bars in the test grating appear wider. However, as indicated by the demonstration in Fig. 1 and the data of Blakemore and Sutton (*1*), adapting to gratings lower in spatial frequency would increase the apparent spatial frequency of a test grating. Thus, a paradox is presented by comparison with the data in Fig. 2: Adapting and test grating frequencies that lead to increases in apparent spatial frequency (for example, 3:2) also increase the apparent width.

Earlier results had presented a similar paradox along with a plausible solution. A process of adaptation that made a set of bars appear to be of higher spatial frequency (*1*), made a single rectangle appear to be wider (*2*). The following rationale for these results was offered, based on assumed frequency channels in the visual system: While the notion of width for a single bar is simple, the spectrum of the bar is more complex than that of a grating. Adaptation to a sinusodial grating is adaptation to a single frequency. The resulting loss in sensitivity around that frequency produces different shifts in the "center of gravity" of the effective spectra of a single rectangle and of gratings, and thus different shifts in apparent size and frequency. The earlier paradox was thus resolved.

But there is no way to rationalize the current findings of inconsistent shifts in apparent size and frequency by appeal to a difference in stimulus spectra, since the same stimulus (multiple bars) was used for both frequency and width judgments. Nor can one escape the paradox by noting that adaptation could increase apparent spatial frequency while increasing the apparent width of the light bars at the expense of that of the dark bars (that is, by altering the apparent duty cycle). No shifts in apparent duty cycle were found when subjects adjusted apparent duty cycle before and after adaptation (*3*). The paradox remains; identical stimuli result in perceptions of both higher spatial frequency and wider size.

We suggest the following resolution: Assume that the observer changes the effective spectrum at will. Attention is a sufficiently familiar (and loosely defined) mechanism for the purpose. Carpenter and Ganz (*4*) have used a similar hypothesis in a not-too-different context. We propose that, when observers are asked to "make a square" with either a single rectangle or multiple rectangles (our shortened grating), they discount all parts of the visual field except those relevant to their task. In terms of frequency, something like a $(\sin x)/x$ function—the window function—might be imposed on an observer's visual system. (The exact nature of the function surely differs. Our data do not suggest a more complete description.) As a result, when observers are asked to make a square, a single rectangle is attended to, whether it is alone or in the presence of other rectangles. Thus, the effective spectra for single and multiple rectangles are much the same (as are the experimentally obtained settings).

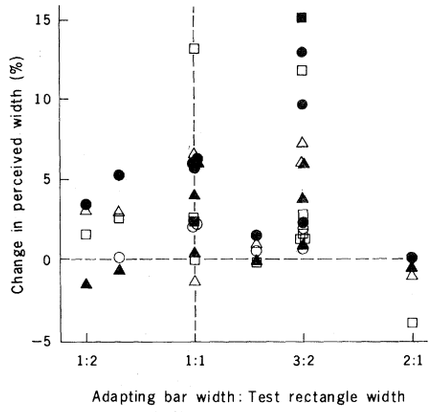

Fig. 2. Percentage increase in perceived widths of rectangles in a short grating after adaptation to a long grating. Different symbols indicate different observers.

The results from the earlier experiments with the single rectangular test stimulus (*2*) indicated that the visual system encodes size on the basis of spatial frequency components. It is natural to think that such a spatial frequency analysis would be performed over a wide region of the visual field (*5*). However, the present results and proposed interpretation suggest that viewers can, when necessary, restrict their spatial frequency analysis to a limited region of the visual field. This selective spatial filtering would reflect an active process, which may provide a link between spatial frequency analysis and selective attention.

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Legionnaires' Disease: Concentrations of Selenium and Other Elements

Abstract. *Selenium concentrations in the serums of 17 acutely ill Legionnaires' disease patients were significantly lower than in their matching convalescent-phase serums. This trend was not observed in ten similarly paired samples of serum from control patients with pneumonia. There were no significant differences in the concentrations of nickel, copper, bromine, rubidium, lead, barium, or titanium in the serums of Legionnaires' disease and control patients.*

We previously reported toxically high nickel concentrations in lung specimens taken at autopsy from the first victims of Legionnaires' disease (*1*). We also reported a significant correlation ($P < .0004$) between nickel concentrations and the dry weights of the lung specimens. We concluded that the high nickel concentrations were probably not caused by a toxic substance but rather by exogenous nickel contamination occurring after death. Subsequently, a bacterium was identified as the etiologic agent in Legionnaires' disease (*2*).

We report here the concentrations of selenium and other elements in 17 paired samples of serum from the acute and convalescent phases of Legionnaires' disease patients and in ten similarly paired samples of serum from control patients with pneumonia. The selenium concentrations in the acute-phase serums of the Legionnaires' patients were significantly lower ($P < .001$) than those in the matching convalescent-phase serums. This trend was not observed in paired samples of serum from acute- and convalescent-phase controls. We found no significant differences in the serum nickel concentrations of the Le-

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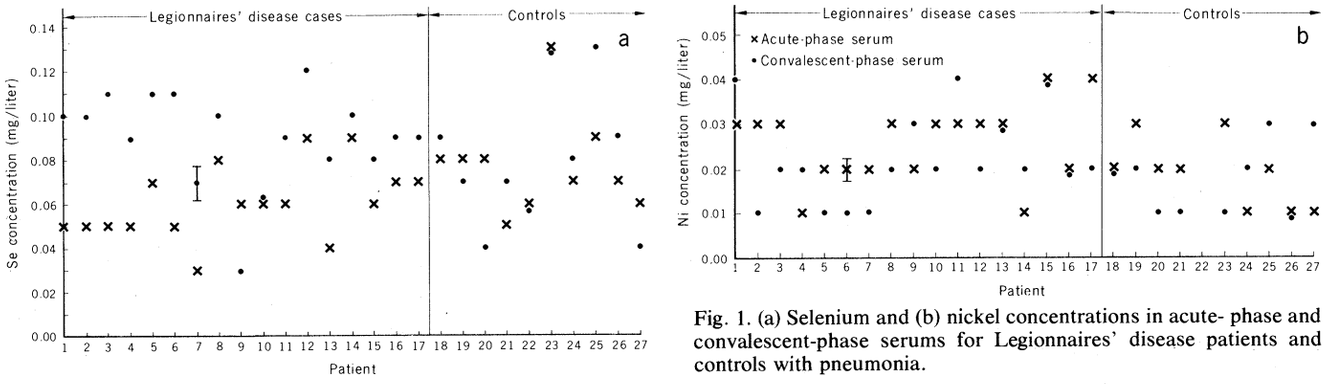

Fig. 1. (a) Selenium and (b) nickel concentrations in acute- phase and convalescent-phase serums for Legionnaires' disease patients and controls with pneumonia.

gionnaires' patients and the controls. Also, we found no significant differences in the serum copper, bromine, rubidium, lead, barium, or titanium concentrations.

We used the method of proton-induced x-ray emission (*3*) to measure the concentrations of elements in the serums. Duplicate portions (30 μl) of each serum were evaporated to dryness and analyzed directly in a 2-MeV proton beam. As described in (*3*), the proton-induced characteristic x-rays were detected in an Si(Li) detector and were normalized to the number of scattered protons detected in an Si surface-barrier detector. Results for all elements were obtained simultaneously during a single run. We performed quantitative calibration and cross-check measurements by analyzing serum samples to which known quantities of selenium, nickel, copper, bromine, rubidium, lead, barium, and titanium had been added.

The 17 Legionnaires' cases consisted of ten "sporadic" cases (patients 1 to 10) and seven "Philadelphia" cases from the 1976 outbreak in Philadelphia (patients 11 to 17). Legionnaires' disease was confirmed on the basis of serologic tests in which indirect fluorescent antibody (IFA) staining was used (*4*). The cases analyzed were selected at random from a pool of cases in which IFA titer increases ranged from 4- to 128-fold and reached a minimum titer of 64. Titers were expressed as the reciprocal of the greatest dilution with distinct fluorescence.

The control patients (patients 18 to 27) were selected from a group initially suspected to be infected with Legionnaires' disease but later shown not to be infected by the absence of a rise in IFA titers. All controls, however, showed chest x-ray evidence of pneumonia, but the etiologic agent was not known. Since the controls were initially thought to have Legionnaires' disease, the handling and storage of all serums were similar.

Figure 1a shows the serum selenium concentrations for the Legionnaires' patients and the controls (*5*). The selenium concentrations in the acute-phase serums of the former are systematically lower than in the matching convalescent-phase serums ($P < .00007$, t-test for correlated paired samples). Since comparisons were made for eight elements in both Legionnaires' patients and controls, the significance level was reduced to $P < .001$ (*6*). The average selenium concentrations in serums of Legionnaires' patients ($N = 17$) and controls ($N = 10$) were as follows: in acute-phase serums, 0.06 ± 0.02 and 0.08 ± 0.02 mg/liter, respectively; in convalescent-phase serums, 0.09 ± 0.02 and 0.08 ± 0.03 mg/liter, respectively.

Figure 1b shows the serum nickel concentrations for the Legionnaire's patients and controls (*7*); there are no significant differences in the acute-phase and convalescent-phase serums. Therefore, if nickel is involved in the etiology of Legionnaires' disease [as was suspected earlier (*1*)], such involvement is not revealed in the serum samples.

The results for selenium are open to different interpretations, however. The rise in the concentration of selenium in convalescent-phase serums suggests its possible mobilization in host adaptation. It is therefore important to determine the source of the selenium (possibilities include compounds used in treatment and tissue-bound selenium, which may be activated and then detected in the serum). The results can also be interpreted as representing a low value for the acute-phase serums of the Legionnaires' patients. This may be due to a deficiency in the patient or to the effects of the Legionnaire bacterium.

These results may have special implications for the fields of inflammation and enzymology. Selenium has been shown to play a role in a variety of enzyme systems in mammals, including leukocyte acid phosphatases and glucuronidases and part of the catalytic moiety of glutathione peroxidase. Selenium has been used to potentiate the role of vitamin E (tocopherols) (*8*). The possibility of a role for selenium in the metabolism of the invading organism must also be considered, since selenium has enhanced bacterial growth in artificial media (*9*).

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5. The number of days that elapsed between acute-phase and convalescent-phase bleedings for patients 10 and 14 (82 and 49 days, respectively) was greater than the average (21 days) for the other patients. The bromine concentration in the convalescent-phase serum of patient 9 was the lowest concentration measured for all patients studied: 1.2 mg/liter compared to an average of 3.6 mg/liter in other patients.
6. For an eight-element comparison, we reduced the level of significance from $P < .00007$ to $P < .0006$. To account for the possibility (however unlikely) that the controls might show a distinct trend with a particular element, we further reduced the level of significance to $P < .001$.
7. The dates of the bleedings for patient 22 were unknown. The nickel concentration values for the two serum samples supplied to us were 0.04 (acute phase) and 0.03 (convalescent phase) mg/liter. The selenium concentrations for the two serum samples were the same and are therefore included in Fig. 1a.
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