This paper combined a presentation of experimen-
tal data concerning internal iron metabolism with a
comprehensive clinical analysis of idiopathic
hemochromatosis. Also included was information
concerning the hereditary nature of this disorder and
evidence that repeated phlebotomies could
remove excess tissue iron with resultant clinical
improvement. [The SC® indicates that this paper
has been cited in over 420 publications since
1955.]

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The pathology curriculum at the University
of Rochester School of Medicine and
Dentistry in the 1930s and early 1940s was
heavily influenced by the interest in iron
metabolism of its eminent dean and Nobel
laureate, George Hoyt Whipple.1 He had
demonstrated that most excess tissue iron of
dogs could be depleted by chronic phlebot-
omy.

Paul Hahn, working in that department,
wondered whether the tissue iron in idio-
pathic hemochromatosis might also be re-
moved and repeatedly bled a patient with
that disease. His attempt to mobilize iron
through creating anemia appeared to be
unsuccessful and phlebotomies were aban-
donned. In retrospect, the patient had a he-
patoma that undoubtedly prevented iron
mobilization.

A few years earlier, my cousin, Clement
Finch, had been a student fellow in
Whipple’s laboratory. There he developed
an interest in the field of iron metabolism,
which subsequently became his lifetime ma-
ajor line of investigation. In the mid-1940s,
while at the Peter Bent Brigham Hospital in
Boston, he undertook iron loading of experi-
mental animals and successfully mobilized
the iron by phlebotomy. On the basis of
these various studies, he and a research
fellow, Charles E. Rath, cautiously initiated a
series of phlebotomies on a patient with
hemochromatosis. The patient was an em-
ployee of the Internal Revenue Service who
was of importance to us in more ways than
one. It proved possible to bleed him 500 ml
weekly without significant anemia develop-
ing.

At about that time, I joined the team, and
in the course of the next few years, a number
of other patients with hemochromatosis
were phlebotomized. Some family studies
were carried out and additional observa-
tions were made in man and experimental
animals.2,3 In the phlebotomized patients,
the lack of progression of their diseases and
even improvement in clinical status gave
credence to the belief that the tissue dam-
age seen in idiopathic hemochromatosis
was simply the result of excessive iron stores.

In late 1948, our laboratory was fortunate
to have a visit from J.H. Sheldon, a practic-
ing country physician in England who in
1936 had written a classic monograph on
hemochromatosis. He urged us to write a
comprehensive summary of both our experi-
mental studies and clinical observations. It
was not until 1955 that all clinical hemo-
chromatosis papers were reviewed and the
paper was finished.

There are probably several reasons the ar-
ticle has been frequently cited. It was a com-
prehensive article in which differences be-
tween excess tissue iron and clinical hemo-
chromatosis due to long-standing tissue iron
deficiency were emphasized. Perhaps the
most appealing aspects of the paper were 1)
the experimental information on storage
iron and its mobilization, 2) a comprehen-
sive summary of the various clinical presen-
tations and complications of idiopathic
hemochromatosis, 3) information concern-

ing therapeutic phlebotomy and the labora-
tory changes that occur, and 4) the demon-
stration that the disease was potentially
reversible through the removal of excess
iron. The genetic information summarized
was more provocative than conclusive, but
it may have set the stage for subsequent
clinical studies that have clearly defined the
genetics and additional clinical aspects of
familial hemochromatosis.4

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3. Haskins D, Stevens A R, Jr., Finch S C & Finch C A. Iron metabolism: iron stores in man as measured by