune system cells. This is accompanied by altered T and B cell function and a general pro-inflammatory environment that is now an accepted component of the aging process.

The dermal-epidermal junction is flattened with smoothing of the rete ridges and decreased adhesion of this critical mechanical defense barrier. The dermis becomes atrophic with reduced numbers of fibroblasts and mast cells, and collagen becomes disorganized with change in synthesis from type 1-3. There is decreased synthesis of elastin and elastic tissue is degraded with overall loss of elasticity. Decreased mechanoreceptors including Meissner and Pacinian corpuscles result in diminished sensation to light, touch, pressure, and vibration.

There are decreased pilosebaceous units, which are composed of hairs, sebaceous glands, and arrector pili muscles that contribute to decreased sebum production. Impaired thermoregulation results from loss of subcutaneous fat, decreased autonomic nerves from the sympathetic nervous system, and decreased dermal vascularity. Loss of sweat glands also contributes to impaired thermoregulation as well as decreased ability to manage water balance in response to anti-diuretic hormone.

COMORBIDITIES OF AGING THAT IMPACT WOUNDS

Along with the accumulation of deleterious changes in aging skin, pathologic changes add to the risk for development of chronic wounds and impair the body's ability to heal. Changes in aging physiology decrease the body's reserve when stressed — a phenomenon known as homeostenosis. The aging body is more susceptible to injuries related to shearing forces, ischemia, pressure, and other forms of trauma. Alterations in barrier function, vascularity, and immune function make aging skin more susceptible to infection, both fungal and bacterial.

Disorders of the vascular system present common underlying factors in development of wounds and delayed wound healing, and arterial and venous ulcers are commonly seen in wound clinics. Venous insufficiency with increased venous pressure is a common result of post-thrombotic syndrome, with risk factors including obesity, pregnancy, inactivity, and gender. Atherosclerotic arterial disease reduces perfusion to the skin and results in suboptimal delivery of oxygen and nutrients. Risk factors for atherosclerosis include hypertension, smoking, hyperlipidemia, and diabetes mellitus.

Approximately 25% of people ages 65 and older are currently living with diabetes mellitus, according to the Centers

for Disease Control and Prevention, and common sequelae include neuropathy, microvascular disease, dysfunctional leukocytes, and altered inflammatory pathways.[5] The rising rates of obesity with the known association with type 2 diabetes will continue to have a great impact on public health and the prevalence of chronic wounds.

Other endocrine imbalances that accompany aging potentially impact wound healing. Hypothyroidism is common in geriatric patients, and a feature of this disease is dry skin from decreased sebum production and eccrine gland secretion. Thyroid hormone accelerates barrier function by increasing enzymes in the cholesterol sulfate cycle, and an evolving literature suggests this hormone is integral to cutaneous physiology.[6]

Reduced estrogen levels in post-menopausal women disturb tissue regeneration through impaired cytokine transduction.[7] The anabolic process of protein synthesis requires anabolic hormones that decrease with age such as testosterone, insulin-like growth factor 1, and human growth hormone. The decrease in normal anabolic hormone activity, along with an increase in catabolic hormones, occurs with aging and chronic illness and results in decreased lean body mass and impairment of the wound healing process.[8]

Physiologic and pathologic changes that decrease delivery of oxygen and nutrients to the skin will adversely impact wound healing. These include anemia, hypoxia, and low cardiac output states. Anemia of chronic disease is common in chronically ill elders and hypoxia results from chronic lung diseases such as COPD. Common causes of edema include congestive heart failure, venous insufficiency, and hypoalbuminemia leading to anasarca. The anatomic and biochemical changes associated with lymphedema also impair wound healing.

Nutritional depletion and altered functional status can contribute to skin fragility and impair wound healing as well. Urinary and fecal incontinence affects a large number of elderly patients. When left untreated, the skin becomes macerated and inflamed and leads to folliculitis, cellulitis, fungal dermatitis, and loss of integrity that can result in pressure ulceration. The wound clinic provider must recognize these factors and provide interventions in the form of patient and family education, discussions with nursing staff and other primary care providers, pressure-relieving interventions, and referrals to dietitians or rehabilitation specialists.

Elderly persons have often been exposed to pharmacologic agents that impair skin integrity or compromise the immune system, and this can additionally impact wound healing. Agents include chemotherapeutic drugs administered for malignancies and immunomodulators for diseases such as rheumatoid arthritis. Systemic corticosteroids and overuse of topical corticosteroids cause thinning and atrophy of the skin because of the suppressive action on cell proliferation and inhibition of collagen synthesis.

THE GERIATRIC APPROACH

The proper approach to caring for the geriatric patient living with a nonhealing wound is to take a comprehensive and multidisciplinary perspective,[9] including consideration of social support systems while taking into account religious and ethical beliefs as well as quality of life. Nutritional status and assessment of comorbidities should be a component of every initial visit and addressed in an ongoing fashion. Although healing is always the best outcome, palliation with symptom control and avoiding infectious complications may be an acceptable outcome.

The social worker assists in psychosocial evaluation, entitlement assessment, placement, advance directives, and supporting the patient and family emotionally. Rehabilitation specialists such as occupational, physical, and speech therapists assist with maximizing mobility and feeding abilities. The nutritionist can assist with optimal protein and caloric intake as well as hydration. Nursing staff is critical for dressing changes, choice of dressing, and reporting changes in wound status. Home attendants spend the most time with patients and often are the best frontline resources for information on intake, functional status, and changes in condition.

Recognizing the palliative wound and educating the patient and family on reasonable and realistic treatment choices is a major role of the wound clinic provider.[10] Palliative care is different from hospice and is not necessarily directed toward people who are actively dying. A palliative care approach to a nonhealing wound begins with education of the patient and family regarding rational choices followed by ongoing counseling and psychological support. Similar to a geriatric approach, the values, culture, and lifestyle of the patient must be taken into consideration when setting treatment goals. Feelings of guilt or failure among caregivers must be anticipated and dealt with in a proactive fashion. Treatment involves symptom control for pain, malodor, and exudate with stabilization of existing wounds while preventing additional wounds and infectious complications if possible. ■

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