INTRODUCTORY REMARKS BY HONORED GUEST:
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A year or so ago my friend JERRY PEARLMAN asked me whether I would be willing to say a few words to set the stage for this Symposium. Not being able to decline any request of Jerry's, I accepted with pleasure. But you will realize my somewhat anxious surprise when a circular letter appeared a couple of months ago, stating that I had accepted to be the honored guest—a distinction of which I knew nothing. Of course, I am very grateful to Jerry, with whom I spent a few happy years in Iowa, that he wanted to single me out in this fashion, but I am also keenly aware that I deserve this honor infinitely less than many another person present here.

Actually, this is a very opportune occasion to take a look backward as well as forward. This is the 10th symposium organized under the auspices of the ISCERG. And while not yet of age, our ISCERG has reached the lovely age of puberty, being 14 years old. As I look around, I think with sadness of the absence of one of our Founder Fathers, ADOLPHE FRANCESCHETTI, who is no longer with us, but I am happy that all others have been spared through many vicissitudes.

While our ISCERG was created 14 years ago, clinical ERG is older. Believe it or not, it is almost 30 years old—27 years to be exact. For it is in 1945 that appeared the monograph of KARPE, the Father of clinical ERG, which was at the beginning of it all. And we must go even farther back, to 1941 when RIGGS designed his contact lens electrode which made routine human, and therefore clinical ERG possible and which in one modification or another is still one of our basic tools.

Allow me at this point to indulge in a little personal reminiscing. I was recuperating from a severe illness in 1948 when, by what accident I do not know, the first issue of Vol. I of the J. of EEG & Clinical Neurophysiology fell into my hands, containing an article by MONNIER on human ERG. My recovery was speeded, for I was inspired. I had found the method which would answer the question which had occupied me for 10 years, whether the seat of the lesion in functional amblyopia was retinal, as some thought or in the CNS. I lived in Boston at the time and DR. DENNY-BROWN kindly let me have a corner in the laboratory of his Department of Neurology at the Boston City Hospital. Being quite naive in these matters, I had my difficulties in setting up the equipment, and the like. I might mention here in passing that at that time I went to Europe and ran into my old friend, Prof. HANS GOLDMANN, for the first time after the war. I told him about my problems with ERG and he said to me, "Why don’t you go and learn from HAROLD HENKES in Rotterdam how to do ERGs?"

Unfortunately, at that time this was not possible for me.

However, the initial problems were overcome, especially after I moved to Iowa and was able to establish a fine electro-physiologic laboratory. And then a number of things immediately became clear. The state of the art at that time was such that ERG could not give the answer to the amblyopia problem because of the inability to produce focal ERGs, due to the factor of stray light and for

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other technical reasons. But I was hooked! I became increasingly fascinated with ERG, and it became evident to me that to make full use of the ERG technique for clinical studies more sophisticated procedures were required than had previously been employed.

It became obvious that the electrical retinal response was first of all determined by two major factors: the stimulation provided and the state of adaptation of the eye. While I was and remained an ophthalmologist, and neither wanted nor could in any way compete with neurophysiologists, I had to investigate for my own satisfaction the importance of these and other parameters. Their significance was such that I felt quite dissatisfied with much of the literature on clinical ERG and with such statements that “the ERG of the patient was normal”, when no indications were given under what conditions the ERG was obtained.

What I am saying here may sound very trite after 20 years, but at the time it was far from trite. Today, the functional testing in clinical ERG, as developed over the years, has become routine.

Of course, I cannot go into any of the details of the history of clinical ERG, but want to point out that it has two aspects, a practical one and a theoretical academic one. From the practical standpoint ERG has confirmatory rather than primary diagnostic value. It is in the nature of this method that it has been of greatest usefulness in vascular anomalies and toxic states of the retina, in degenerative diseases particularly of the retinal periphery and carrier states, and in infants. On the other hand, from an academic standpoint, ERG studies in certain abnormal conditions, for example in achromatopsia and other forms of color deficiencies were most significant in contributing to an understanding of the ERG itself.

Such, at least, was the situation until a few years ago. Meanwhile refined techniques have greatly added to the possibilities offered by ERG. I refer especially to the various methods which now make it possible, partly thanks to computer techniques, to produce more or less focal ERGs. There is also the promising field of the clinical study of the oscillatory potentials.

Furthermore, entirely new techniques have come up which singly, or in combination with ERG, would seem to add greatly to the usefulness of the electrophysiologic methods. These techniques are the EOG and the VER. Many among us are making use of these methods, as evidenced also by some of the papers on the program of this symposium. I wonder whether the time has not come to change the name of our Society from ISCERG to International Society for Clinical Electro-Physiology of the Visual System. I know that such a suggestion, made some years ago, was rejected as premature. Possibly the time has come now to consider seriously such a change.

In spite of all the difficulties, I have never given up on the amblyopia problem. The more refined electrophysiologic methods allow one now, I believe, to answer the question with which I started. Methods avoiding stray light, including the use of patterned stimuli as applied in the laboratory of Dr. Lawwill in Louisville, Kentucky, in which I have had the privilege of working during the last few months, have given definite evidence that the electrical response of the retina of the amblyopic eye does not differ from that of the fixating eye.

2