

# Alterations in oxygen transport: pernicious anemia

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The paper "Alterations in Oxygen Transport: Pernicious Anemia" is a good example of a case study on nursing. This paper uses a case scenario involving a 56-year old patient to develop a comprehensive understanding of pernicious anemia. This autoimmune disorder is one of the most common causes of cobalamin (vitamin B12) deficiency in the world and is historically typified by gastric mucosal atrophy, discriminatory loss of parietal and chief cells from the gastric mucosa, as well as submucosal lymphocytic infiltrate (Kumar, 2007). It is of immense importance for the nursing practitioner (NP) to ask the patient about paresthesias and ataxia as these two sensory abnormalities underscore deficiencies of vitamin B12 in patients (Kumar, 2007). Owing to the fact that vitamin B12 deficiency causes pernicious anemia, the NP will be better placed to verify a diagnosis of pernicious anemia if the patient reports sensations of tingling, numbness, crawling, deadness, constriction in the abdomen and chest, or gait abnormalities. In terms of pathophysiology, vitamin B12 is an important component in the formation of red blood cells (erythrocytes) which are required to transport oxygen from the lungs to other organs of the body. Patients with paresthesias and ataxia (e. g., unsteady gait, numbness, etc) are likely to have a vitamin B12 deficiency as this important vitamin is needed for the proper functioning and health of nerve tissue. As such, knowledge of whether the patient has episodes of paresthesias and/or ataxia will be instrumental in identifying vitamin B12 deficiency, which in turn is known to cause pernicious anemia (Rote & McCane, 2014; Toh & Alderuccio, 2014). Research has demonstrated that individuals suffering from pernicious anemia are unable to generate adequate amounts of IF (intrinsic factor) in their stomach

to aid in the absorption of vitamin B12 which is needed to make red blood cells (Toh & Alderuccio, 2014). As such, individuals with pernicious anemia need to be facilitated to absorb vitamin B12 using other mechanisms due to the incapacity of IF to bind vitamin B12 and also to assist in its absorption. Drawing from this explanation, it is clear that the NP prescribed vitamin B12 by intramuscular injection rather than orally to aid in its absorption since the patient's digestive system is unable to absorb B12 properly. It is important to note that the loss of stomach cells (parietal cells) that nurture and develop the IF may be due to a host of factors, including obliteration of the body's own immune system, untreated stomach infections, and surgery to the stomach or small intestine (Rote & McCane, 2014; Toh & Alderuccio, 2014). Research is consistent that pernicious anemia is strictly an autoimmune disorder that is caused by insufficient levels of IF in the stomach or a genetic defect that prevents young children from making IF, resulting in vitamin B12 deficiency (Toh & Alderuccio, 2004). If the patient in the case scenario is diagnosed with paresthesias and ataxia in addition to the described symptoms (fatigue, pallor, dyspnea with exertion and palpitations), for example, the NP can make a firm conclusion that she is suffering from pernicious anemia arising from the inability of her body to absorb vitamin B12. As already mentioned, vitamin B12 insufficiency can be caused by inadequate diet (e. g., meat, eggs, and milk), the incapacity of the small intestine to absorb vitamin B12 due to surgery or abnormal bacterial growth, as well as certain intestinal diseases such as the Crohn's disease. Anemia with high mean corpuscular volume (MCV) values (e. g., exceeding 100 femtoliters) is technically referred to as megaloblastic anemia, while anemia

with normal MCH values (27-31 picograms/cell) is referred to as normochromic anemia (Aslinia, Mazza, & Yale, 2006). Drawing from this description, it is evident that the patient's pernicious anemia is megaloblastic in nature due to the high MCV which is often precipitated by vitamin B12 or folate deficiencies.