

# Sickle Cell Anemia: A Fictional Reconstruction\*

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## Part I – The Inquiry Begins

It was a brisk fall day in Boston—the type of day that Dr. William Castle preferred to start with a cup of coffee while he caught up on his correspondence, which often appeared to be an endless task. As a faculty member of Harvard Medical School, he had always received a fair amount of inquiries, but after he had published his data indicating that pernicious anemia was due to a vitamin B<sub>12</sub> deficiency, the amount of mail he received was sometimes overwhelming.

Sitting in his reclining chair he sorted through the large pile that had accumulated. He began to meticulously segregate it into smaller piles he would open in a prescribed order. Usually the delegation of a particular envelope was a relatively easy choice. There were a few that caused him to pause for a moment, such as the one he was currently holding. The return address indicated it was from an Irving Sherman at Johns Hopkins University. Since it was from someone he had never heard of, he was inclined to place it in the pile to be opened later. But on this day he decided to take another sip of coffee and see what Mr. Sherman had to say (see next page).



\* *Disclaimer:* This case is a work of fiction that refers to real events and people. All of the discoveries mentioned in Section 1 were made by the individuals they are attributed to, as were the observations made by Dr. Vernon Hahn described in Section 2. The time between discoveries has been dramatically condensed, however. Every effort has been made to present the scientific considerations concisely and accurately. Any errors should be attributed to the author and not the original investigators.

# Sickle Cell Anemia A Fictional Reconstruction Answer

**Fernando Ferreira Costa, Nicola Conran**

## **Sickle Cell Anemia A Fictional Reconstruction Answer:**

**Sickle Cell Anemia** ,1990      **Sickle Cell Anemia** Judy Monroe Peterson,2008-08-15 Sickle cell anemia is a genetic disease of the blood It is caused by a defect in one gene of a person Genes are the elements in cells that carry the information that determines traits such as hair or eye color In sickle cell anemia a defect in the gene controls how hemoglobin is made This defect can be passed from parents to their children Students explore the history of sickle cell anemia the pioneering doctors who studied its cause and early treatments They also investigate hemoglobin S who gets sickle cell and how the gene mutation is inherited They learn about the different types of sickle cell disease and treatments including blood transfusions and bone marrow transplants and some of the promising new research in medicines and gene therapy      [Answers to Common Questions about Sickle Cell Disease](#) ,1973      **Sickle Cell Anemia** Fernando Ferreira Costa,Nicola Conran,2016-03-29 Although sickle cell anemia was the first molecular disease to be identified its complex and fascinating pathophysiology is still not fully understood A single mutation in the beta globin gene incurs numerous molecular and cellular mechanisms that contribute to the plethora of symptoms associated with the disease Our knowledge regarding sickle cell disease mechanisms while still not complete has broadened considerably over the last decades Sickle Cell Anemia From Basic Science to Clinical Practice aims to provide an update on our current understanding of the disease s pathophysiology and use this information as a basis to discuss its manifestations in childhood and adulthood Current therapies and prospects for the development of new approaches for the management of the disease are also covered      [Focus on Sickle Cell Research](#) Ralph L. Plasmar,2004 Sickle cell anaemia is an inherited blood disorder characterised primarily by chronic anaemia and periodic episodes of pain and occurring in approximately 1 in every 400 African American infants born in the United States each year Individuals of Mediterranean Arabian Caribbean South and Central American and East Indian ancestry can also be affected The underlying problem involves haemoglobin a component of the red cells in the blood The haemoglobin molecules in each red blood cell carry oxygen from the lungs to the body organs and tissues and bring back carbon dioxide to the lungs In sickle cell anaemia the haemoglobin is defective After the haemoglobin molecules give up their oxygen some of them may cluster together and form long rod like structures These structures cause the red blood cells to become stiff and to assume a sickle shape Unlike normal red cells which are usually smooth and donut shaped the sickled red cells cannot squeeze through small blood vessels Instead they stack up and cause blockages that deprive the organs and tissue of oxygen carrying blood This process produces the periodic episodes of pain and ultimately can damage the tissues and vital organs and lead to other serious medical problems Unlike normal red blood cells which last about 120 days in the bloodstream sickled red cells die after only about 10 to 20 days Because they cannot be replaced fast enough the blood is chronically short of red blood cells a condition called anaemia Sickle cell anaemia is caused by an error in the gene that tells the body how to make haemoglobin The defective gene tells the body to make the abnormal haemoglobin that results in

deformed red blood cells This book gathers the latest research in this important field      **Understanding Sickle Cell Disease** Miriam Bloom,1995 For general readers a guide to understanding a debilitating genetic disease that affects tens of thousands who are of African heritage      Menace in My Blood Ola Tamedu,2011      Sickle Cell Anaemia Organisation for Sickle Cell Anaemia Research,1979

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