Letter to the Editor

RE: “LONG-TERM EFFECTS OF SMOKING ON RETINAL MICROVASCULAR CALIBER”

As a clinical ophthalmologist, I was honored to be the corresponding author of the first publication to introduce into the literature in 2005 the clinical observation that smoking was positively associated with retinal venous dilation (1, 2). I believe it is instructive, as one reviews the origins of medical thinking, to know its genuine background.

My most memorable demonstration to medical colleagues of smokers’ veins occurred during Neurology Grand Rounds at Concord Hospital in Sydney, Australia, in 1993. I was asked to examine a patient’s ocular fundus. At the meeting was the finest neuro-ophthalmologic thinker and teacher that Australia has produced, Dr. Medduma B. Kappagoda (3)—my original neuro-ophthalmologic mentor.

It was almost certainly he who suggested the concept of smokers’ veins. From the podium, I was able to indicate that the patient had dilated retinal veins and may well have been a smoker. I suggested that he may have smoked 18–20 cigarettes per day. The patient was impressed. This was followed in short order by a question from the neurologic floor, requesting me to divulge a response to “Which brand?”

Coincidentally, this was the precise year in which the Blue Mountains Eye Study in Sydney, led by Professor Paul R. Mitchell, was in the throes of initial data collection, just 2 years before the study’s first publication (4). Thus, I was delighted to read in the December 1, 2007, issue of the Journal the report by Kifley et al. (5), Professor Mitchell’s research group. These epidemiologists documented a moderately statistically significant correlation between derived central retinal vein equivalent measurements in a large study of smokers and their level of smoking. Thus, this statistical correlation was published 8 years after I initiated the first of several communications with Professor Mitchell in 1999, suggesting to him its possibility and potential value.

One of Kifley et al.’s main conclusions was interesting. They found that the mean retinal venular caliber (MRVC) (derived from the central retinal vein equivalent) of current smokers was 236 μm (see their Table 3) (5). By contrast, the nonsmokers’ MRVC was 226 μm. Surprisingly, and somewhat counterintuitively for me as a clinician, this was statistically significant up to P values of <0.0001 (Tables 3 and 5) (5). Indeed, the MRVC difference of only 14.2 μm between the 2 cohorts would seem to be a difference that is well nigh impossible to detect clinically. In other words, even the most astute clinician would probably not be able to detect that difference in the state of dilation of the retinal veins, despite utilizing the latest hand-held condensing lens and the best slit lamp biomicroscope.

By contrast, my colleagues and I have found that clinical inspection of the retinal venous dilation of a smoker generally provides a striking observation (1, 2) and in fact is usually quite difficult to miss. Given Kifley et al.’s findings (5), the reason for the difference between the observations of these epidemiologists and we clinicians is, at this stage, unclear and may be worth further investigation. Kifley et al.’s observation is, however, consistent with that of a recent study (6) which showed that in some patients with reduced cerebral blood flow, a tiny, clinically indistinguishable difference of 5 μm occurred when reduced arterial oxygen saturation was associated with larger retinal venular diameters.

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REFERENCES

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Editor's note: In accordance with Journal policy, Dr. Kifley and colleagues were asked whether they wished to respond to this letter, but they chose not to do so.

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