Chapter 7 Upper Torso & Abdomen

"We don't stop exercising because we grow old...We grow old because we stop exercising." Mike Banks

Transition from the NFPE of the HEENT to the upper torso. The upper torso is anything above the waist and below the neck including the shoulders, arms, hands, chest, and back. Strength and endurance in the upper torso is one indicator of overall fitness. Muscle strength is defined as the ability of a muscle to contract and produce force in a single effort. Endurance is the amount of time a muscle can perform a specific task before failure. Malnutrition, obesity,



low physical activity, sarcopenia, injuries, surgery are things that can limit muscle strength. NFPE of muscle strength is performed to determine baseline strength compared to improvements following nutrition interventions (Bohannon, 2019; Ciesla, 2011).

Numerous medical conditions and medications are associated with muscle pain, muscle atrophy, and weakness. Refer to Tables 7.1-7.2. These conditions may or may not be concurrent with malnutrition. Pay close attention to signs of nutrition-related conditions based on data in the medical record and information gathered during the interview. Some data to note include a change in frequency and/or type of injuries or falls, changes in physical strength to do activities of daily living, increased fatigue, difficulties chewing or swallowing, and unplanned weight loss or gain. Document the presence of medical devices or dressings that limit hands-on access to the upper torso for NFPE (Campins, 2017; Janssen, 2020; Kalyani, 2014).

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Table 7.1. Medical Conditions Associated with Adverse Effects on Muscles

Medical Conditions	Medical Conditions
Amyotrophic lateral sclerosis (ALS)	Multiple sclerosis (MS)
Chronic kidney disease (CKD)	Muscular dystrophy (MD)
COPD	Osteoarthritis
Growth hormone deficiency	Osteoporosis
Heart failure	Parkinson's disease
HIV/AIDS	Peripheral artery disease (PAD)
Hypercortisolism	Rheumatoid arthritis
Hyperthyroidism	Spinal muscular atrophy
Hypogonadism in men	Stroke
Inflammatory diseases	Type 2 diabetes
Injuries	Vitamin D deficiency

Table 7.2. Drugs Associated with Adverse Effects on Muscles

Drug class	Examples
Analgesics	tramadol
Anti-anxiety	alprazolam
Antibiotics	sulfonamide, penicillin
Antidysrhythmics	amiodarone
Anti-epileptic	clonazepam
Antineoplastics	chemotherapy, leuprolide acetate
Antiretroviral	lamivudine, zidovudine
Anti-thyroid drugs	methimazole, propylthiouracil
Biguanides	metformin
Biological response modulators	interferon
Fibric acid derivatives	gemfibrozil
GI drugs	cimetidine, omeprazole
Illicit drugs	cocaine
Lipid-lowering agents	statins, atorvastatin
Sedative	zolpidem
Sulfonylureas & glinides	glipizide, nateglinide
Thiazolidinediones	rosiglitazone, pioglitazone

Table 7.3 offers some questions to better understand the client's perceived upper body strength and usual activity patterns.

Table 7.3. NFPE Questions/Observations: Activity Patterns & Upper Torso

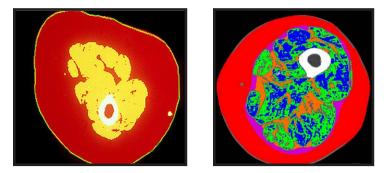
Global	Focused
Tell me about your usual routine every day.	Think about the last 24 hours. How many hours did you spend sleeping?
	Think about the last 24 hours. How many hours did you spend sitting?
	Think about the last 24 hours. How many minutes or hours did you spend doing light exercising?
	Think about the last 24 hours. How many minutes or hours did you spend doing moderate or vigorous exercising?
How would you describe your up- per body strength?	Has your upper body strength changed over the last year? (in- creased, decreased or no change)
	Do you have more difficulty open- ing jar lids or food packages than a few months ago?
	Do you have more difficulty picking up and carrying grocer- ies or other packages than a few months ago?
Do you intend to be more physically active in the next 3-6 months?	How do you plan to increase your level of activity?
Have you noticed a change in the way your clothing fits?	Is the client wearing clothing that is too large, i.e. shoulder seam far below the shoulder line or sleeves that hang below the tips of fingers

The upper body is more susceptible to muscle loss than other parts of the body. Loss of upper body muscle volume is largely independent of function. Muscles lose function when fat infiltrates the tissues. The muscles in the upper torso will atrophy with aging, lack of use, unplanned weight loss, and malnutrition. Patients often lose up to 25% of muscle mass in the first year following,

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bariatric and metabolic surgery (Ito, 2017). Losses are more evident in the upper body than other areas of the body.





Note: 7.1 Outer red area is fat, bone is white area with the dark center, yellow is muscle mass. 7.2 Outer red area is fat, bone is white area with the dark center, green areas are muscle.

Figure 7.1 is a cross section of muscle of a physically active, probably overweight, adult with some fat infiltration of the muscle. Figure 7.2 is a a cross section of muscle sedentary adult with considerable fat infiltration.

Case Study 7.1

- Observe the HEENT and upper torso of Case 7.1.
- What is your first impression client's overall nutrition status and general well-being?
- Does he look well-nourished or undernourished?

Observations of the HEENT and upper torso are at the end of the chapter.

Images from Deposit Photos and CDC PHIL



During the interview, ask the client about usual physical activity. In your practice, you may use a self-reported activity log or complete a validated exercise questionnaire. For most healthy adults, the Department of Health and Human Services (DHHS) recommends moderate intensity aerobic exercise of at least 150 minutes per week or 30 minutes per day. Strength training twice a week is also recommended. Examples of strength training include lifting weights, working with resistance bands, push-ups, sit-ups, digging and shoveling (DHHS, 2019).

Level of Exercise	Objective Measures	How Client Feels	Examples
Low-intensity	Heart rate is the same as it is at rest	Breathing is basi- cally unchanged You can talk and sing doing low intensity exercise	Walking slowly Sitting at the computer Eating food Cooking Making a bed
Moderate intensity	Exercise that raises heart rate up to ~50- 70% higher than heart rate at rest	Breathing quick- ens, but you are not out of breath May perspire after 10 minutes of activity You can talk, but you cannot sing doing moderate intensity exercise	Brisk walking Sweeping or vacuuming floor Water aerobics Riding bike Dancing Doubles tennis Mowing lawn Hiking
Vigorous intensity	Exercise that raises heart rate up to ~70- 85% higher than heart rate at rest	Breathing is deep and rapid Perspire readily after 10 minutes of activity You cannot say more than a few words without pausing to breathe doing vigorous intensity exercise	Running Shoveling snow Swimming Jumping rope Carrying heavy loads (bricks)

Table 7.4. Intensity of Exercise

Level of PA	Determining Level of PA	
Inactive	 Not getting any moderate or vigorous PA PA limited to basic movements for activities of daily living (ADLs) 	
Insufficiently Active	 Doing some moderate or vigorous PA < 150 minutes of moderate intensity PA/week or < 75 minutes of vigorous intensity PA/week 	
Active	 Doing 150-300 minutes of moderate intensity PA/ week 	
Highly Active	Doing >300 minutes of moderate intensity PA/week	

Table 7.5. Levels of Physical Activity (PA) in Healthy Adults

Table 7.6. Levels of PA in Adults with Special Needs

Special Needs	Determining Level of PA
Older adults	 Balance training Aerobic and muscle strengthening activities Do 150 minutes of moderate intensity PA/week Do what is consistent with client's medical condition and abilities
Pregnancy/ Postpartum	 Do at least 150 minutes of moderate intensity PA/ week Consult health care provider to determine appropri- ate level of PA intensity
Adults with Chronic Conditions	 Do at least 150 minutes of moderate intensity PA/ week or 75 minutes vigorous intensity PA/week Do what is consistent with client's medical condition and abilities

(DHHS, 2019)

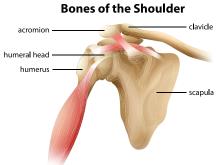
Practical Applications for NFPE: Upper Torso

NFPE of the upper torso includes observation and light palpation of the deltoid, trapezius, pectoral, supraspinatus, infraspinatus, biceps, triceps, interosseous, and thenar muscles. Ask client to engage or flex muscles, if able. Observe the client straight on with arms at each side to assess the deltoid muscles. Look for loss of roundness at the junction of the shoulder and neck and at the acromion process. Confirm you observations when you palpate the back muscles.

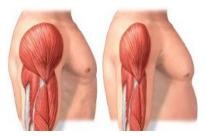
Figure 7.3 Deltoid Muscles

Figure 7.4 Acromion Process





Figures 7.5- 7.6 Deltoid Atrophy





In well-nourished individuals the deltoid curves are rounded at the junction of the shoulder and neck and at the junction of the shoulder and arm. The acromion process (highest point of the shoulder) is not evident. In moderate malnutrition the deltoid appears flat and there is a slight protrusion of the acromion process. In severe malnutrition, the deltoid is flat and acromion process is prominent or protruding suggesting significant loss of the deltoid muscle.

Next, observe the anterior trapezius muscles around the clavicle. In well-nourished men the clavicle bone is not prominent, but is often visible in women.

Images from Deposit Photos, Dreamtime, and CDC PHIL

Figures 7.7-7.8 Trapezius Muscles, Anterior & Posterior



Figures 7.9-7.11 Differences in Clavicle Prominence



Figure 7.12 Clavicle Prominence

In moderate malnutrition there is a slight protrusion of the clavicle. In severe malnutrition, the clavicle bone is prominent and protruding. Next, observe



the patient's chest region. Observe and palpate the pectoralis muscles. The pectoralis muscles connect the front walls of the chest with the bones of the upper arm and shoulder. There are two pectoralis muscles on each side of the sternum. In wellnourished individuals the curves of the pectoralis muscles are rounded and the sternum is evident, but does not protrude. In moderate malnutrition there may be a slight protrusion of the sternum, the ribs are somewhat prominent.

Images from Deposit Photos and CDC PHIL

Chapter 5 Tools for Assessing Skin, Hair & Nails

During a general survey of overall appearance, observe the skin on the face, arms and hands, nails and hair. The characteristics the RD is looking for are signs that might



suggest a nutrient deficiency or toxicity. Patients often notice changes in their skin, hair or nails before the clinician does. Some questions to consider include:

Skin:

- Have you noticed any changes in your skin?
- Does the skin have an increased (brownness) or decreased pigmentation?
- Does the skin have a red cast or tone?
- Does the skin have a blue cast or tone?
- Does the skin have a yellow cast or tone?

Figure 5.1. Follicular Hyperkeratosis (Vitamin A deficiency)

Figure 5.2. Pellagra (Niacin deficiency)





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- Evaluate the moisture of the skin.
- Does the skin look dry and scaly, or sweaty or oily?
- Is the skin warm or cold to the touch?
- Is the skin rough or smooth?
- Are there lesions or rash on the skin?
- Does the patient have acute surgical wounds?
- Does the patient have a history or skin breakdown?
- Does the patient have nutrition risk factors for skin breakdown such as low BMI, unplanned weight loss, low nutrient density intake?
- Is the patient at increased risk for prolonged pressure on bony prominences or other areas of the skin?
- Does the patient have skin breakdown?

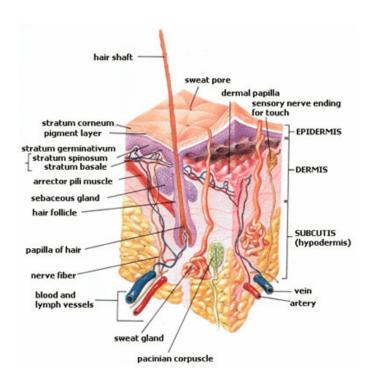


Figure 5.3. Anatomy of Skin

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The skin is the largest organ in the body that contributes about 10 percent of the total body weight. The skin is a bilayered organ that serves as the first line of defense against a variety of assaults. The epidermis is the thin outer layer and the dermis is the thicker inner layer.

Skin changes over time with age, exposure to sun, hydration, medications and nutrition. It is often ignored until it is injured. Skin and nails are thinner at birth, increase in density through adolescence. Once adulthood is reached, the dermis decreases in thickness by about 20 percent, but the epidermis is about the same thickness. However, the epidermal turnover time increases making wound healing a longer process. In young adults the epidermal turnover is about 21 days, however, by age 35 the turnover time is more than 40 days.

The skin of an older adult is more easily injured due to changes in the epidermal-dermal junctions. Skin elasticity also decreases with age. Collagen fibers appear to unwind and elastin fibers breakdown. The degradation of elastin begins to occur around age 30, but is markedly observed by age 70 (Braverman, 1986).

The barrier function of the skin decreases with age making skin irritations more common. Protection against ultraviolet radiation is diminished with age due to a reduced number of melanocytes which in turn contributes to wrinkling and sagging skin. The sagging is due in part to the loss of underlying tissue and the loss of elastin and collagen. Wrinkles are due to an increase in dryness of the skin.

The sensory receptors in the skin are diminished in capacity with aging. The risk of being burned or injured is more

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common due to a decreased sensation. Vitamin D production in the skin decreases with age and supplementation of vitamin D may be necessary for individuals with limited exposure to sunlight.

Nutrient deficiencies and toxicities can result in changes in skin color and texture and changes in the mucous membranes. While nutrient deficiencies are rare, these are reported in adults with malabsorptive disorders and following weight loss surgery. Toxicities are reported in individuals taking megadoses of vitamin mineral supplements.

Numerous medications affect the skin as noted on Table 5.1 (Potts, 1990). Corticosteroids are known to interfere with epidermal regeneration and collagen synthesis (Ehrlich, 1968).

Analgesics	• Diuretics
• Antibacterials	Hypoglycemic agents
Antihypertensives	Oral contraceptives
• Antihistamines	• Sunscreens
Antineoplastic agents	Tricyclic antidepressants
Antipsychotic agents	

Table 5.1. Categories of Medications that can Affect Skin

Skin & Nutrient Deficiencies

The skin color, texture, dryness, temperature, abnormal mucous membranes and presence of lesions or injuries are all characteristics to note in NFPA. Abnormalities should be corroborated with data from lab test results, anthropometric data, dietary history, vital signs and other clinical assessment data noted by the other medical team members Review the medical history for contributors to impaired nutrient utilization.

Practical Applications for NFPA: Color

The color of normal skin depends primarily on four pigments as noted in Table 5.2.

Pigments	Color
Melanin	Brown
Carotene	Golden yellow
Oxyhemoglobin	Bright red
Deoxyhemoglobin	Blue

Table 5.2. Skin Pigments

Changes in skin color may also be due to a number of medical conditions. Inspect the skin for abnormal color. Compare the findings to other evidence in the medical records that suggest a change in medical condition or functional status. The nutrition professional is looking for changes that suggest impaired ability to prepare meals, impaired functional status to eat, nutrient deficiency or nutrient overload.

Increased Brownness of Skin

The amount of melanin in the skin is genetically determined. It can also be increased with exposure to sunlight. Individuals with areas of darker, thick, velvety skin in body folds and creases may have acanthosis nigricans. It is more often seen in patients of African and Hispanic/ Latino descent. Acanthosis nigricans usually appears slowly and doesn't cause any symptoms other than skin changes. It results in velvety light-brown to black markings in body creases such as in the armpits, neck folds, and over the joints of the fingers and toes. Refer to Figures 5.4.-5.6. Acanthosis nigricans is seen in patients with obesity, insulin resistance, metabolic syndrome, hypertension, dyslipidemia, Addison's disease, disorders of pituitary gland, hypothyroidism and growth hormone therapy. It has been shown to be a reliable early marker for metabolic syndrome in children (Otto, 2010).

Weight management and improved glucose control can help prevent acanthosis nigricans as well as improve medical management of endocrine disorders (Kong, 2010). Patients with lymphoma, cancers of the gastrointestinal or genitourinary tracts can also develop severe cases of acanthosis nigricans.

Occasionally acanthosis nigricans is medication-induced. Medications associated with acanthosis nigricans, include nicotinic acid, insulin, pituitary extract, systemic corticosteroids, diethylstilbestrol, triazinate, oral contraceptives, fusidic acid, and methyltestosterone (Miller, 2011, Otto, 2010).

Increased Yellow Cast to Skin

Jaundice or icterus occurs when excess amounts of serum bilirubin dissolve in the subcutaneous fat, causing a yellowish appearance of the skin and the whites of the eyes. The yellowing may extend to other tissues and body fluids as well as turn the urine a darker color. Jaundice is not a disease, but a sign of a variety of medical conditions. It can occur when the normal process of destruction of red blood cells and elimination of bilirubin is interrupted. This occurs when there is excessive hemolysis, liver disease that reduces the ability of the liver to remove and modify bilirubin, or obstruction to the flow of bile into the intestine (Bickley, 2009). Refer to Figure 5.7. A lemon-yellow-tinged pallor to the skin is reported in pernicious anemia. Other physical signs and symptoms include raw-beefy red tongue lacking filiform papillae, paresthesia (sensation of pins and needles) in extremities, loss of sense of touch, changes in gait, stiffness in arms and legs, dementia, hallucinations and paranoia (Bickley, 2009). Refer to Figure 5.15 and Chapter 9.

Increased Yellow to Orange Cast to Skin

Carotene exists in subcutaneous fat. Hypercarotenemia or carotenemia is characterized by a yellow to orange skin color. It is due to consuming too much beta carotene supplements or eating too many dietary sources of beta carotene, (orange juice, carrots, carrot juice or pumpkin) In hypercarotenemia, the whites of the eyes remain white, unlike jaundice who have a yellowish tinge to the eyes (Schwartz, 2011). Refer to Figure 5.8.

Increased Bluish Cast of Skin

The form of hemoglobin that circulates without oxygen is called deoxyhemoglobin. Increased concentration in cutaneous blood vessels gives skin a bluish cast called cyanosis. There are two forms of cyanosis. If the arterial blood oxygen level is low, cyanosis is central. Central cyanosis is best identified in the lips, oral mucosa and tongue. In dark skinned individuals, look at the palms, nails and soles of feet. If the arterial blood oxygen level is normal, cyanosis is peripheral. Peripheral cyanosis occurs when the cutaneous blood flow decreases or slows and tissues extract more oxygen than usual from the blood. It is a normal response to anxiety or a cold environment.

Cyanosis that is seen in only one part of the body may be due to a blood clot. It is often seen in patients with chronic heart or lung problems (Bickley, 2009). Refer to Figure 5.9.

Decreased Color of Skin

The form of hemoglobin that circulates carrying oxygen is called oxyhemoglobin. Very pale skin or pallor and pale conjunctivae suggest a lack of oxygen possibly due to a nutritional anemia. See anemia section. Refer to Figures 5.10.-5.11.

Increased Redness of Skin

An increased redness of skin is caused by an increase in blood flow through the arteries to the capillaries causing a reddening of the skin. Blushing is a normal body response to strong emotion. Flushing of the face may be related to high fever, menopause, hyperthyroidism, rosacea, intense exercise, spicy foods, caffeine, niacin supplements, or medications. Increased erythema in other parts of the body may suggest inflammation, tissue injury, rash, skin disorder or infection (Bickley, 2009).

Clinical Nutrition Pearls: Skin Color

Diseases and disorders associated with changes in skin are medical diagnoses. The nutrition professional is identifying nutrition diagnoses. PES statements may incorporate NFPA observations specific to skin color, medical signs and symptoms related to changes in skin color, dietary data and anthropometric data. Nutrition diagnoses that may apply include altered nutrition-related lab test results, excessive or suboptimal intake of micro or macronutrients, excessive bioactive substance intake or harmful beliefs about food. Use NFPA findings as a benchmark to monitor and evaluate effectiveness of nutrition interventions.

Practical Applications for NFPA: Nutritional Anemias

Anemia is symptomatic of a disease and is a biomarker for increased morbidity, hospitalizations, mortality and increased healthcare costs. The prevalence of anemia increases with each decade of life over age 70 and is associated with both frailty and mobility impairment (AMDA, 2007).

A very pale skin color and pale conjunctivae are two signs of anemia. Anemia may be caused by acute or chronic blood loss, deficient erythropoiesis, or excessive hemolysis. There are four common types of nutrition related anemias;

- iron deficiency anemia
- pernicious anemia
- megaloblastic anemia
- anemia of chronic and inflammatory diseases

Other rare types of nutritional anemias include vitamin B₆ deficiency, an overload of zinc resulting in a copper deficiency or lead toxicity causing iron deficiency anemia.

Common symptoms of nutritional anemias include fatigue, unusually rapid heart rate, shortness of breath with exercise, difficulty concentrating, dizziness, leg cramps and insomnia. Individuals with iron deficiency may also have pica, soreness of the mouth with stomatitis and koilonychia (spooned nails). Iron deficiency anemia results from insufficient dietary intake or impaired utilization or a combination of factors (Santo, 1991)

Physical signs uniquely associated with pernicious anemia include lemon-yellow-tinged pallor with raw-beefy red tongue lacking filiform papillae (Figure 5.15), paresthesia in extremities, loss of sense of touch, changes in gait, stiffness in arms and legs, dementia, hallucinations and paranoia. Pernicious anemia results from insufficient dietary intake of vitamin B12 or impaired utilization of vitamin B12 or insufficient intrinsic factor required for absorption or a combination of factors.

Individuals with megaloblastic anemia may have increased weakness, impaired cognitive function, irritability and anorexia. Megaloblastic anemia results from insufficient dietary intake of folate or impaired utilization of folate or a combination of factors.

Individuals with anemias caused by inflammatory and chronic diseases may also complain of syncope, palpitations, cold intolerance, and anorexia. Chronic diseases associated with anemias include congestive heart failure, chronic kidney disease and other inflammatory disorders that result in redistribution of iron stores and poor iron utilization.

Vitamin B₆ deficiency contributes to an impaired transsulfuration of methionine to cysteine resulting in elevated levels of homocysteine. It is also required to convert folate into its active form for DNA synthesis. Vitamin B₆ deficiency is rare, but may be secondary to malnutrition, malabsorption, sickle cell anemia, alcoholism or use of pyridoxine-inactivating medications (i.e. anticonvulsants, isoniazid, cycloserine, hydralazine, corticosteroids and penicillamine). It is associated with microcytic anemia, dermatitis with cheilosis and glossitis, pellagra-like syndrome, peripheral neuropathy depression and confusion, and weakened immune function.

Signs and symptoms of anemia caused by lead poisoning

include a blue-black line on the gums (Figure 5.17. lead line), abdominal pain, constipation, nausea, vomiting changes in mood, peripheral neuropathy, memory loss and encephalopathy. Lead interferes with iron utilization and dietary supplements are ineffective (Bickley, 2009).

Copper deficiencies are rare, but can be the result of zinc overload or malabsorption. Cooper deficiencies have been reported in individuals following malabsorptive weight loss surgeries. Physical signs of a copper deficiency include numbness in lower extremities, abnormal gait, myelopathy, poor wound healing or dehiscence of wounds. Copper related anemia does not respond to iron or vitamin B12 supplementation.

When an anemia is suspected, look for other corroborating evidence in the medical record or from the interview. Review lab reports for abnormal test results for hemoglobin, hematocrit, serum iron, ferritin and MVC. Determine if the anemia is microcytic, normocytic or macrocytic. If labs suggest macrocytic anemias or vitamin B12 deficiency, additional tests are needed including serum vitamin B12, methylmalonic, acid and folate. Review medications for folate antagonists.

For individuals with suspected lead-poisoning, review lab reports for abnormal test results for hemoglobin, hematocrit, serum iron and ferritin since lead interferes with the absorption of iron. Follow up testing is needed for confirmation to include lead levels and zinc protoporphyrin.

Abnormal labs reported in copper-related anemia include very low hemoglobin, hematocrit and leukocytes. However, MCV, vitamin B12, folate, ferritin and platelet count are

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usually within normal ranges. **Figures 5.11-5.18** are examples of physical signs associated with nutritional anemias.

Figures 5.4.-5.6. Acanthosis Nigricans



Figure 5.7. Jaundice

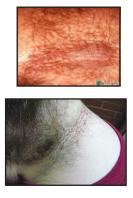


Figure 5.8. Hypercarotenemia



Figure 5.9. Cyanosis

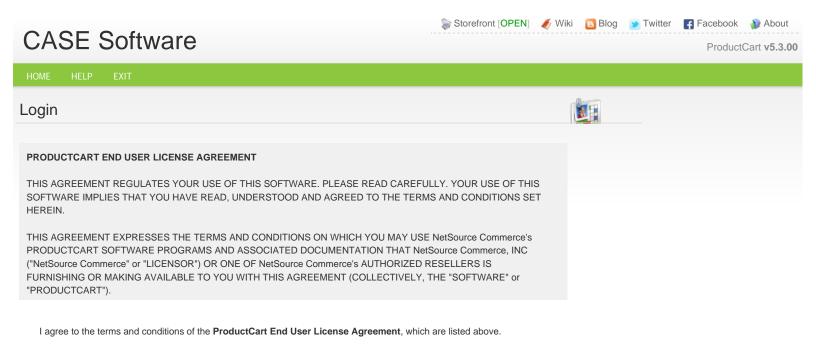




Figure 5.10. Pallor



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