LIGHTING, PERFORMANCE AND AGE VARIATION

R. A. Weale, Ph.D., Sc.D.

Dr. Weale: I want to talk as briefly as I may about some of the physical aspects of the eye, aspects which will make you wonder without any question whether it would not be wiser to consider units of luminance rather than illuminance, which have been so carefully distinguished by Dr. Halldane. When I want to concern myself with senile changes in the eye, then, as I say, I want to deal primarily, though not exclusively, with physical aspects, and that means with the ability of the eye to utilize such light energy as is capable of reaching it. It means a sine qua non. It means, as I hope I will show you, that no matter what we do, what levels we decide as being suitable for the eye in general, certain compensations will have to be made for senile changes which have been observed throughout the years in all of us. I am very conscious in this that one depends, to a large extent, on average data. This has severe drawbacks, especially insofar as establishment of casual relations is concerned. But we will come to that later.

By way of introduction, allow me to show you a section through the human eye. This is a schematic section through the eye which illustrates some of the salient points which distinguish an old eye from a young eye. I want to concentrate on two. This opening is the black of the eye, the pupil; and you will notice that it is smaller in the old half, which is shown down below, than in the young eye. It is a matter of common observation that the old pupil is smaller. This has been called senile miosis and is almost certainly due to the fact that the muscle which dilates the pupil atrophies at a faster rate than the muscle which constricts it. Consequently, the constrictor wins and the old pupil is smaller than the young one. None of this throws any light, any information on how easily the pupil constricts. In fact, contrary to conventional wisdom, it can be shown that the old pupil is more mobile than the young one.

The second point I want to stress relates to the lens. I do not want to bore you with details relating to the size, which I indicated here; but I do want to bore you with a very important point. Namely, the fact that the older lens is shown darker than the younger one. To cut a long story short, the older lens transmits less light than the young one. This is important and does not take into account that the older lens may, and in many cases does, scatter more light than the young one. Even if there were no change in the scattering properties of the lens, you would still find that, as though there were an increasing density in some gelatin filter, the old lens transmits less light. In addition to this quantitative change, this reduction in the light transmitted, there is a qualitative change in the sense that the increase in density and absorption predominates at short wavelengths. So, while we get a reduction in transmissivity of the lens as a whole, this is dominant at short wavelengths; in other words, in the blue and violet part of the spectrum. Well, so much for the setting of the scene.

The next slide (Figure 1) shows one slightly different from that shown by Mr. Crouch a little earlier and shows the distribution of visual acuity plotted on a decimal basis along the ordinate—that is the more conventional basis here, along the five meter basis—as a function of age. Umpteen distributions of this sort have been published. It really depends on how you select your patients. You can be pessimistic like Mr. Crouch and get a diminution which extends more or less throughout adult life. Or you can be more optimistic as the authors of these papers were and you can show that, if pathological conditions are absent, visual acuity is preserved up to about fifty and then starts decreasing at various rates. Now, the photometric points which I mentioned earlier—namely, the reduction in

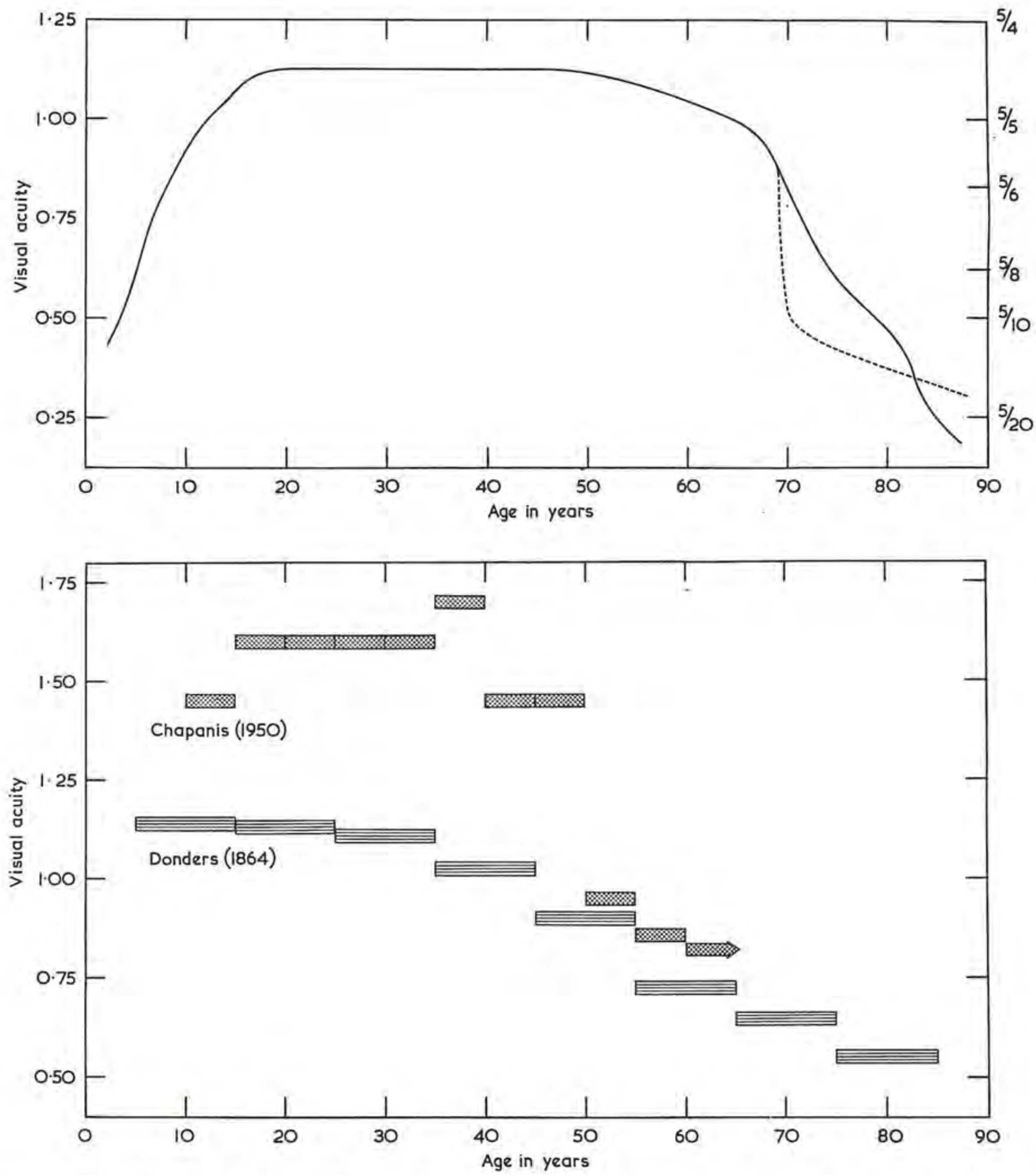

FIGURE 1.—Variations of visual acuity with age. Slataper's data (top) were collected probably under better conditions of illumination than were those due to Chapanis and Donders. (For details, see The Aging Eye, R. A. Weale (1963), H. K. Lewis, 179 Gower St., London W.C. 1. England.)

the diameter of the pupil as we get older and the reduction in the light transmitted by the lens—necessitate that, for an equal retinal performance, we have to make up the loss entailed in this case and in this simply by increasing the amount of light sent into the old eye, both insofar as pupillary area is concerned and—to add a qualitative factor—insofar as the spectral distribution of the light is concerned. If you want to look at things purely from the physical point of view, it is absolutely inevitable that these deficits ought to be made up. Now, before we get too alarmed, let me stress that, as far as I am concerned, I think this problem arises seriously only under threshold conditions. It is only where you have light available in such small amounts and, for example, in the proverbial corridor, or where light is available merely as a safety measure, for example, in photographic departments and so on, then, in my view, you have to compensate for this. Normally, the amount of light present is such that a correction due to this factor can probably be overlooked. And the new code, which you allowed me to mention this morning, used by the British Illuminating Engineering Society no longer distinguishes between the light requirements between older and younger workers, the idea being, as I mentioned earlier, that the amount of light needed to do the task shall be determined more or less in loco.

There are some data here which also have bearing on something Mr. Crouch mentioned and they relate to the differential, the incremental sensitivity of the eye as a function of age. The data here are plotted as sensitivity, not as threshold, and are expressed as ratios of the reciprocal of the incremental intensity to the field intensity along the ordinate and age along the abscissa. And the important point, I think, which these data show, is that, in the absence of glare, the decrease in sensitivity as a function of age is only about twenty-five percent over the range shown; whereas, note that it is very much more appreciable where a glare source is present. It seems to me, therefore, that one way of improving the photic environment of the older worker is by minimizing the number of glare sources of intensity present in their visual field.

There is a correlation between the data shown in the previous slide and this slide which shows the number of defects, ocular defects, as a function of age—or rather the probability of your suffering a defect. You see, by the time you come to seventy, the chances of you suffering from some trouble or other is ninety-two percent.

Mr. Nelson: Is that over and above these aging characteristics?

Dr. Weale: This is an unselected population.

Mr. Nelson: Yes, but I mean are you talking about defects other than the aging characteristics?

Dr. Weale: Yes, that is right. Since the other conditions, on the average, occur in everybody, they ought to be considered as physiological as distinguished from pathological. I think these cases are pathological, precisely because they do not happen to everybody.

Now, you can produce a good fit between this curve and the rather scrappy data which I showed in my previous slide. And people have suggested that, small wonder, more and more goes wrong with the eye. So your incremental sensitivity is reduced where your incremental threshold rises.

Mr. Crouch: I wanted to ask about a previous slide. Without glare, would this be a uniform brightness background or no background?

Dr. Weale: No, there must be a background, because there is a "B."

Mr. Crouch: I meant the surroundings.

Dr. Weale: I do not remember the exact experimental details, but it involved an incremental measurement.

Dr. Blackwell: If the field was large, you have stray light in the uniform field, which amounts to seven percent in the young and twenty-one in the old.

Dr. Weale: I will grant you this; but, from my recollection, this involved a separate glare source, whereas the data on the right did not.

Dr. Halldane: Was that surround or peripheral?

Dr. Weale: A peripheral source, yes.

Dr. Halldane: So it was basically entoptic scatter which influenced the contrast.

Dr. Weale: That is right, but it was focal scatter, a focal source, if you know what I mean.

Dr. Halldane: Okay.

Dr. Weale: It does not follow, necessarily, that there is a causal relation between these data and the results shown in the previous slide, because Voss showed, for example, that a result such as this has to receive careful statistical attention. He measured incremental thresholds and light scatter, I believe, by photography of the lens in a large number of observers. Although, statistically, there was an increase in the scatter and in the threshold as a function of age, when you compared the data obtained for each observer, the correlation that emerged was the sort of thing that you get if you correlated the color of hair, gray hair or baldness, with age. It did not follow that the two were related causally; and I think the same caveat has to apply to these results here.

One other result I think that ought to be mentioned, and that relates to the speed with which the eye reacts. The reaction time, as it is called, which is plotted here along the ordinate, and, again, age along the abscissa, with the number of observers shown with various symbols. You see then that there is a general rise in the higher age groups. The system appears to react more slowly. Well, these values are only fractions of a second, but it seems to me that they illustrate a point which it is worth making in connection, for example, with visual performance. It is not enough to consider the physical requirements which we have to meet as a result of the factors which I mentioned; namely, the pupil and the lens. There is what Dr. Blackwell might refer to as a field factor or what I would, perhaps, more likely refer to as a jog factor. Older people have to be jogged along, encouraged along, need a stronger stimulus, perhaps because it takes them longer to make up their minds. This is, of course, one of the hazards. This is one of the reasons you get accidents in the street. When older people cross the road, they do not know whether the car is far enough or not. They can see it; the visual part of the performance is accomplished. It is the decision which may need a reinforcement of the stimulus.

Mr. Nelson: What are you categorizing as older people, I am wondering?

Dr. Weale: Those that are not younger.

Mr. Nelson: I am trying to superimpose the normal retirement age across your graphs because I am interested in a working population, and I am just wondering whether the person who is casual in the street, incapacitated in the street, I usually think of as postretirement age. And I am just trying to determine whether we are talking about a total population or a total population subjected to working conditions. The different researchers seem to disagree rather widely on that one.

Dr. Weale: I, for one, am not going to be tied to any averages. I may use them, but I am not going to be tied to them, because I do not think they exist. But, of course, for the purposes of graphic presentation, they are a sine qua non. You see here we have a marked rise in the sixth decade. You may say that these data are not representative; but, of course, there is a tendency for a rise in these data as well. And you can put it operationally: you are old when you can not make a decision. And this may differ in various walks of life. Financially, it may happen pretty early in life. When it comes to crossing the road, it may happen a little later. When it comes to certain tasks which require decisions, it may very well happen in the fifties. I do not think we can really say that a person is old for a task if we confine our attention to his biological age. I think it must be accepted—I was turning gray at twenty—that there are various indices of age; and, whenever an index involves a difference in something that by common consent is referred to as young, in this particular respect, the person is old. That is why, if you will allow me, I would rather not generalize on this particular point.

When all is said and done, the results shown in this particular slide can, with confidence,—or such confidence as we can muster —be referred to only to the criterion which was used in order to arrive at them. Whether we are going to extrapolate from these data to other tasks altogether depends on the decision which we have to make.

These data refer to so-called dark adaptation curves which were also partly referred to by Mr. Crouch. They have a bearing on, again, threshold conditions. I know that this relates to a level much lower than we are contemplating, not to reduced but to very low conditions. It seems to me that we have to stress the physical aspects of a performance which depends on faulty conditions. I have only brought them along to show that, although it is commonly said that older people dark adapt much more slowly than young ones, what we mean by that is that, if a different level of illumination is, say, where I put it here, then, if people have been exposed to bright light, it will take them longer to reach this criterion than if they are young. The fact of the matter is that, to a large extent, you can account for these differences purely and simply in terms of reduced pupillary diameter and increased light absorption of the lens which older people suffer from. In other words, under conditions where there is no effect, no evidence of any pathology, the only effect which hinders vision in older people is attributable directly to the physical factors which I mentioned; namely the transmission of the lens and pupillary diameter.

Dr. Blackwell: Do you think more than a hundred to one can be explained that way?

Dr. Weale, Well, oddly enough, this was obtained with violet light, and the . . .

Dr. Halldane: Oh, no wonder, that is much lower (and slower).

Dr. Weale: We are talking about the rod section here, and you are dealing with people who are getting on toward ninety; and this is where you put two log units—

Dr. Blackwell: It is more than that.

Dr. Weale: Yes, but I said the major part. Alright? The major part of this difference is attributable to changes in the lens.

Dr. Riegel: Is what you said equivalent to saying that, if you regard that family of curves as a set of decaying exponentials, that the same time constant is associated with each one?

Dr. Weale: To a first approximation, I will produce evidence to support that in a moment.

Dr. Blackwell: Would you agree that, also, there is a degeneration of photoreceptors?

Dr. Weale: There is no evidence of that. I would like to know about it if there is.

Dr. Blackwell: I thought Ernst Wolf showed that.

Dr. Weale: I am not aware that he looked at photoreceptors. Did he?

Dr. Blackwell: Yes. He said there was a deterioration, a loss of photoreceptors.

Dr. Weale: But Wolf was a psychophysicist. I am not aware that he has done any histological studies.

Dr. Blackwell: He delved into this. I have the paper.

Dr. Weale: Well, I would be very interested and grateful to have this.

Here you have summarized data showing the reduction in the pupillary diameter, both in the dark-and light-adapted eye as a function of age. And, using these data, of course, one can predict how the light requirements of the eye are going to change as a function of age. And, similarly, here, you have measurements for the light transmission of the lens obtained by a variety of workers as a function of age. And here, again, these data can be used to predict the changes which you would expect in basic visual functions.

And here (Figure 2) you see one example of the fulfillment of this prediction. The threshold of vision is plotted along the ordinate here as a function of age and shown by these dark rectangles. The data which I have shown you in the last slide relating to the pupillary area are shown by the light oblongs. And you see that, to a first approximation—not wholly, I agree—this data can be explained in terms of senile miosis. Let me stress that the threshold here, contrary to the data which I showed you a little earlier, was measured with white light, in which case, the changes in the transmission of the lens would not play a primary role.

Here there are some data obtained by Gouras and Gunkel. These were obtained for normal observers, normal subjects as they would be in this case—no, they are observers—and they are shown with these white symbols; and then some people who had lost their lenses, with aphakic eyes, shown with circles. Now, the point which this illustrates is this. Those, of course, who have their lenses intact will experience the sort of yellowing, the increase in absorption that I have mentioned; and, as you get older, you need more and more light to compensate for the light absorption of the media. Consequently, the violet threshold will rise pretty appreciably—there you are, Dr. Blackwell, two log units—over the age spectrum. On the other hand, the aphakes—and look, there are quite some young ones amongst them—are virtually horizontal. You see, there is hardly any change at all in the violet part of the spectrum. Similarly, since the crystalline lens does not absorb much red light, you would expect a very much smaller change in the red part of the spectrum. In fact, this is parallel for both the normals and the aphakes insofar as one can judge from these results. White light, of course, is of practical importance and, as I mentioned a little earlier, for white light, as such, one has to make some allowance; but it is not very, very critical. Well, you see from this, my contention that the lens is responsible for visual performance, especially in the short wavelength part of the spectrum, is supported to a considerable extent.

Of course, one asks oneself whether the changes can account for other visual functions. By way of example, I have brought along Weeker's data, who compared the fusion frequency as a function of age when the pupil was both natural and dilated. And you see that there is prima facie evidence for the suggestion that the fusion frequency, which we know is reduced when the retinal illumination is reduced, is reduced, to a considerable extent, by senile miosis.

Well, all of this can be summarized very glibly but, perhaps qualitatively, in my last slide where we show the amount of light, not

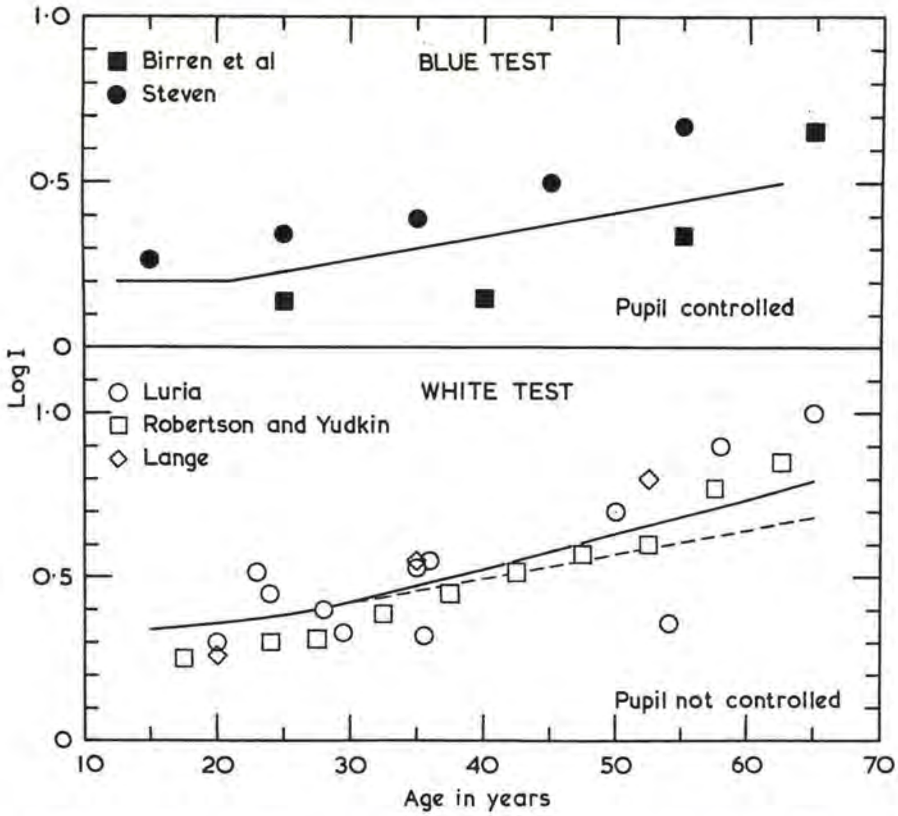

FIGURE 2.—Ordinate: Log visual threshold in arbitrary units. Abscissa: Observer's age. Top: Measurements obtained with a blue test-field and an artificial pupil. The line shows the rise in threshold expected on the basis of the senile rise in the density of the crystalline lens. Bottom: Measurements obtained with a white test-field and natural pupils. The dashed line shows the rise in threshold expected on the basis of senile miosis, and the full line the rise expected if, in addition, the senile rise in the density of the crystalline lens is also considered. (The Aging Eye, see reference figure 1.)

reaching the eye, but getting through to the retina as a function of age under photopic and scotopic conditions. The photopic conditions are shown with the light circles and the scotopic with the dark ones. Along here, we have considered only the lens and here we have considered the lens and the pupil. You see that, as a rough and ready measure, one can say that the eye at sixty needs about three times the amount of light which is needed by the eye at twenty, under threshold—that is under physical—conditions, without taking into account what might be needed at higher levels.

Well, it seems to me worth stressing this because I feel that, if we are going to get involved in reducing levels of illumination, we should, on the one hand, be reassured about what can and cannot be done for old people, and, on the other, remember that any appreciable vital amounts of light which old people may need are probably available because we are working well above threshold. Thank you.

DISCUSSION

Mr. Caplan: Dr. Weale, did you have any indication as to the reason for this change?—Was it strictly a presbyopic type of thing or was it as a result of exposure to something or what causes this change? Or is it the circulatory effect that was mentioned earlier?

Dr. Weale: I am sorry, which change are you referring to?

Mr. Caplan: The requirement of three times as much light to perform the same task for a sixty-year-old compared to a twenty-year-old. What causes that?

Dr. Weale: Well, I think this is due to two changes. On the one hand, you find that smooth muscle tends to atrophy; and, in the case of the pupil, its size is governed by the interaction, or we might say antagonism, of two types of muscle, the pupillary dilator and the pupillary sphincter. My view, for what it is worth, is that the pupillary dilator atrophies at a rate faster than the sphincter. It simply loses its function at a considerably faster rate. This means that the sphincter wins. In the young, they work against each other, but in the old there is no antagonism.

Mr. Caplan: Is this a normal function?

Dr Weale: This is normal, yes. I can not buttress this with great force because, believe it or not, as far as I know, there have been only three investigations since 1923 dealing with the senile histology of these muscle fibers. I think what I am saying is reasonable, just for once. As regards the lens, the lens is a very peculiar tissue, as is, of course, obvious. It has no blood supply. It has great difficulty in getting rid of its waste products. Moreover, as I indicated in my slide but I did not want to draw your attention to other problems, it goes on growing throughout life. It keeps on laying down layer after layer. There seems to be no halt to that. Well, this is also a problem; and the upshot of all this is that the inner layers are subjected to greater and greater pressure, to an environment which you can not really call biological any more. You get waste products; they do not know what to do with it, so the layers change color.

Mr. Caplan: It is not an external environment.

Dr. Weale: It is not the external, it is the internal environment, or rather the lack of an internal environment, which I think is primarily responsible in the case of the lens.

NIOSH

The Occupational Safety and Health Effects Associated with Reduced Levels of Illumination

Proceedings of Symposium

Cincinnati, Ohio

July 11 - 12, 1974

NIOSH
NIOSH
NIOSH

THE OCCUPATIONAL SAFETY AND HEALTH EFFECTS ASSOCIATED WITH REDUCED LEVELS OF ILLUMINATION

Proceedings of Symposium

Cincinnati, Ohio

July 11 - 12, 1974

Allan P. Heins, Ph.D.

Symposium Chairman

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health
Division of Laboratories and Criteria Development

Cincinnati, Ohio 45202

MARCH 1975

These proceedings were compiled and arranged by Allan P. Heins[1] with the assistance of William E. Murray of the Physical Agents Branch, Division of Laboratories and Criteria Development, NIOSH.

[1] Western Area Occupational Health Laboratory, National Institute for Occupational Safety and Health.

HEW Publication No. (NIOSH) 75—142