ported. The authors did not specify the type of CO-oximeter used for the initial analysis. However, from the tabulated data the reader is apt to conclude that the measurement was done on an IL 482 instrument (Instrumentation Laboratory, Milan, Italy).

The IL 282 and IL 482 CO-oximeters have not been constructed for determination of sulfhemoglobin. Nevertheless, these instruments can give a strong indication of the presence of sulfhemoglobin in a blood sample on the basis of a combined positive result for methemoglobin and a negative value for carboxyhemoglobin. This bizarre phenomenon was first observed by Zwart et al. and published in an evaluation report of the IL 282 CO-oximeter in 1981 [2]. In the IL 282 and IL 482 operator’s manuals, it is mentioned that these instruments were not constructed for the determination of sulfhemoglobin and that sulfhemoglobin may interfere with the determination of methemoglobin. The wording of the statement, however, may be unclear: “The spectral absorbance of sulfhemoglobin is similar to that of methemoglobin and the same limitations would apply. Sulfhemoglobin is not commonly found in blood, however, making such interference rare.” We would favor wording such as: “The spectral absorbance of sulfhemoglobin is similar to that of methemoglobin. Therefore, the presence of sulfhemoglobin in a blood sample will cause a positive interference or false-positive result for methemoglobin. The presence of sulfhemoglobin in a blood sample is suspected whenever a methemoglobin result is displayed ≥10%, combined with a negative carboxyhemoglobin value.” IL now markets the model 682 CO-oximeter, which will detect and actually indicate sulfhemoglobin concentrations >1.5%, making this clarification in the new instrument’s manual unnecessary. However, manufacturers could help users if they clearly mention all known clinically relevant limitations in instrument manuals and update the manuals with all essential information published in the scientific literature.

References


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Hair Iron Content: Possible Marker to Complement Monitoring Therapy of Iron Deficiency in Patients with Chronic Inflammatory Bowel Disease?

To the Editor:

Bisse et al. [1] suggest that measurement of the concentration of iron in hair might prove useful in assessing iron status. They state: “To our knowledge, however, no attempt has been made to include the measurement of hair iron in an evaluation of body iron.” Older references are sometimes difficult to locate, but may be very important. In 1945 Flesch and Rothman found that trichosiderin is a red hair pigment that contains iron [2]. This finding suggests to some of us who were interested in iron metabolism that the use of hair iron concentrations would not be a satisfactory indicator of patient iron status. However, several investigators did investigate the iron content of hair as an index of iron status. In 1956, Green and Duffield [3] measured the iron content of the hair of patients with pulmonary tuberculosis, chronic rheumatoid arthritis, diabetes, pernicious anemia, hemochromatosis, and, notably, 53 patients with iron deficiency. In their investigations they took into account the color of the hair of their patients. Nonetheless, they concluded that “The results of this study would justify the conclusion that the iron content of scalp hair gives no indication as to the state of the body iron stores in an individual patient. While serial analyses of hair samples suggested that in some individuals the iron content of the hair fell when they became iron depleted and rose as iron stores were restored, this was not a consistent observation.” Later, Lovric and Pepper [4] quantified the iron of various segments of hair shafts of children, comparing the hair iron of 22 controls, 60 children with iron deficiency, and 9 children with iron overload. They concluded “...no significant differences in iron levels could be elicited in the three groups of children studied.”

The data of Bisse et al. are interesting, but in view of the extensive earlier observations, it seems to me unlikely that hair iron concentrations will prove useful in assessing body iron stores.

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References


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Two of the authors of the paper referred to above reply:

To the Editor:
We appreciate the interest expressed by Beutler in our recent publication [1], particularly as the references
[2–4] quoted in his letter were unknown to us.

We believe that the topic is so important that it deserves a wider base than that published in the quoted references.

Regarding the hair pigment trichosiderin, Flesch and Rothman [2] found this substance only in red-haired subjects. The structure as well as the biological function of trichosiderin have not yet been elucidated. In our study we examined no red-haired individuals. But if one considers the world population at risk of iron deficiency as a whole, the proportion of red-haired individuals is small. Despite some differences in hair iron concentrations that could be related to hair color [5], it is speculative to regard trichosiderin as a potential hindrance in the clinical use of hair iron concentration. Moreover, Creason et al. [5] found that vanadium and iron in hair were significantly correlated. This finding is interesting because vanadium is bound, as is iron, to transferrin in blood.

The main problem in measuring trace elements in hair is the absence of a scientifically based reference procedure, and hair iron values differ considerably among laboratories. Before examining hair iron in patients and in healthy subjects, the iron status of all subjects should first be clearly described and staged by the currently established tests. The quality management of the data is also very important. Unfortunately the authors quoted by Beutler have used only serum iron for the evaluation of iron status. Therefore they could neither clearly characterize the groups nor distinguish between iron-deficiency anemia and the anemia related to infections, inflammation, or malignancy. This and the analytical problems may partially explain the inconsistent trends observed [3, 4].

The low ferritin concentrations found in our patients clearly indicate that the organism was not basically influenced by the inflammation. Thus our results allow no general statement for patients with chronic bowel diseases or patients with other inflammatory diseases.

Extended studies that include different groups of patients as well as healthy individuals must be performed before we can obtain deeper insight into the metabolic pathway of hair iron. The iron status must be evaluated by using transferrin receptor and zinc protoporphyrin in addition to the conventional tests of iron status.

Until solid experimental data are available, it is premature to reach final conclusions on the clinical value of hair iron concentration.

References

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To the Editor:
It is not uncommon for investigators to incline toward the view that any studies performed >10 years ago must be flawed because of the primitive nature of science before current technology was available. However, hematologists were able to diagnose iron deficiency quite accurately even 40 years ago [1] without the advantage of serum ferritin concentrations. Hematologists such as Green and Duffield, and Lovric and Pepper were well qualified to make the distinction between the anemia of iron deficiency and that of inflammation, and I do not believe that their findings should be dismissed lightly merely because they were performed many years ago.

Reference

Equivalence of Critical Error Calculations and Process Capability Index $C_{pk}$

To the Editor:
The concept of process capability has been used by the manufacturing industry to quantify the relation between product specifications and the measured process performance [1]. Various ratios and indices have been developed to describe this relation. We have previously reported the application of the simplest of these, $C_p$ (the capability index or capability ratio), to the selection of quality-control (QC) algorithms appropriate to the specification limits and analytical imprecision [2]. $C_p$ is defined as $(USL - LSL)/6\sigma$, where USL and LSL are the upper and lower specification limits of an analytical process, and $\sigma$ is the standard deviation of the process.

In contrast to the approach taken by us, the use of medically important critical systematic error ($\Delta SE_c$) and critical random error ($\Delta RE_c$) calculations for the selection of QC algorithms has been promulgated [3–5]. Westgard and Burnett [6] have also described the relation between $C_p$ and $\Delta SE_c$ and have shown that, assuming zero bias, $C_p$ can be directly related to $\Delta SE_c$ by equation:

$$\Delta SE_c = 3C_p - z \quad (1)$$

where $z$ is a factor for a one-tailed test of significance (usually set at 1.65 for 95% confidence, assuming a Gaussian distribution).