THE BIOPHYSICAL BASIS AND CLINICAL APPLICATIONS OF RHEOENCEPHALOGRAPHY

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Dr. John H. Seipel, Chief, Neurology Laboratory, Georgetown Clinical Research Institute, was awarded the S. Weir Mitchell Award on April 29, 1966, by the American Academy of Neurology during their annual meeting in Philadelphia, Pennsylvania.

Dr. Silas Weir Mitchell, 1829–1914, was a pioneer physician and neurologist who lived and practiced in Philadelphia. His interests ranged widely; in addition to his many medical papers and books he became equally well known as a poet and novelist publishing over 180 novels and books of poetry. Several of the medical syndromes, signs, and treatments he devised still are in use and carry his name.

The S. Weir Mitchell Award, consisting of a bronze medallion, a honorarium, and the opportunity of presenting the research at the Academy meeting, is given annually to encourage research by young physicians and scientists in the general field of neurology.

Dr. Seipel was honored for his Georgetown Clinical Research Institute studies in rheoencephalography, a simple, rapid, innocuous method of studying the brain circulation which had previously received little favorable interest in the United States. In his paper, "The Biophysical Basis and Clinical Applications of Rheoencephalography," Dr. Seipel has demonstrated the high level of accuracy inherent in the method in diagnosing various forms of brain disease, developed a reliable procedure for its use, demonstrated its value in screening normal subjects for early, asymptomatic disease and demonstrated various hitherto unknown syndromes.

This research was undertaken to develop rheoencephalography into a simple office procedure, comparable to electrocardiography, which could be used for certification and screening purposes to detect brain problems likely to cause sudden incapacity, such as stroke, prior to their occurrance and in sufficient time to begin adequate treatment.

Dr. Seipel's receipt of the award in Philadelphia was particularly appropriate since he received part of his medical training at Pennsylvania Hospital; Dr. Mitchell was a member of that hospital's Board of Managers and probably also practiced there.

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SUMMARY

1. The electrochemical and biophysical basis of rheoencephalography (REG) and its clinical applications are discussed.
2. The effect of flow on blood resistivity is reviewed and is shown to be probably negligible in REG.
3. Theoretical analysis suggests REG is a quantitative method which reliably reproduces changes in the cranial blood volume.
4. The presence of significant and valuable extracranial circulatory components in the REG tracing is confirmed; these components usefully represent the external carotid arteries.
5. A simple, standardized, reproducible tracing procedure and various methods of analysis are described in detail. Sources of error are discussed and methods of compensation and avoidance are presented.
6. Individual extracranial and intracranial arterial contributions to the tracing are demonstrated and measured for the first time. These measurements afford a quantitative evaluation of the patient's cranial circulation.
7. Analyses of 136 normal and pathological tracings are presented; a 95% diagnostic accuracy is demonstrated clinically.
8. Difficulties in REG diagnosis using previously reported criteria are presented and elucidated.
9. Incidences and patterns of arterial involvement and collateral compensation are presented and discussed.
10. The potential value of REG screening of normal populations for cerebrovascular anomalies and early disease is demonstrated.
11. Several previously unreported REG syndromes are described.

THE BIOPHYSICAL BASIS AND CLINICAL APPLICATIONS OF RHEOENCEPHALOGRAPHY*

JOHN H. SEIPEL, PH.D., M.D.

ntroduction

Many reviews of cranial impedance plethysmo-raphy, or rheoencephalography (REG), a nethod of studying the cranial circulation, have een published.[1-11] While it is agreed that heoencephalograms are at least partially plethys-nograms, the above references and their bibliog-aphies indicate considerable controversy con-erning the exact relationship of the rheoen-ephalogram to the intracranial circulation. his controversy has engendered disagreement ver the value of REG in both clinical diagnosis nd physiological research.

Much of this controversy arises from factors nherent in the use of novel or unfamiliar meth-ds. Such factors include comparisons based on lifferent methodologies and concepts of tracing nterpretation, unfamiliarity with vascular hy-raulics in general and intracranial hydraulics n particular, unfamiliarity with the scope, ap-lications, and inherent limitations of the method, ack of experience with its use, and predetermined avorable or unfavorable but enthusiastic preju-lice.

The author's attention was first drawn to heoencephalography by his continuing search or methods which could be used to study living ystems without perturbation or injury.[12,13] It vas immediately apparent that the method was otentially quantitative, that the cerebral circu-ation could be monitored, and that individual arotid arterial contributions might be deter-nined by tracing during brief manual carotid cclusions. It also appeared likely that the nethod had the great potential for disease de-ection and diagnosis claimed by Jenkner.[1] These intuitive considerations have sustained the author's studies throughout the above controversy.

This paper is intended as a summary of more than five years research into the biophysics of REG and experience with its clinical use. Some theoretical considerations will first be presented followed by a more practical demonstration of the possible value of REG in clinical diagnosis.

Theory

I. Electrochemical Basis of Impedance Plethysmography.

A detailed discussion of the many factors influencing conduction of an electric current by solutions of electrolytes is beyond the scope of this paper.[14,15] There are, however, a number of concepts which should be presented to assist the understanding of biological impedances. The discussion will be deliberately superficial and is intended only to indicate present reasoning and conclusions. The occasionally difficult underlying mathematics have been omitted and will be presented in future publications.

An inherent property of matter is its exhibition of an impedance (Z) to the passage of an electrical current; such impedances are composed of a real component, resistance (R) and an "imaginary" component, reactance (X) where:

$$(1) \qquad Z=(R^2+X^2)^{1/2}$$

Reactance can be a variable combination of capacitance (C) and inductance (L):

$$(2) \qquad X=\omega L-\frac{1}{\omega C}$$

where ω is the angular frequency of the applied current. Inductance is a negligible component of the reactance of biological materials, if present at all, and will not be considered further.

For a given electrolyte solution, for example, aqueous sodium chloride, containing two im-

*A contribution from the Neurology Laboratory, Georgetown linical Research Institute, Office of Aviation Medicine, Federal viation Administration and the Department of Neurology, eorgetown University Hospital, Washington, D.C.

mersed electrodes, the resistance and capacitance measured between the electrodes are described by simple equations:

$$R=\frac{\rho l}{a} \tag{3}$$

$$C=\varepsilon\varepsilon_0\frac{a}{l} \tag{4}$$

where ρ is the resistivity of the solution, ε is the relative dielectric constant of the solution, ε_0 is the dielectric constant of free space, "l" is the distance between the electrodes, and "a" is their area.* The above equations apply to any solution or material so long as all factors except l and a are held constant and the applied measuring current is sufficiently low; changes in l and a affect both the resistance and capacitance and, therefore, the impedance.

If one solves for the impedance of a biological material in terms of the geometrical factors l and a, one obtains (5) and (6):

$$X=-\frac{1}{\omega\varepsilon\varepsilon_0 a} \tag{5}$$

$$Z=\left(\rho^2+\frac{1}{\omega^2\varepsilon^2\varepsilon^2_0}\right)^{\frac{1}{2}}\frac{l}{a}=k\frac{l}{a} \tag{6}$$

Equation (6) indicates that geometrical alterations in a material which alter its resistance will also alter its capacitance. Further, such reactance and resistance changes affect the impedance change in a similar manner. For simplicity the following discussions will be restricted to the resistive component; analogous reasoning is applicable to the reactance and impedance. It should be noted that the resistivity and dielectric constant of a biological material may vary markedly with the frequency of the measuring current.[16] The frequency therefore must be kept constant for intercomparability of measurements.

Reference to a standard handbook[17] indicates the resistivity of a solution varies inversely as the electrolyte concentration in a nonlinear manner (Figure 1) and falls as the number of con-

*These equations hold only in the special case where l is small compared to the electrode dimensions and edge effects can be neglected.

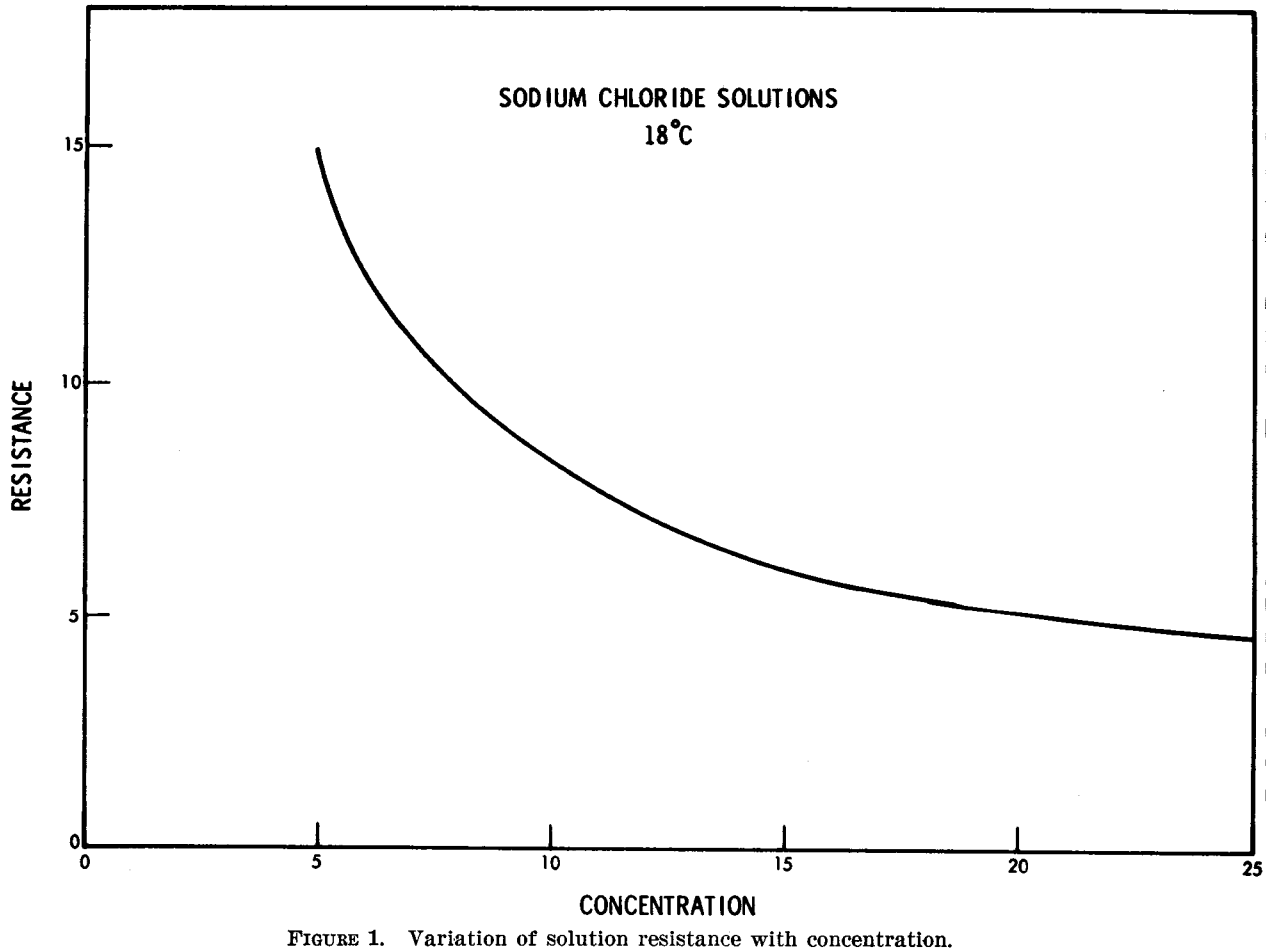

FIGURE 1. Variation of solution resistance with concentration.

ducting species per unit volume increases. The resistivity also varies with temperature[17] (Figure 2) and falls as the ionic mobilities rise with temperature. In effect, the resistance of a biological material at constant temperature is a quantitative measure of the total number of conducting species in the current field.[14]

Within a living tissue the cellular and interstitial constituents probably change very little over moderate time periods; the major changes in conducting species would be due to blood content. The total resistance of a tissue thus consists of an almost constant resistance and a variable resistance due to its blood content; both resistances usually have unknown magnitudes. Since more conducting species appear between the electrodes with systole, the total tissue reistance will *decrease* in an amount quantitatively related to the net *increase* in blood volume.

The above discussion indicates there should be a quantitative relation between changes in tissue resistance and blood volume. A second mechanism is also considered to contribute to the observed tissue resistance changes. The resistivity of flowing blood is less than that of static blood and decreases with increases in flow rate.[18–25] Therefore, the observed systolic tissue resistance decrease is partly due to the lowered resistivity of the rapidly inflowing blood; the diastolic resistance increase is also partly dependent on the resistivity increase of the slowing blood. Such tissue resistance changes are undecipherable mixtures of changes due to volume and flow.[5,26] This mechanism has been cited sufficiently frequently against the use of impedance methods as a reliable means of studying cranial hemodynamics that detailed consideration is warranted.

Present understanding of electrode mechanisms, particularly those involving electrokinetic phe-

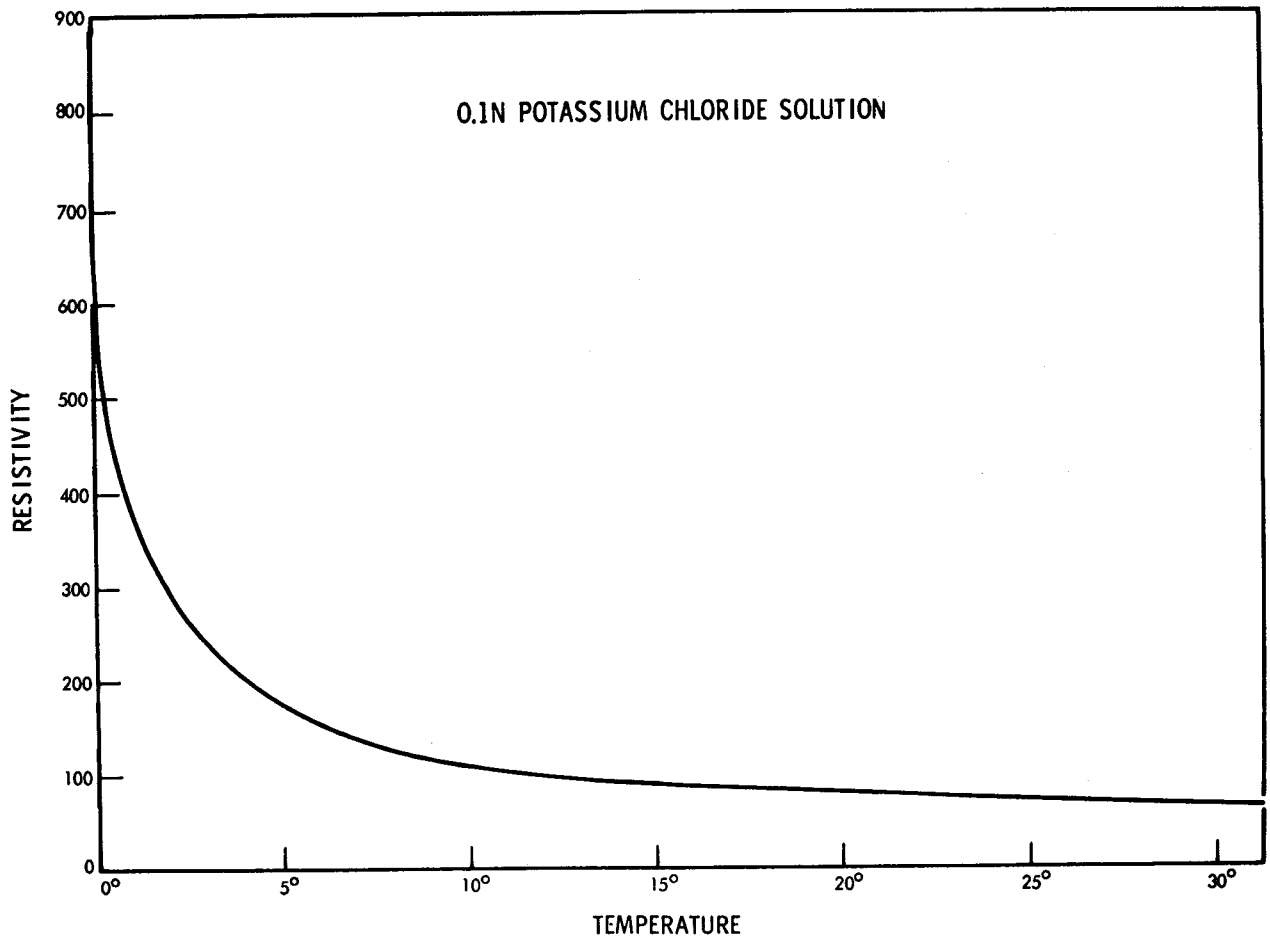

FIGURE 2. Variation of solution resistivity with temperature.

nomena, is based on the double layer model[14,27-31] of the region about an electrode (Figure 3). According to this theory the distribution of positive and negative charges about an electrode is nonuniform. The electrode attracts a firmly adherent layer of oppositely charged ions, possibly covered by a second layer of similarly charged ions. Surrounding this thin inner layer is a second diffuse layer in which the electrode electrostatic field attracts oppositely charged ions and repels similarly charged ions until equilibrium charge and ionic concentration gradients have been attained. This latter layer extends a considerable distance until it becomes indistinguishable from the solution. These layers exist about any electrode immersed in a solution, whether or not externally charged, since the electron cloud within the metal itself can attract ions from the solution. Movement of the solution past the electrode disturbs the equilibrium concentration gradient and its electrode potential producing streaming potentials. Movement of the electrode with respect to the solution produces the familiar "movement artifacts" frequently seen in biological studies.

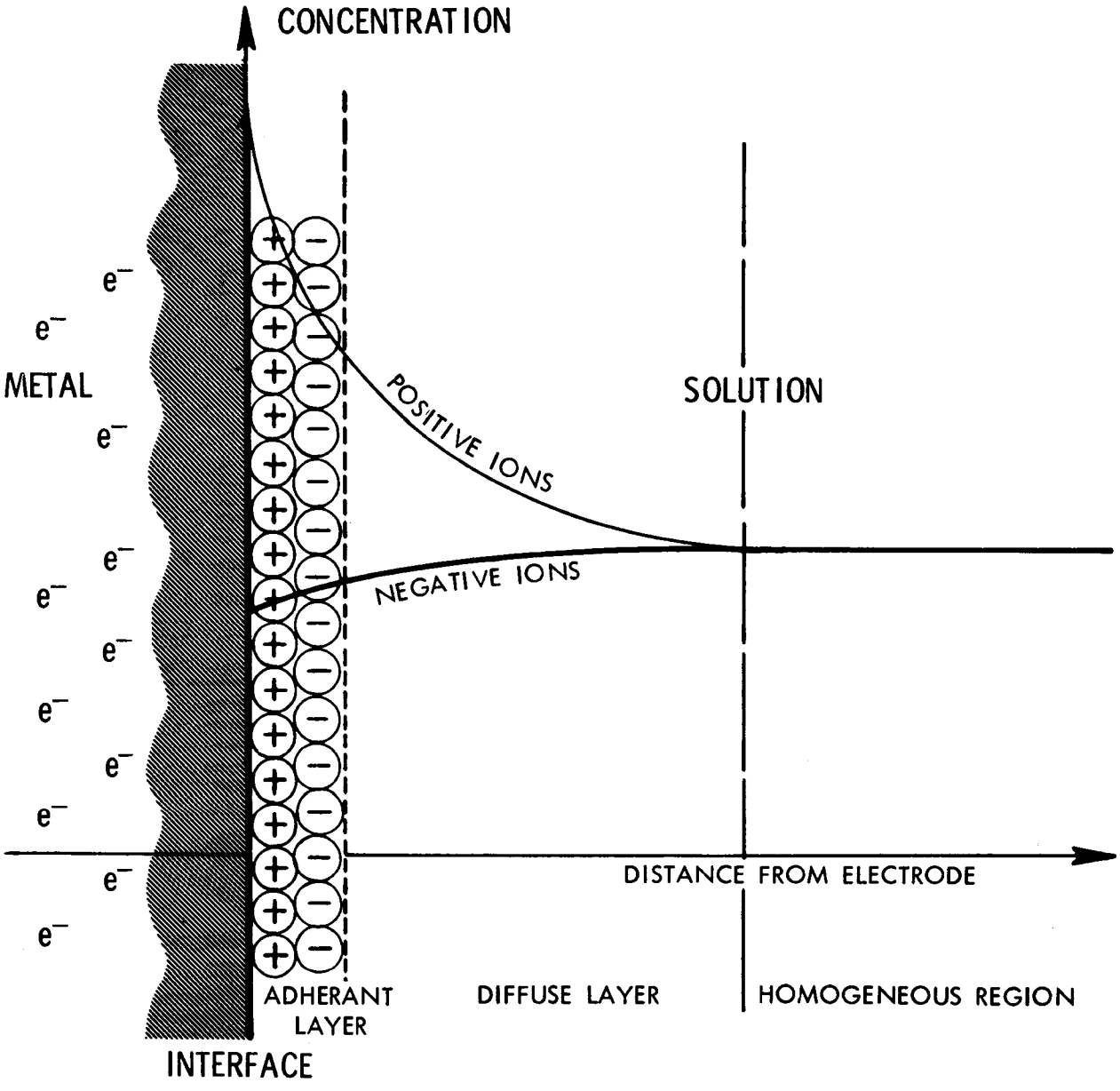

FIGURE 3. The double layer model of the region about an electrode.

In the static case, the layers of altered concentration should have a somewhat higher electrical resistance than would be expected for the same volume of unaltered solution and should increase the steady-state resistance measured between the electrodes. Since solution movement constantly brings more ions into this region, different levels of constant flow should establish different steady-state electrode layers which would become thinner with more rapid flow. Therefore, the steady-state resistance falls as flow increases. Further, the effect should be non-linear, lessening with higher flow levels until a minimum resistance is reached which is independent of further flow increase. Since the measured resistance determines the resistivity of the solution, the latter should also apparently decrease with flow. The reported blood studies[18-25] were carefully reviewed for evidence of the above electrode effect; the investigators used conductivity cells which were directly or indirectly subject to the above electrode effect in most of the measurements.

However, Sigman et al[18] describe experiments which unambiguously demonstrate a true decrease in blood specific resistance with flow. First, they modified the conductivity cell designs used for their initial studies by placing the electrodes in sidearms where there was no disturbance due to the moving blood and found identical resistivity changes with flow. Second, they studied a series of solutions and suspensions in the unmodified apparatus and observed a measurable flow effect only with blood. Finally, the magnitude of this effect was directly related to the concentration of erythrocytes in the moving blood. Somewhat similar results are found by others.[19,25] Different authors postulate various mechanisms to explain the effect but all agree it is associated with the presence of erythrocytes in the flowing blood.

Therefore, while it is uncertain to what degree electrode effects contribute to the observed decrease in blood resistivity with flow, they are probably negligible in comparison with the total flow effect. Thus, the contribution of this flow effect to tissue resistance changes must be evaluated in reference to rheoencephalography.

Liebman et al[24] measure a maximum decrease in blood impedance of 8% at a steady 20 cm./sec. flow in a 3.5 mm I.D. tube; the change with pulsatile flow is considerably smaller (1.4% estimated from graph). Velick and Gorin[19] find 8.2% at an unspecified flow rate which gave complete erythrocyte orientation. Coulter and Pappenheimer[20] measure maximum effects (estimated from graphs) of 18% at 63% hematocrit, 9% at 53% hematocrit, 11% at 44% hematocrit, and 9% at 40% hematocrit. At 12 cm./sec. Moskalenko and Naumenko[22] measure a 2.7% change. Sigman et al[18] measure a maximum effect of 5.3% at comparable steady flows but observe average changes of 2.2% (3.4%–1.0%) at 60% hematocrit, 1.3% (3.1%–0.0%) at 40% hematocrit and 0.17% (+1.0% to −1.0%) at 20% hematocrit. Of the above, only the studies by Moskalenko and Naumenko[22] and Liebman et al[24] were performed on human blood.

The contribution of the flow effect to the rheoencephalogram may be difficult to estimate accurately from the above range of values. However, the maximum blood resistivity change that might occur at high flows and normal human hematocrits would be about 10%. At steady flows equivalent to maximum levels likely to be encountered in the bulk of tissue circulation, the effect should be smaller, probably about 2–4%. If the further pulsatile flow reduction observed by Liebman et al[24] is valid, the final flow resistivity change may be less than 0.4-0.8%, a negligible error. Thus, tissue resistance (impedance) variations associated with blood circulation almost certainly arise solely from blood volume variations and contain little or no "flow contamination." Tracings of these variations are plethysmograms and are analogous to volumetrically obtained tracings.

The total magnitude of the resistance is not plotted in impedance plethysmography; instead, variations about a particular constant resistance value are recorded. Such tracings are analogs of the total resistance from which a constant resistance has been subtracted to accentuate variations, exactly as volume plethysmographic tracings reproduce variations about a constant volume. Therefore, impedance plethysmograms should be capable of yielding information entirely equivalent to that obtained by any other plethysmographic method *provided* due allowance is made for the various inherent methodologic differences. The great advantages of the impedance method lie in its safety, its simplicity, and its essentially instantaneous accurate response

capabilities and its applicability to tissue volumes which would be difficult or impossible to study by other methods.

It should be emphasized that any plethysmogram is a volumetric measurement which depends on the time integral of the difference between two unknown dynamic quantities, arterial inflow and venous outflow. Plethysmography by *any* method cannot give either arterial or venous flows or total tissue blood volumes without modification. Conversion of an impedance plethysmogram to its corresponding volumetric changes is particularly difficult and cannot be successful without detailed knowledge of current fields throughout the monitored volume.

It is apparent that any disease which sufficiently alters the normal circulation of blood *anywhere* in its course *to*, *through*, or *from* the monitored tissue volume or which sufficiently alters the normal conductivity characteristics of the tissue can alter the tracing. Thus, the clinical use of impedance plethysmography can be established on a purely empirical basis; such use need not be postponed awaiting elucidation of the biophysical basis of the method.

II Biophysical Basis of Rheoencephalography.

The simplest head analog is a homogeneous, isotropic, conducting sphere. Measurement of surface voltages between two current-carrying point electrodes diametrically placed on the surface of such a sphere gives a non-linear voltage curve (Figure 4).

This surface voltage variation is described by equation (7) :[32]

$$(7) \qquad \Phi = \frac{J}{2\pi\sigma}\left[\frac{1}{R_1} - \frac{1}{R_2} + \psi\right]$$

where:

$$\psi = \frac{1}{2a}\left[\sinh^{-1}\frac{a+r\cos\theta}{r\sin\theta} - \sinh^{-1}\frac{a-r\cos\theta}{r\sin\theta}\right]$$

$$R_{1,2} = (a+r\mp 2a\cos\theta)^{1/2}$$

The non-linearity arises from the concentration of the current field in the volume near the electrodes and its dispersion as the volume increases with distance. In the illustrated case, the non-linear "near-field" region extends to 5 cm. from the electrodes; the voltage curve is approximately linear in the intervening region. This concept of an electrode "near-field" volume is introduced for discussion purposes, recognizing that the properties of a conducting volume are continuous and that such division is artificial.

A better head approximation is a multishelled sphere having homogeneous isotropic layers corresponding to the scalp, skull, and cerebrospinal fluid surrounding a central spherical volume corresponding to the brain. Each region is assigned "lumped" passive electrical properties and average dimensions corresponding to the appropriate tissue. Few human cranial tissue resistivities have been measured under living conditions at any frequency. The values for blood, 150 ohm cm.,[33] and cerebrospinal fluid, 60 ohm cm.,[34,35] are perhaps the most reliable. Intact living cat brain measures 225 ohm cm.[36] Values for the remaining tissues may be estimated from measurements on somewhat analogous tissues.[16] Skin and subcutaneous tissue are assumed comparable to muscle at 700 ohm cm. (30 kHz). Bone, having a dense matrix which should constrain ion movements despite its blood and electrolyte content, is assigned 3000 ohm cm.; the true value may be much smaller.[37,38] The model is illustrated in Figure 5. In this model the "intracranial" region has a much lower resistance than the remaining layers; the cerebrospinal fluid and the brain with its high blood content[39] offer a marked shunt path beneath the meninges and bone. The cranial near-field volume for such a model must be highly distorted. It probably has a relatively constant radius in the subcutaneous tissue, a considerably shortened radius within the bony layer, a longer radius in the cerebrospinal fluid, and a somewhat shorter radius in the brain for diametrically placed electrodes. The deepest penetration would coincide with the interelectrode axis. Since the intracranial contents offer paths of lesser resistance, the majority of the current should traverse the intracranial cavity; the highest current density most probably lies along the interelectrode axis (Figure 6).

Since the major portion of the total resistance lies in these near-field volumes which contain large fractions of subcutaneous tissue and bone, the total resistance is heavily weighted by these tissues. Therefore, extracranial blood volume variations will give rise to larger resistance changes than equivalent intracranial blood volume variations. Reasoning from the effects of

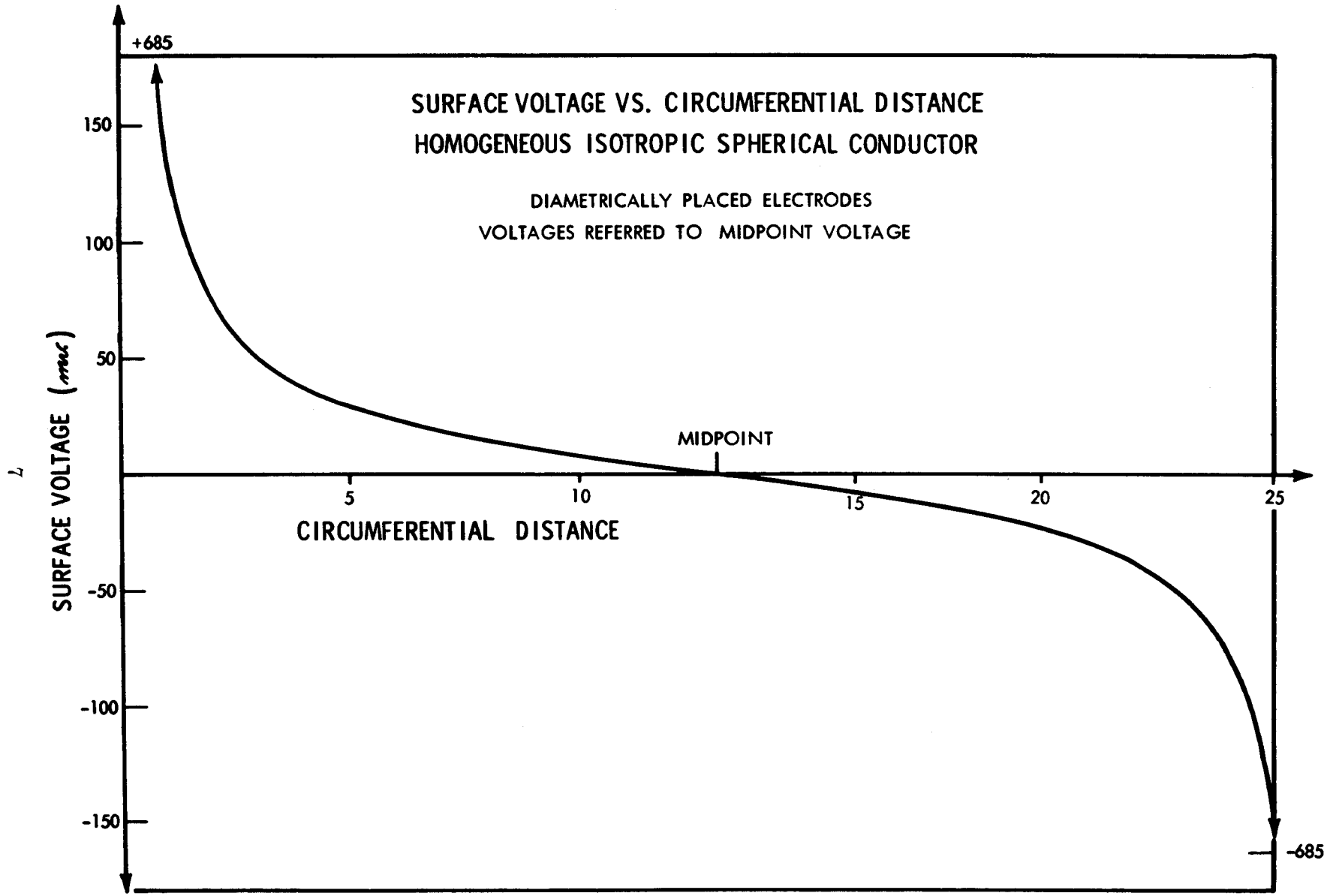

FIGURE 4. Surface voltage versus circumferential distance for a sphere.

electrode size, Lifshitz[5] reaches a similar conclusion.

Qualitative predictions about the influence of interelectrode distance can be made from the model. Initially, surface electrodes will establish separate, relatively "independent" near-field volumes as the interelectrode distance shortens from diametrical placement; such volumes will have extracranial/intracranial tissue ratios approximately equivalent to those at diametrical placement (assuming reasonably constant anatomy). This situation will remain relatively unchanged with shorter interelectrode distances until the near-field volumes begin to overlap. Here the electrodes register less "independent," partially coalesced, near-field volumes whose subcutaneous tissue content is increased. As coalescence becomes pronounced with shorter distance, the subcutaneous weighting will rise rapidly. At sufficiently close electrode placements, practically all of the current may shunt extracranially. Thus, at long interelectrode distances the majority of the current necessarily traverses small volumes of subcutaneous tissue and bone, despite their high resistance, to reach a large low-resistance volume of intracranial contents; this path offers lower overall resistance than paths traversing only the subcutaneous layer. At short interelectrode distances the inverse is true; the direct subcutaneous paths offer less resistance than the more oblique paths through large volumes of bone and a small volume of intracranial contents. Similar reasoning can be applied to the total resistance. Initially, the latter should remain relatively constant with decreasing interelectrode distance and then should fall, at first gradually but later rapidly, as the interelectrode distance shortens beyond a particular value. Such curves of total resistance versus interelectrode distance have been presented by Grey Walter.[38]

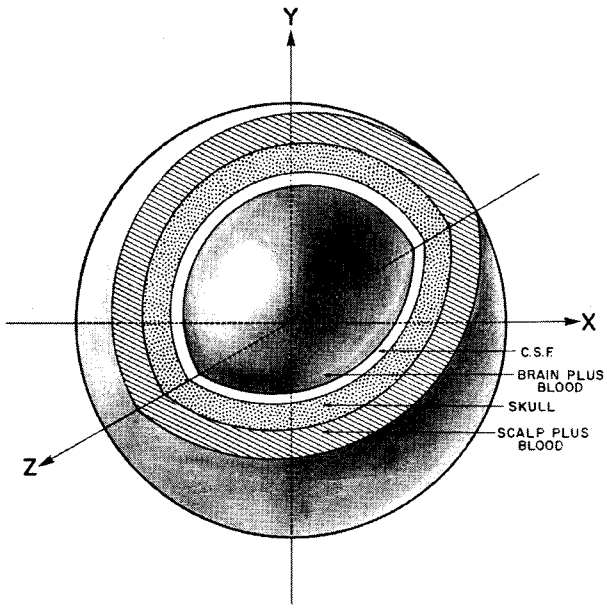

FIGURE 5. Multishelled spherical model of the head.

The preceding discussion indicates an appreciable volume of subcutaneous tissue will always be monitored despite wide electrode separation. Thus, the total impedance plethysmogram can be expected to arise in part from the extracranial circulation. Further, since the current density is greatest in the tissues closest to the electrodes, blood volume variations in the near-field region will have a greater influence on the tracing than *equivalent* variations occurring at a distance, for example, intracranially. The magnitude of this extracranial "artifact" depends on the magnitude of the extracranial blood volume variations occurring primarily in the near-field volumes. The contribution from blood variations occurring more remotely from the electrodes in the fraction of the current shunting wholly extracranially is probably slight. The above conclusions have been anticipated in part by Lifshitz.[5]

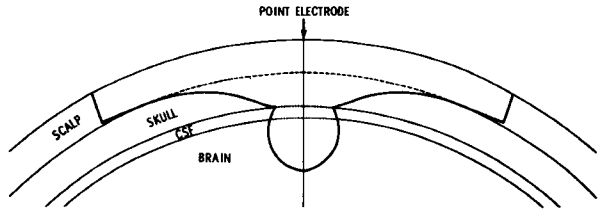

FIGURE 6. Geometrically approximated electrode "near-field" volume for the multishelled cranial model.

On the other hand, since most of the current eventually traverses the intracranial region but is only partially concentrated in the intracranial portion of the near-field volume, almost the reverse situation would exist for intracranial blood volume variations. Both tracing components may be appreciable; their ratio will depend both on the current distribution throughout the head and on the ratio of the extracranial to intracranial circulatory changes. In some subjects, the extracranial "artifact" may comprise a large fraction of the total tracing despite widely separated electrodes and the known favorable ratio of scalp circulation to cerebral circulation.

With close electrode placement, significant near-field volume overlap can be expected to occur; the tracing will then contain a larger fraction of extracranial "artifact". At sufficiently close placement it is reasonable to expect that almost no intracranial changes are likely to be detected. Nyboer[2] illustrates frontal electrode placement without comment. Simonson[40] presents evidence for a high level of extracranial "contamination" in tracings obtained from normal subjects using various electrode positions which include close (¾") frontal placement. The pulse waveforms he obtains from electrode pairs positioned on the forehead and occiput, however, show definite contour dissimilarities which are probably not significant considering the different interelectrode distances and the dissimilarity between his right and left forehead-occiput tracings. Simonson[41] and Dontas and Simonson[42] describe studies using forehead electrode placement and consider that such tracings represent essentially extracranial circulation. It is difficult to accept recent uncritical claims that techniques using relatively close electrode placement register localized or "regional" cerebral circulation and monitor blood volume variations in the circulatory fields of individual cerebral arteries without extreme extracranial contamination.[43-46]

The current field extends throughout the entire head despite the location of the major portion of the total resistance in the near-field volumes of tissue about the electrodes. Variations in blood volume anywhere in the head will affect the total resistance but will be detectable only to the degree they modify a sufficiently large fraction of the total current. This suggests that impedance plethysmography may detect changes in blood volume anywhere in the head although weighted to detect changes in the near-field volumes (see also Lifshitz[5]).

With electrodes placed at opposite ends of a diameter of the head, for example, forehead and occiput, the current field is symmetrically distributed throughout the entire intracranial and extracranial volume. If the intracranial and extracranial structures and circulation are perfectly symmetrical about the midline, half of the current will flow through the right and half through the left side of the head (Figure 7). The resultant plethysmogram will be a composite of identical contributions from each side where each side contributes half of the total amplitude.

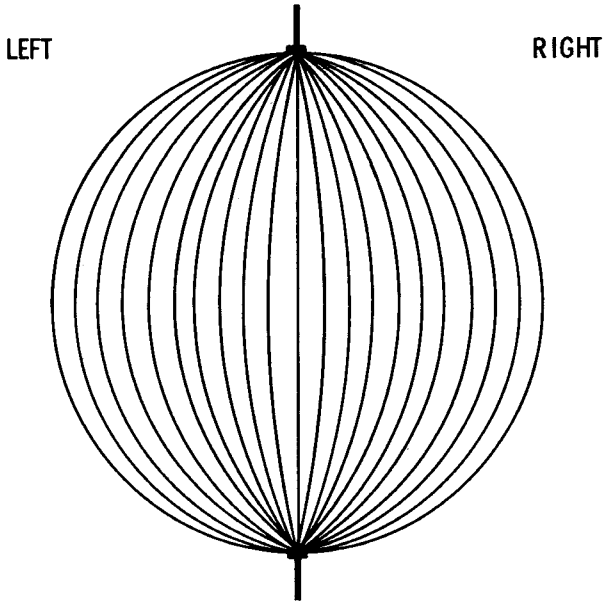

FIGURE 7. Estimated current distribution with anteroposterior electrode placement.

With placement of the posterior electrode closer to the anterior electrode on the same ideally symmetrical head, the current field still permeates the entire volume, but the previous

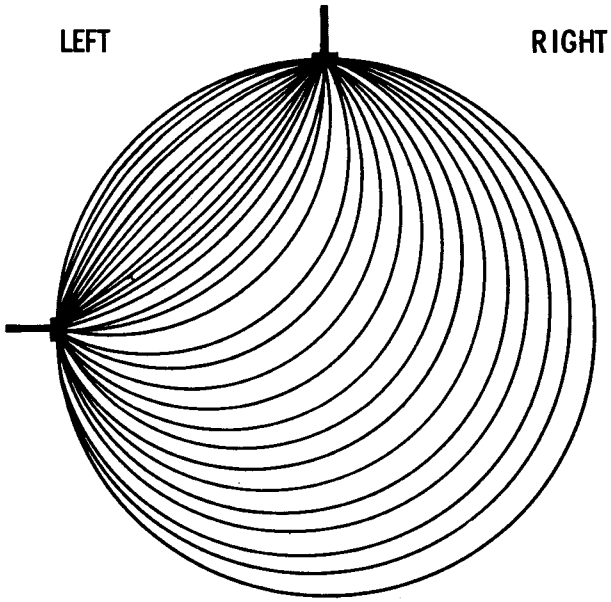

FIGURE 8. Estimated current distribution with anterolateral electrode placement.

symmetrical distribution is lost (Figure 8). The current field now concentrates ipselaterally al-

though an appreciable fraction continues to permeate the contralateral volume; the fraction of the total current flowing ipselaterally is greater than half of the total current, while the contralateral fraction has fallen in the same amount. The resulting plethysmogram, while still composite, no longer contains equal contributions from each side. The intracranial tracing component is weighted in favor of the ipselateral side in approximately the ratio of the intracranial current fractions. The extracranial component is much more heavily weighted in favor of the ipselateral side since the extracranial current path is much shorter along the arc directly between the electrodes than around the remaining portion of the circumference. As the interelectrode distance lessens, the contralateral extracranial contribution to the plethysmogram vanishes relatively quickly and more rapidly than the contralateral intracranial portion of the plethysmogram.

In summary, the cranial impedance plethysmogram, or rheoencephalogram, is a complex quantitative analog of changes in cranial blood volume and is composed of component "sub-plethysmograms" from the ipselateral and contralateral extracranial and intracranial regions. The weighting of these components in large part depends on electrode size and placement, normal and pathological anatomic and circulatory asymmetries, and normal and pathological passive electrical properties of the various component biological materials. At present, most of the above factors can only be estimated; conversion of the various component impedance plethysmograms to blood volume values and extrapolation of these values to values representative of the total blood volume variations in each tissue region are qualitative at best.

III. Individual Arterial Contributions to the Rheoencephalogram.

A. Basic Assumptions: The previous discussions indicate that the following assumptions may be reasonably applied to rheoencephalography:

1. Cranial resistance changes are quantitatively associated with changes in blood volume.

2. With bilaterally symmetrical placement, each electrode pair monitors similar extracranial and intracranial tissue volumes and their contained blood.

3. Each electrode pair monitors a major fraction of its ipselateral tissue volume. Monitoring of the contralateral tissue volume is negligible.

4. Appreciable fractions of the tracing may arise from both the extracranial and intracranial circulations.

5. Additive contributions from each artery to the total blood volume change are reproduced as additive changes in the tracings.

6. Extracranial changes are intercomparable and intracranial changes are intercomparable; but extracranial changes are not intercomparable with intracranial changes unless their relative weighting is known.

The following additional assumptions will be made:

1. Each electrode pair monitors its current field independently; i.e., no interaction exists between channels.

2. Barring catastrophic circulatory occurrences, cranial hydrodynamics remain constant throughout the tracing interval and are not perturbed significantly by the tracing procedure.

3. Brief, deliberate, complete arterial occlusion removes only the appropriate components from the tracing. Collateral compensation occurs slowly, if at all, during the compression interval unless the latter is unduly prolonged.

B. Anatomic Models for Rheoencephalography:

1. *Basic Circulatory Model:* A relatively small number of arteries may be expected to contribute most of the circulation monitored by rheoencephalography in the normal subject. For the scalp, skull, and meninges, these are the angular, superficial temporal, posterior auricular, and meningeal branches of the external carotid arteries and the supraorbital branches of the internal carotid arteries. For the brain, these are the internal carotid and vertebral arteries. The meningeal, internal carotid, and vertebral arteries are not easily accessible for manual occlusion while the angular, posterior auricular, and occipital arteries are small and probably add negligible contributions to the rheoencephalogram. The following model was chosen as a first-order approximation of the cranial circulation for use in analysis of the rheoencephalogram (Figure 9).

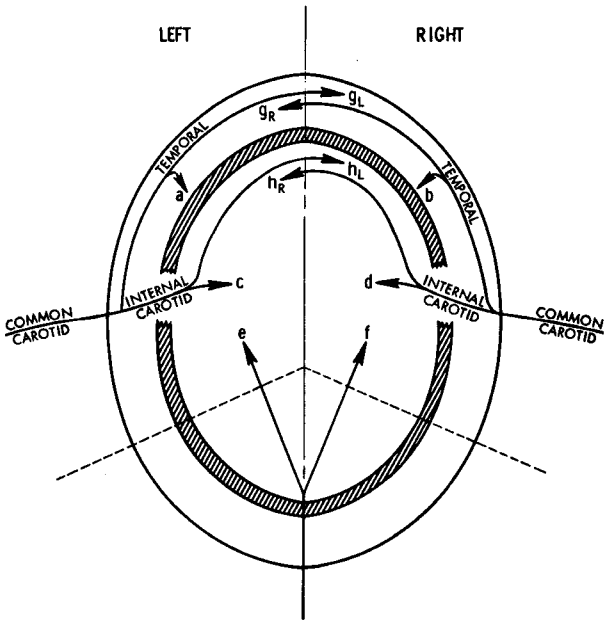

FIGURE 9. Model for the analysis of the rheoencephalogram.

Notation:

1. Representative tracing amplitudes:
   - $R_1 = -\triangle R_1$ = Average peak-to-peak pulse amplitude on the right without arterial manipulation (reference or "baseline" amplitude)
   - $R_2 = -\triangle R_2$ = Average peak-to-peak pulse amplitude on the right during occlusion of the right superficial temporal artery
   - R_3 = ibid, left superficial temporal artery
   - R_4 = ibid, both superficial temporal arteries
   - R_5 = ibid, right common carotid artery
   - R_6 = ibid, left common carotid artery
   - R_7 = ibid, both common carotid arteries
   - L_1, L_2, L_3, L_4, L_5, L_6, L_7 = corresponding $-\triangle R$ values on the left

2. Representative vascular component amplitudes:
   - a = Ipselateral tracing component contributed by the left superficial temporal artery
   - b = ibid, right superficial temporal artery
   - c = ibid, left internal carotid artery
   - d = ibid, right internal carotid artery
   - e = ibid, left vertebral-basilar system
   - f = ibid, right vertebral-basilar system
   - g_L = contralateral tracing component contributed by the left superficial temporal artery
   - g_R = ibid, right superficial temporal artery
   - h_L = ibid, left internal carotid artery
   - h_R = ibid, right internal carotid artery

NECS = net transmidline extracranial component (collateral shunt from the opposite superficial temporal artery)

NICS = net transmidline intracranial component (collateral shunt from the opposite internal carotid artery)

The following equations can be written for each tracing period:

1. Baseline:

(8) $R_1 = b + g_L + d + f + h_L$

(9) $L_1 = a + g_R + c + e + h_R$

2. Right superficial temporal arterial compression:

(10) $R_2 = g_L + d + f + h_L$

(11) $L_2 = a + c + e + h_R$

3. Left superficial temporal arterial compression:

(12) $R_3 = b + d + f + h_L$

(13) $L_3 = g_R + c + e + h_R$

4. Bilateral superficial temporal arterial compression:

(14) $R_4 = d + f + h_L$

(15) $L_4 = c + e + h_R$

5. Right common carotid arterial compression:

(16) $R_5 = g_L + f + h$

(17) $L_5 = a + c + e$

6. Left common carotid arterial compression:

(18) $R_6 = b + d + f$

(19) $L_6 = g_R + e + h$

7. Bilateral common carotid arterial compression:

(20) $R_7 = f$

(21) $L_7 = e$

If the initial assumptions are valid within the error of the recording procedures, the above equations are simultaneous and can be solved for the values of the various arterial tracing components.

A check on the simultaneity of the values is possible if bilateral compressions have been performed. Values for the bilateral compression amplitudes can be calculated:

(22) $R_{4C} = R_2 + R_3 - R_1$

(23) $L_{4C} = L_2 + L_3 - L_1$

(24) $R_{7C} = R_5 + R_6 - R_1$

(25) $L_{7C} = L_5 + L_6 - L_1$

The differences between the observed and calculated values are a measure of the simultaneity of the equations.

Solution of the simultaneous equations gives the following formulae for the various vascular components:

(26) $a=L_1-L_3$
(27) $b=R_1-R_2$
(28) $c=L_3-L_6$
(29) $d=R_2-R_5$
(30) $e=L_5+L_6-L_1=L_7$
(31) $f=R_5+R_6-R_1=R_7$
(32) $g_R=L_2-L_5$
(33) $g_L=R_1-R_3$
(34) $h_R=L_2-L_5$
(35) $h_L=R_3-R_6$

The contributions arising from the various arteries can be calculated from the above components:

(36) RT=Right temporal artery$=b+g_R$
(37) LT=Left temporal artery$=a+g_L$
(38) NECS=Net extracranial shunt$=|g_R-g_L|$
and is directed:
right to left if $g_R>g_L$ or
left to right if $g_L>g_R$
(39) RIC=Right internal carotid artery
$=d+h_R$
(40) LIC=Left internal carotid artery$=c+h_L$
(41) NICS=Net intracranial shunt$=|h_R-h_L|$
and is directed:
right to left if $h_R>h_L$ or
left to right if $h_L>h_R$
(42) RCC=Right common carotid artery
$=b+g_R+d+h_R$
(43) LCC=Left common carotid artery
$=a+g_L+c+h_L$
(44) RP=Right cerebral perfusion$=d+f+h_L$
(45) LP=Left cerebral perfusion$=c+e+h_R$
(46) PA_R=Right vertebral-basilar shunt$=f$
(47) PA_L=Left vertebral-basilar shunt$=e$

The above vertebral-basilar components are worthy of brief discussion. The scalp is essentially completely supplied by the external carotid arteries while the majority of the structures within the anterior compartment of the skull is supplied by the internal carotid arteries. If REG monitors only these structures, as is highly probable, the conclusion is inescapable that any remainder seen during bilateral common carotid compression arises from the vertebral-basilar system. These values, therefore, represent to some degree the blood volume supplied to the anterior compartment by the vertebral-basilar system.

This simple model and its treatment is the basis for our method of rheoencephalographic analysis. This approach to plethysmography is probably general and can be used to identify and determine the circulatory contribution of any artery if measurements can be made during appropriate occlusions.

The above model is presented because of its simplicity of calculation and its circulatory relevance. There is an alternate, somewhat similar model which will be briefly presented.

2. *Model Assuming Partial Contralateral Monitoring:* In this model, assumption No. 3, page 10, is modified by assuming that the current field from an electrode pair may be sufficiently diffused throughout the entire cranial volume that the pair monitors an appreciable fraction of the contralateral circulatory events, as Berta et al indicate.[47]

The previous notation will be used plus:

α=extracranial cross-registry fraction
β=intracranial cross-registry fraction

Assuming that the intracranial and extracranial collateral shunts (g_R and h_R) arise on the right and appear on the left, equations can be written such as the following for the reference amplitude:

$$R_1=b+\alpha(a+g_R)+d+f+\beta(c+e+h_R)$$
$$L_1=a+g_R+\alpha b+c+e+h_R+\beta(d+f)$$

The remaining equations for the set will not be illustrated.

The solutions are somewhat more complex than in the previous model:

$$\alpha=\frac{R_1-R_3}{L_1-L_3} \qquad \beta=\frac{R_3-R_6}{L_3-L_6}$$

$$a=L_1-L_3 \qquad b=\frac{R_1-R_2-\alpha\ (L_1-L_2)}{1-\alpha^2}$$

$$c=L_3-L_6 \qquad d=\frac{R_2-R_5-\beta\ (L_2-L_5)}{1-\beta^2}$$

$$e=\frac{L_5+L_6-L_1-\beta\ (R_5+R_6-R_1)}{1-\beta^2}$$

$$f=\frac{R_5+R_6-R_1-\beta\ (L_5+L_6-L_1)}{1-\beta^2}$$

$$g_R=\frac{L_1-L_2-\alpha\ (R_1-R_2)}{1-\alpha^2}$$

$$h_R=\frac{L_2-L_5-\beta\ (R_2-R_5)}{1-\beta^2}$$

The formulae for the arterial contributions are more complex and will not be illustrated.

In the above treatment, both shunts are assumed to arise on the right and appear on the left. Anatomically, these shunts can arise on either side or not exist at all; thus, there are nine separate cases which require individual treatment. Further, two sets of criteria must be derived to indicate which of the nine cases must be used in the analysis of a particular rheoencephalogram.

Since the current field of a pair of electrodes permeates the entire head, it is reasonable to expect that contralateral circulatory events contribute to the resistance changes. The problem lies not in the existence of these contributions but in whether they must be considered at the level of accuracy required of tracing analysis. The above method was thoroughly tested for several years in the routine analysis of clinical tracings. The extracranial cross-registry fraction was found to average 0.07% while the average ipselateral change was 0.014 ohm. The resulting 0.001 ohm tracing contribution falls within the inaccuracy of the method and can be ignored safely. Analysis of the intracranial cross-registry fraction is more difficult. In 58 patients with intracranial vascular disease, it averaged 0.38 with a range of 0.00–0.93; for 29 normal subjects it averaged 0.65 with a range of 0.22–1.00. Of the total subjects, 22 (25%) fell below 0.35. Four values (5%) were zero. One normal subject is included in the above 25%; other findings suggest cerebrovascular disease in this man.

The derivation of the cross-registry fraction assumes all contralateral tracing changes arise from cross-registration of ipselateral changes and denies any possibility of blood distribution around a patent circle of Willis. Unilateral carotid compression can be expected to cause bilateral circulatory reductions if the circle is patent; the contralateral reduction should be smaller than the ipselateral reduction. Since this contralateral effect would be added to the true cross-registry effect, the calculated cross-registry fraction would be larger by a possibly significant amount. The high cross-registry fraction seen in the normal subject group supports this conclusion as does the lower cross-registry fraction observed in the group of patients with cerebrovascular disease. Six (10%) of this latter group gave fractions of 0.12 or less (average 0.02); their average ipselateral change was 0.038 ohm (0.009 ohm–0.068 ohm). Of particular note is one patient who gave the large change of 0.068 ohm with *zero* contralateral change. It would appear the intracranial cross-registry fraction is small and possibly negligible within the limits of the present methodology. This question is under further investigation.

3. *Model Without Temporal Arterial Compressions*: The first model can be simplified by omitting the compressions of the scalp arteries. While the following treatment is no longer used by the author, much of the clinical material to be presented was obtained before the extracranial contribution to the tracing was recognized. As the data deficiencies cannot be repaired, this method must be presented.

Notation:

The previous notation will be used plus:

m = ipselateral tracing component contributed by the left common carotid artery

n = ibid, right common carotid artery

S_L = contralateral tracing component contributed by the left common carotid artery

S_R = ibid, right common carotid artery

Equations similar to those in the first model can be written:

(48) $R_1 = n + S_L + f$

(49) $L_1 = m + S_R + e$

(50) $R_5 = S_L + f$

(51) $L_5 = m + e$

(52) $R_6 = n + f$

(53) $L_6 = S_R + e$

(54) $R_7 = f$

(55) $L_7 = e$

The solutions are:

(56) RCC = Right common carotid $= R_1 - R_5 + L_1 - L_5$

(57) LCC = Left common carotid $= R_1 - R_6 + L_1 - L_6$

(58) NS = Net shunt $= |S_R - S_L|$ and is directed: right to left if $S_R > S_L$ or left to right if $S_L > S_R$

(59) PA_R = Right vertebral-basilar shunt = f

(60) PA_L = Left vertebral-basilar shunt = e

IV. Conclusions:

It should be emphasized that the above general approach to rheoencephalographic analysis has been derived from theory alone and is based on a number of explicitly stated or implied as-

sumptions. Several of these assumptions, while reasonable in the ideal normal case, perhaps are oversimplifications, are overly rigid and may not hold within experimental error in certain patients or during particular tracing periods. The analytic approach can be expected to give equivocable or even misleading results when applied to such tracings; further, various dissimilar diseases can be expected to cause either similar alterations in those portions of the cranial circulation monitored by REG or to be indistinguishable in the analysis. Thus, if the above approach is to have any practical clinical usefulness, the validity of its predictions must be clinically substantiated and its limits of reliability and requirements for further modification must be clinically determined.

The above analytic method suggests a further, more general method which could greatly extend the value of REG. The derivation of the above equations uses the average peak amplitude of a series of pulse waveforms to represent various circulatory situations. Such an amplitude is, at best, an approximation to its average pulse waveform; a better representation can be obtained by averaging the pulses on a suitable computer as Lifshitz has done for the reference tracing[5,48]. The average waveforms can then be computer-processed further according to the above equations to give waveforms representative of each arterial component. This approach is under investigation.

Certain predictions can be made at this point. After REG has been thoroughly tested and established, it should be possible to:

1. Detect, lateralize, and diagnose carotid occlusive disease, localize the major site above or below the carotid bifurcation, estimate the degree of impairment, and estimate the extent of collateral compensation;
2. Detect, lateralize, and diagnose other forms of intracranial disease and estimate their degree of development; and
3. Use rheoencephalography reliably in any of the myriad situations in which a knowledge of the cranial physiological events which it monitors would be valuable.

Hopefully, since the above methodology is time consuming, clinical experience may develop objective criteria for the diagnosis of disease reliably by eye alone similar to present practice in electroencephalography.

Clinical Studies

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The controversy regarding the clinical value of REG is illustrated by the marked dichotomy between the reports of Jenkner[1], in whose hands it has been extraordinarily successful, and those of Perez-Borja and Meyer[10] and McHenry[11] who found it not only of little diagnostic value but also frequently misleading. Thus, the first question any clinical study of REG must answer is the basic one that all potential additions to the clinical armamentarium must answer—can the method detect and diagnose disease with sufficient reliability to warrant its clinical use?

The rheoencephalographic studies in the author's laboratory have followed two major interlocking plans simultaneously: first, to collect a sufficiently large series of clinical cases to determine if the method has clinical value and, if so, to determine its range of applicability; and second, to elucidate as much of the biophysical basis of the method as possible. The clinical studies will be discussed; the biophysical studies will be presented in later papers.

I. Methodology:

A. Standardization: Rheoencephalography has not escaped the methodologic diversity which frequently arises following the introduction of a new research or clinical tool. It was recognized at the inception of this study that rheoencephalography might be markedly influenced by apparently minor variations in methodology and that much of the diversity of opinion probably arose from such sources. Therefore, a rigidly standardized method was necessary for intercomparability within the projected clinical series and for comparison with results obtained by other investigators. A standard method might also permit the isolation and study of individual factors in the procedure.

Preliminary analysis suggested particular sensitivity to variations in a number of factors for which standardization would be important. These were:

A. Patient factors:
   1. Body position
   2. Head position

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3. Skin preparation
4. State of relaxation

B. Electrode factors:
1. Anatomic placement
2. Lateral symmetry
3. Fixation
4. Area
5. Contact medium
6. Material

C. Instrumentation factors:
1. Lead resistance
2. Calibration
3. Rheoencephalograph characteristics
4. Recorder characteristics

The above factors will be discussed during the description of our tracing procedure.

B. Pretracing Procedure: Since the senior author was aware of Jenkner's extensive studies and had obtained a "Doppel-Rheograph" from Dr. Schufried, he arbitrarily selected as standard a tracing procedure similar to that reported by Jenkner.[1] While experience has suggested slight modifications, our present procedure is almost identical with that originally employed.

1. *Patient Position*

Constant patient position relative to gravity is absolutely necessary for tracing intercomparability.[5] Our patients lie comfortably recumbent in slight semi-Fowler's position with a head elevation of 7° relative to horizontal. The shoulders, neck, and occiput are supported and partially immobilized by a thin pillow; the head is slightly extended; and the neck is exposed to permit access to the carotid arteries. The wrists, with attached EKG electrodes, lie naturally, slightly away from the sides (Figure 10). Shoes are usually removed.

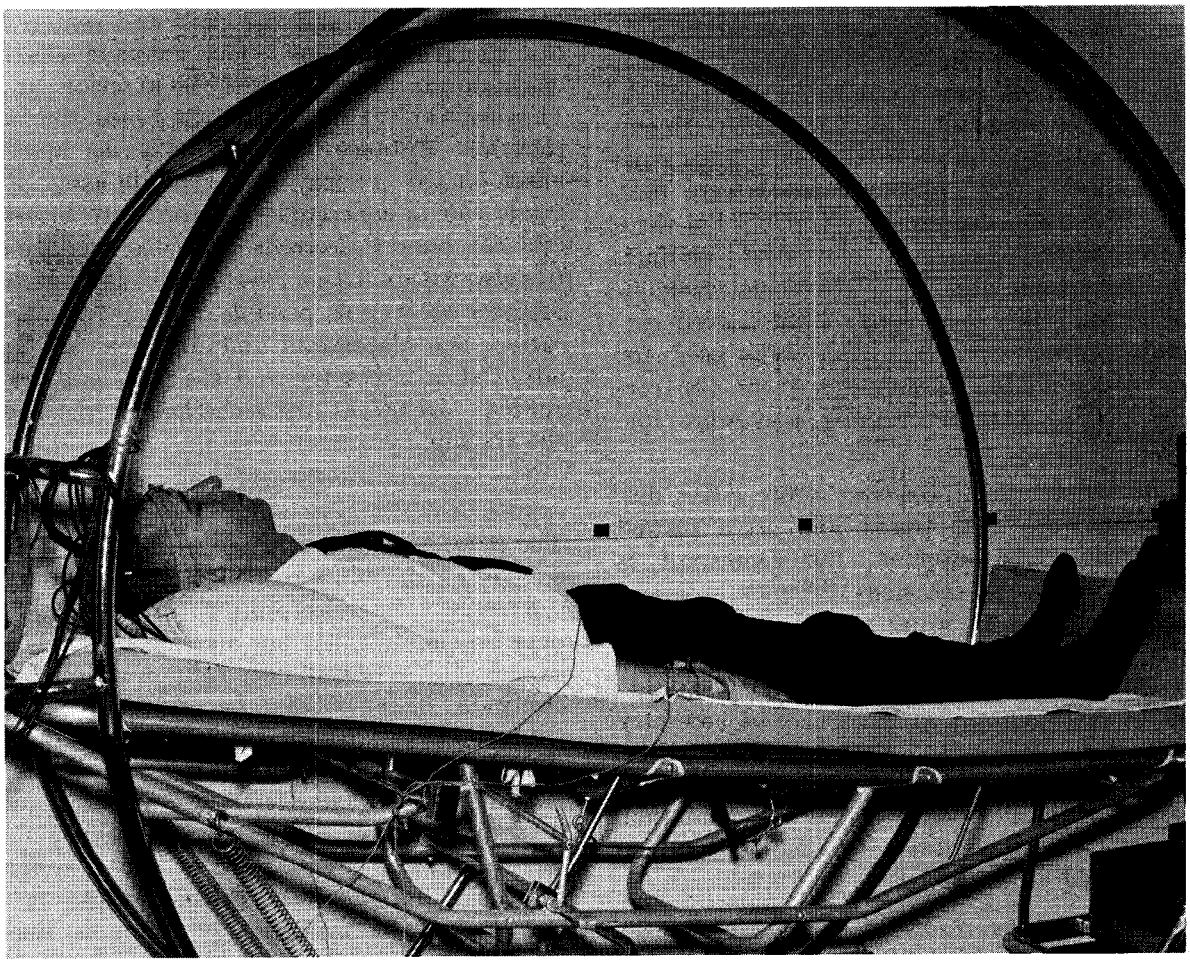

FIGURE 10. Patient position for rheoencephalography.

The procedure is described to the patient who is encouraged to relax, breathe evenly, remain still and quiet, and sleep if possible. Between recording periods, the patient is permitted to readjust position slightly, speak, sneeze, swallow, and otherwise perform those small actions employed by the human to relieve boredom or tension. He is informed such actions cause artifact during recording periods and prolong his stay—a most effective way of insuring cooperation! Pads or eye shields are used if eye movement and blink artifact are excessive. Conversation is kept to a minimum during the tracing period. Room lighting is subdued and glare is avoided. Adequate walkway space is preserved about the bed to permit easy access from all sides without patient distraction or movement.

2. *Electrode Preparation and Placement.*

a. *Electrodes:* The 7.5 cm.^{2} aluminum block electrodes supplied with the Schufried instrument were originally employed without modification. While satisfactory when relatively new, internal corrosion from the salts in the electrode paste rapidly rendered them unreliable. Heavy (0.020″) platinum sheets of the same dimensions with welded 0.020″ platinum leads were next tried. These afforded excellent tracings but their rigidity caused excessive movement artifact. New Rheograph aluminum electrodes vacuum-impregnated with clear epoxy resin proved reasonably corrosion-resistant and were used for most of the study. Eventually, these also corroded despite careful cleaning and were discarded.

Standard 9.4 mm.^{2} German silver EKG disc electrodes were next tested and have proved satisfactory. As supplied, the centerpost is somewhat large and occasions difficulty in lateral electrode placement using standard perforated rubber electrode straps. These posts were removed and replaced by solder nubbins. Enlargement of the binding-post holes may be necessary to accept the Rheograph electrode leads. Electrode faces are cleaned with fine emery cloth as necessary to remove surface film and oxidation. The slightly roughened metal surface so obtained is considered advantageous.

b. *Skin preparation:* Electrode placement sites are carefully cleaned with acetone or alcohol before placing and pasting the electrodes. Female patients are requested to shampoo their hair before reporting.

c. *Electrode placement:* Electrodes were originally placed as suggested by Jenkner[1] and illustrated in our previous paper.[49] They are secured firmly in place by a not uncomfortably snug, perforated rubber electrode strap encircling the head. The anterior electrodes are placed as close together as the band perforations permit without touching and are centered on the sagittal midline of the forehead. The lower electrode edges are slightly above the frontal eminences. Further consideration of possible current flow paths in the Jenkner placement suggested that the electrodes should be positioned both to minimize paths through bone and extracranial tissue and to maximize paths through the intracranial volume supplied by the carotids. The frontal "indifferent" electrodes were already positioned slightly above the cranial floor. The lateral "active" electrodes were shifted to a similar level between the ears and the skull and centered on the bi-auricular line. The anterior electrode edges do not extend beyond the anterior ear line (Figure 11). This change shortened the interelectrode distance slightly with no apparent effect on the tracing. After preliminary placement, the interelectrode distances are measured and the lateral electrode positions are adjusted to agree within 0.5 cm., keeping the interelectrode distance as long as possible. After paste application, the placements and distances are again adjusted as necessary and the distances are recorded. Distances are rechecked at the end of the tracing.

The importance of careful electrode placement cannot be overemphasized, as Jenkner[50] indicates. The previous theoretical discussion indicates the electrode pairs must be placed in close anatomic symmetry. Interelectrode distances should not only be closely equal but also should be as long as possible to avoid near-field overlap. Interelectrode distance can be expected to vary slightly from patient to patient. However, standard anatomic electrode placement and bilaterally equal interelectrode distances insure that the fraction of the cranial volume monitored by each lateral pair remains reasonably constant throughout the patient series. A possible source of methodologic error is thereby minimized and intercomparability is strengthened.

3. *Electrode Contact Material.*

a. *Criteria for choice:* We chose to use commercial electrode pastes rather than the saline-soaked sponges used by Jenkner.[1] Such pastes offer the advantages of considerably higher conductivity, more easily reproducible thickness of the layer between electrode and skin, better continuity over the entire electrode area, greater dimensional stability after application, and slower drying rate.

Measurements (1963) using Radiometer conductivity equipment* on a variety of such pastes at room temperature disclosed that one preparation† had a conductivity half that of saturated saline solution and 12 times that of the next highest paste. This paste was therefore chosen as standard. Of the other preparations, the creams in particular were considerably less conductive and often contained substances which absorbed firmly on the platinized electrode surfaces rendering them hydrophobic. These substances could not be removed by repeated washing with water and a variety of organic solvents.

b. *Method of Application:* Electrode paste is applied in sufficient quantity to wet thoroughly the skin and hair beneath the retracted electrodes and is rubbed in well. The electrodes are lowered into the paste and moved about slightly to seat them and equalize band tension. More paste is added as necessary to achieve a thoroughly wet, stable, contact area which should not extend more than 0.5 cm. beyond the electrode edges. The small excess forced from under the electrodes is permitted to remain; marked excess is removed. Electrode placement and symmetry is again checked and final adjustments are made. If properly performed, the above procedure results in an electrode–patient contact which adds negligible resistance to the circuit, remains stable throughout an hour or more of tracing, and gives a minimum value for the patient resistance.

4. *Instrumentation.*

The Schufried instrument* has been described by Jenkner.[1] While not without its peculiari-

*Radiometer Type CDM 2d Conductivity Meter and appropriate cells. The London Company, Westlake, Ohio.

†"Telectrode" electrode jelly, Telemedics, Inc., Southampton, Pennsylvania.

*Available from Schick X-Ray Company, 444 North Lake Shore Drive, Chicago, Illinois.

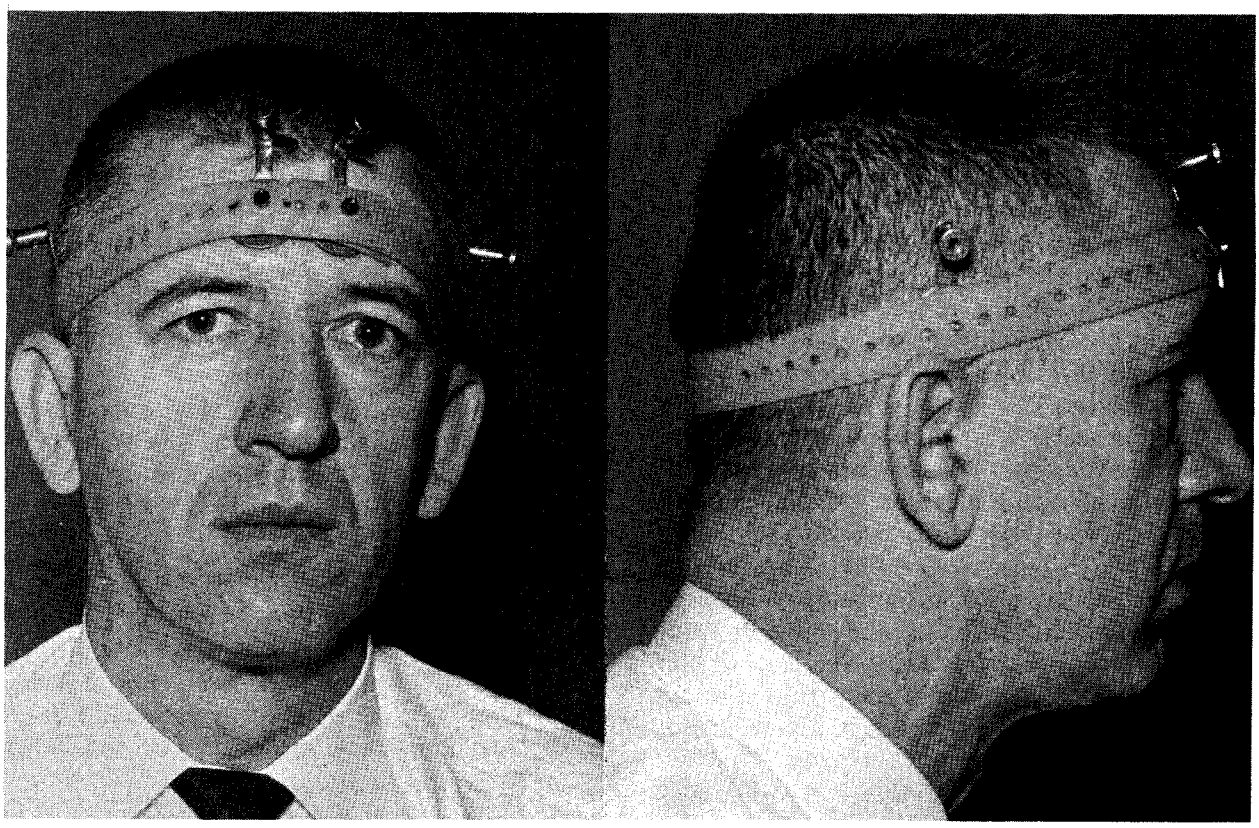

FIGURE 11. Electrode placement for rheoencephalography.

ties, we have found it more reliable than other instruments we have tested and have used it almost exclusively in our series without modification. Since this section is devoted to a description of our tracing procedure, the instrument's characteristics will not be presented in detail except where required to support specific procedures.

a. *Line Current Stability:* The Doppel-Rheograph is quite sensitive to sudden line current variations and will reproduce such artifacts on the tracing. Elevator artifacts were particularly annoying but even lights switched on or off on the same line could be detected. After unsuccessfully testing several methods, a 2000 VA constant voltage transformer† was installed to power the entire instrumentation train. It eliminated line transients almost completely.

b. *Preparation of Rheograph for tracing:* The Rheograph should be placed on a firm surface without tension on the leads to the recorder or to the patient. The patient leads should be fixed to the bed to prevent motion artifacts. The Rheograph and amplifier-recorder are turned on and allowed to stabilize for at least 15 minutes before starting a tracing. The electrodes are connected to the Rheograph and the bridges are carefully balanced on the patient setting. Several balance checks are usually performed. Resistance values are read from the dials, converted to true values from previously determined calibrations, and compared. If the procedure has been carried out properly, the values usually agree to within the balancing error of the instrument, about 5 ohms. If the values differ by more than 10 ohms, insufficient paste is usually indicated on the higher side but electrode placement, interelectrode distances, and amount of paste are rechecked. The bridges are rebalanced and the new values are compared; these will usually agree within 10 ohms. If not, a small amount of paste may be added to the higher side and the balance rechecked. Any lack of agreement at this point is probably valid anatomic variation. Balance and interelectrode distance values are recorded on the tracing. If unfamiliar with the instrument, the operator should practice balancing various known standard resistors in the patient circuit. Balance points can be repeated to within 5 ohms with practice. Balancing is most easily performed with the room darkened or the electronic indicator well shaded. The capacitance should be balanced first.

c. *Recording instruments:* A variety of strip chart recorders can be used; we have obtained useful clinical tracings from a single-channel bedside EKG machine, Grass Models III and VI and Offner Type T EEG's, and Grass, Offner, and Sanborn polygraphs. In emergency bedside situations, an interpretable tracing can be obtained using an EKG machine to record the right and left tracings in succession. However, this is far from satisfactory as tracings should be obtained simultaneously. We presently follow Jenkner's practice[1] and trace the EKG, right and left REG's, and their derivatives. The derivatives were not recorded during the earlier portion of our series.

Our earliest several recordings were taken on an Offner Type T EEG machine.* This instrument gave excellent, linear, full-scale deflections; a simultaneous EKG could not be obtained. The difficulty was later traced to interaction of the high-frequency Rheograph patient current with the preamplifier choppers. We next employed a Grass Model IIID EEG machine† which permitted simultaneous EKG and REG tracing. This instrument is reasonably linear throughout the center 20 mm. of its pen deflections; signals extending beyond this range are attenuated in amplitude with some loss of accuracy. A D.C. coupled Sanborn Model 850 polygraph§ was used for a short period; its tracings were linear but were unsatisfactory because of the wide baseline swings with respiration and movement.

Our present recording instrument is an Offner Type R polygraph modified by installing low-pass filters on the EKG inputs. This instrument has a linear amplitude response throughout most of its pen range.

We have also had excellent results using a Grass Model VI EEG and Grass Model V and VII polygraphs.

d. *Preparation of Recorder for Tracing Amplification:* The EKG tracing should be of reasonable amplitude according to customary

†Sola Harmonic Neutralized Constant Voltage Transformer, Sola Electric Company, Elk Grove Village, Illinois.

*Offner Electronics, Inc., Chicago, Illinois.

†Grass Instrument Co., Quincy, Massachusetts.

§Sanborn Company, Waltham, Massachusetts.

practice; it need not be calibrated. The REG and differential tracing channels should be set and equalized preliminary to the start of the tracing and should afford sufficient amplification for final tracing equalization using the output gain controls on the Rheograph. Once determined, these settings rarely require future change.

Frequency Response: The importance of the low frequency cutoff must be emphasized. The normal heart rate varies from 60–100 per minute while respiration rate varies from 10–20 per minute. The normal rheoencephalographic waveform thus contains significant and important low-frequency components which must be reproduced by the recorder. Our early experience quickly indicated that the low-frequency cutoff at the usual EEG setting was too high and that much of the waveform was not reproduced (Figure 12). Figure 12 illustrates how the waveform differs with low-frequency cutoff level and may mislead the investigator; here the higher cutoff level gives an almost normal appearing tracing.

A D.C. response was thought ideal. In practice, the wide baseline swings with movement and deep respiration required reduced amplification which obscured the waveforms and made tracing interpretation difficult. We therefore chose a low-frequency cutoff level of 0.2 Hz (one second time constant) for our routine tracings. This response setting offers an excellent compromise between maximum pulse wave amplitude and respiratory excursion.

The high frequency response is usually limited by the mechanical system of the recorder and is not changed. 60 Hz and muscle filters are not routinely used. Appreciable 60 Hz artifact usually indicates poor electrode contact or tube failure in the Rheograph.

Paper Speed: Standard EEG paper and corresponding paper speeds (25–30 mm. per second) are used. We have found the 100 mm. per second speed used by Jenkner[1] of little advantage.

e. *Calibration:* Adequate and reliable tracing calibration is perhaps the most important single factor in rheoencephalography. Simonson[51] has indicated that a standard tracing amplitude should be adopted for rheoencephalography and has suggested a value of 0.10 ohm per 10 mm. This value affords tracings of adequate amplitude and good visual quality; it is used routinely in our tracings.

The Rheograph calibration dial is set to the 0.10 ohm (100 mohms) calibration setting and the recorder is started. Several calibration pulses are applied to the tracing during the smoothly falling, diastolic portion of the pulse; the recorder is stopped; and the calibration pulse

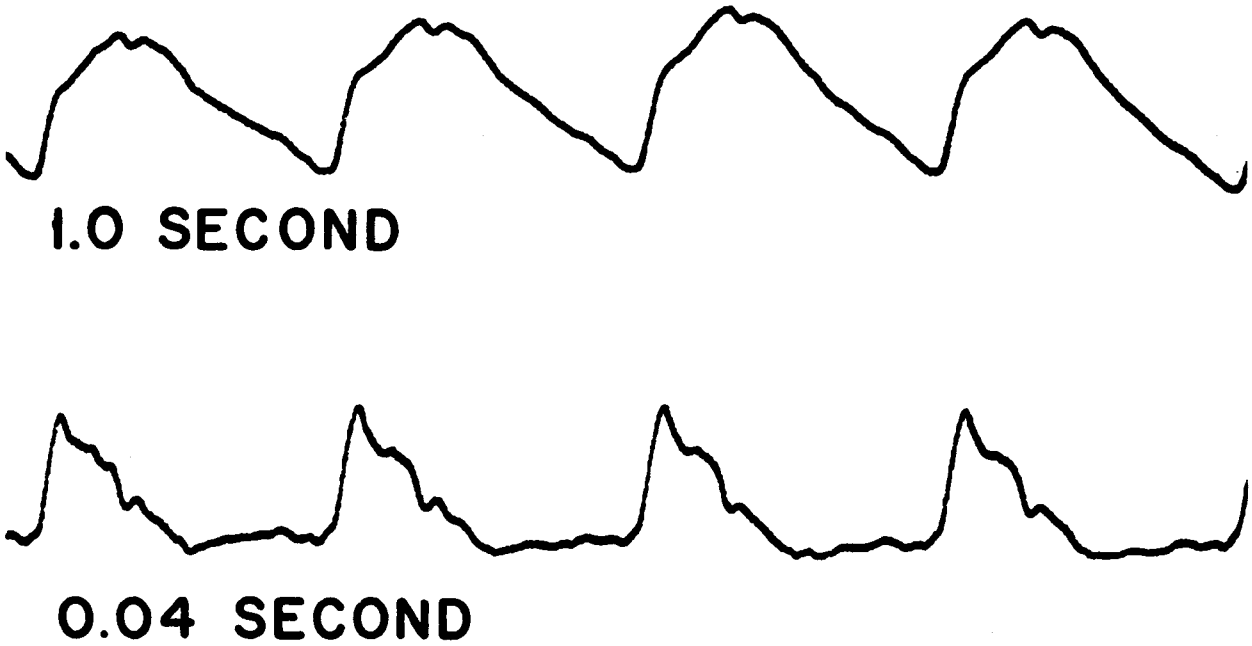

FIGURE 12. Influence of time constant on the tracing.

amplitudes are measured. If not 10 mm. on the REG channels, the Rheograph output dials are adjusted towards this value and the procedure is repeated. Several readjustments may be necessary until the channels are reasonably equalized close to the above value; exact equalization at 10 mm. is difficult to attain and is not necessary. Minor variations affect visual waveform comparisons only slightly and the quantitative measurements not at all. If the tracing amplitude is less than 6 mm. at 0.10 ohm per 10 mm., the 0.05 ohm (50 mohms) setting can be used with the tracing amplified to 0.05 ohm per 10 mm.

Once satisfactory equalization has been attained, the patient is requested to relax and refrain from moving, blinking, coughing, and swallowing. The recorder is again started and at least 30 *adequate* calibration pulses are placed on the record. These pulses should occur not more frequently than every second pulse, should appear on the falling phase after the dichrotic notch, and should be contained within the linear pen excursion range. The calibration pulse amplitude and waveform will depend slightly on the calibration button action. Firm, rapid, deliberate pressure is recommended. A certain amount of practice is usually required before properly timed, identical calibration pulses are routinely attained. Since these pulses are, in theory, square waves, the pulse appearing on the tracing should be as close to a square wave superimposed on the REG activity as the recorder frequency response will permit (Figure 13). Trains of calibrations are also entered at various stages of the tracing and at its end. These serve as a stability check during the run and help compensate for unsuspected changes which may have occurred.

A certain amount of variation in calibration pulse amplitude and shape must be accepted as peculiar to the instrumentation and tracing procedure. Under ideal conditions, the amplitude will not vary more than 0.4 mm. throughout a series but may vary as much as 2.0 mm. with some recorders. Irregular calibration pulses usually indicate dirty contacts within the button assembly; these should be cleaned as indicated. Occasionally, careful calibrations will be irregular, too small, or otherwise appear aberrant on a seemingly clinically adequate tracing. This is one of the innate perversities of inanimate objects for which we have no explanation; it may be inherent in the Rheograph circuitry. The best solution we have found is to shut off the equipment and try again later.

A word of warning is necessary. Should it be necessary to readjust, repaste, or otherwise disturb the electrodes at any time during the tracing, or if it is suspected that the electrodes have slipped or otherwise changed position, *the bridges must be rebalanced and the tracing must be recalibrated.* This is necessary since any such disturbance changes the balance point and, therefore, the tracing calibration amplitude. Further, since the bridges cannot be rebalanced closer than to within several ohms of the same point, recalibration must always be performed after balance checks or changes during a tracing. Balance values should always be recorded on the tracing.

It should be noted that the use of the standard resistance setting on the Rheograph has not been mentioned as part of our procedure nor has its possible use in calibration been presented. This Rheograph setting is intended for use in the preliminary adjustment of the recorder gains as suggested in the Rheograph instruction manual. Since we equalize channels using calibration pulses on the patient tracing, we have found this setting of little use except when trouble-shooting. Further, one investigator[52] has advocated calibration on this setting alone and has indicated not only that further calibration on the patient tracing is unnecessary but also that calibration on subsequent patients throughout the day is unnecessary. This usage is contrary to that presented in the Rheograph instruction manual and to that of Jenkner[1], many of whose illustrations clearly show calibration pulses on the tracing. We have investigated this point and find that *neither* the calibration pulse amplitudes *nor* their ratios transfer from balance on the standard setting to balance on the patient setting. Although inconvenient and somewhat wasteful of time, it is absolutely necessary to calibrate *each* time the bridge has been balanced whether on a new patient or during a tracing run.

Our pretracing procedure has been detailed at length for several reasons: first, to facilitate its repetition by others; second, to propose it as a standard method for rheoencephalography; third, to indicate possible sources of error inherent in rheoencephalography; and fourth, to present our reasoning and approaches. This

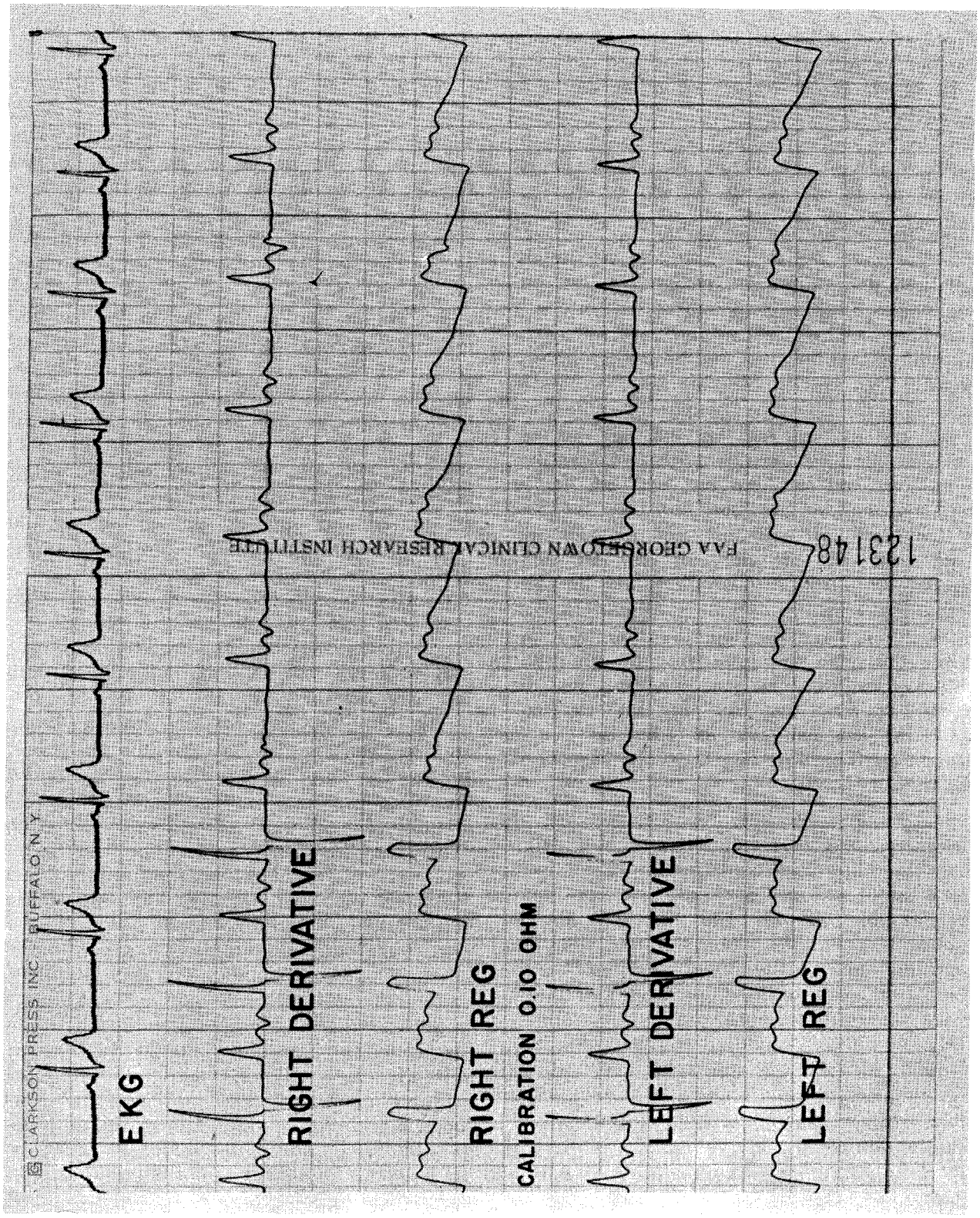

FIGURE 13. Page from a normal REG illustrating channel sequence and calibrations.

pretracing procedure is followed on all human tracings performed in our laboratory, whether diagnostic or research studies, and has been very successful in affording interpretable tracings; it can also be used for animal studies with appropriate modifications. Our diagnostic tracing procedure will be similarly detailed.

C. *Diagnostic Tracing Procedure:*

1. *Reference Tracing:* After an adequately equalized and calibrated tracing has been obtained, the recorder is permitted to run for a sufficient period of time to give an *artifact-free* train of at least 30 pulses and/or five respiratory cycles. This portion of the tracing will later be used as the reference, comparison, or baseline tracing; it is important that sufficient artifact-free tracing be obtained to represent the patient reliably throughout subsequent analyses. If the patient is tense or movement-prone, the recorder is stopped and he is again cautioned that movement delays the procedure. If the patient is restless and unable to coperate, a long artifact-free sequence may be difficult to obtain. In such cases, sufficient tracing should be obtained to assure that enough representative pulses are available for analysis. Movement artifact may be lessened by a not uncomfortably tight electrode strap. Various manipulations or maneuvers may next be performed depending on the purpose of the tracing.

2. *Arterial Occlusions:* The importance of rheoencephalographic monitoring during deliberate arterial occlusions has been apparent from the onset of our studies. Such occlusions have been employed as part of all clinical diagnostic studies in our series[49] except where clinically contraindicated or prevented by circumstances. Partial or complete deliberate carotid occlusion is not new to rheoencephalography[1,5] but apparently has not been employed routinely. We now routinely occlude the superficial temporal arteries unilaterally and bilaterally. Other accessible arteries may be occluded as part of the study as desired.

a. *General:* We usually wear insulating plastic or rubber gloves when performing occlusions, as suggested by Jenkner[53] in questioning the validity of our results. We have investigated this question in a series of patients by performing each occlusion alternately with and without rubber gloves; no difference between the procedures could be detected and identical results were obtained.

The procedures are always described to the patient and he is warned to expect a small amount of localized discomfort, especially during carotid manipulation. He is also again warned that movement of any sort may disturb the tracing and make repetitions necessary.

The operator may stand behind the patient's head or on either side as desired. He should be in a position which is as comfortable as possible and which he can sustain throughout the manipulation without transmitting movements or tremors to the patient. The operator should not contact the bed frame during the tracing period.

The recorder is always positioned so the operator can monitor the tracing visually. This factor is important in obtaining adequate tracings safely. Artifacts and variations in technique are immediately apparent to the operator and permit immediate correction; the completeness of the occlusion can be estimated; and the need for further repetition quickly determined. Further, if a carotid unsuspectedly supplies a majority of the total cranial circulation, this will also be immediately apparent and the procedure can be terminated quickly.

The artery to be occluded is found manually with one hand, using the other for assistance or for head bracing, and is lightly held beneath one or two fingers. The artery should not be occluded during these preliminary maneuvers. When ready, the operator requests that the recorder be started and observes the tracing. When the waveform has stabilized and appears similar to the reference waveform, about a page of tracing is obtained. The artery is then compressed as the operator simultaneously signals his assistant who marks the tracing. The occlusion is held until the tracing stabilizes and a sufficiently long, representative sample has been obtained. The artery is then quickly released with a simultaneous signal to the assistant who again marks the tracing. The procedure is repeated after the tracing has returned to stability. At least two apparently identical occlusion tracings should be obtained, preferably three. More may be necessary if difficulty is encountered. The operator should hold his position until the run is completed. Comments may be made to the assistant for immediate chart entry where indicated but should not attract the patient's

attention. Thus, we usually report "Good" if the operator believes the compression is complete or "Poor" if incomplete or artifactual.

Calibrations may be entered as desired and are routinely entered after the temporal and carotid occlusions but before the final reference waveform run.

Occlusions should be performed firmly, quickly, and completely, without hesitancy, fumbling, slippage, or movement artifact. Sufficient pressure should be applied over sufficient vessel length to obliterate the flow and insure *complete* occlusion. The area surrounding the vessel and included in the compression is kept to a minimum to avoid interference with venous return. Significant venous occlusion is quickly recognized by an increase in pulse amplitude rather than the expected *decrease* and is probably the basis for the amplitude increases observed by Perez-Borja and Meyer[10] during carotid compressions. In our experience, one finger is sufficient for smaller arteries, such as the superficial temporals, while two or more may be required for the carotids. If the operator is properly positioned and his fingers are properly placed, only the latter need to be moved in effecting an occlusion. Compression techniques will vary according to the operator's previous training and personal preferences. The important criteria are completeness, non-interference with venous return, rapidity, and minimum motion and circulatory artifact. Practice is usually required before reliable tracings are obtained routinely, no matter how experienced the operator may be.

b. *Superficial Temporal Arterial Occlusions:* These arteries are easily accessible and are easily occluded completely. They contribute most of the extracranial tracing component and can be considered to represent their related external carotid arteries as will be demonstrated later. We have found that positioning the hands for bilateral simultaneous compression is of value prior to starting the temporal occlusions; during single artery occlusions, the contralateral hand braces the head without touching the contralateral artery. With both hands in place, the operator can rapidly compress one artery, the opposite artery, and then both, with a minimum of artifact. As previously noted, sufficient time should be allowed for the tracing to stabilize between compressions.

c. *Common Carotid Arterial Occlusions:* We have had only brief, transient, reversible reactions to this manipulation throughout our experience. We have had no fatalities, no prolonged or irreversible sequellae, and few reactions lasting more than 1–2 minutes. We ascribe our experience to good fortune, caution, and the carefully safeguarded techniques we employ. We are well aware of the possible dangers of carotid manipulation,[54] would much prefer to eliminate it from our procedure and will do so when a substitute which is equally effective in diagnosis is developed. In the interim, we will continue to include common carotid arterial occlusions in our rheoencephalographic armamentarium since we consider that its diagnostic value far outweighs its possible dangers to the patient. Since we have placed ourselves in the hopefully temporary position of advocating the use of common carotid occlusions during diagnostic rheoencephalography, we will present our method at length to illustrate the safeguards employed and our methods of avoiding potentially dangerous reactions.

Operator: A physician is always present throughout the procedure and performs all compressions. He is responsible for patient evaluation and controls the tracing situation.

Patient Selection: We do not routinely perform carotid occlusions on patients who show cardiac irregularities, who are in extremis, obviously seriously ill, or markedly debilitated; who have calcific, rigid, inelastic, or possibly friable arteries; or who are so restless, irritable, or uncooperative that an interpretable tracing probably cannot be obtained. We have performed carotid occlusions on such patients when the possible diagnostic value of the manipulations outweighed the patient risk.

We also have one final category. We do not perform occlusions on patients whom our medical intuition suggests may be poor risks.

Patient Position: This has been discussed. Proper initial patient position reduces the total amount of carotid manipulation and the need for multiple repetitions. It is particularly important with obese or thick-necked patients in whom carotid access is difficult.

Occlusion Site: Particular care is taken to compress the common carotids at a site as distant as possible from the carotid sinus and

bifurcation. Attention to this one point has completely eliminated carotid sinus reactions and has probably avoided the sequellae arising from manipulation of diseased bifurcations.

Patient Warning: As with other portions of our procedure, the manipulation is described to the patient and he is warned to expect some local neck discomfort. Possible neurological symptoms such as faintness, dizziness, weakness, numbness, and other unusual or peculiar feelings are described and he is cautioned to report them and *any* other *odd sensations immediately* so the compression can be discontinued. He is also cautioned of their possible significance and bravery is discouraged.

If the patient makes any report, reacts, *or attempts to do so*, the occlusion is immediately discontinued; the reported symptoms are entered on the tracing. The occlusion may or may not be repeated after the tracing has stabilized, depending on the adequacy of the immediate and previous similar occlusion tracings which may have been obtained.

Visual Monitoring: We have one important safety factor in our favor; we can visually monitor the cranial circulation via the REG tracings. With experience, the operator soon recognizes abnormally low pulse amplitudes and can discontinue occlusions after 5–10 representative pulses have been recorded but *before* symptoms develop or are reported.

Duration of Occlusion: If not discontinued for other reasons, the occlusion is held until 15–20 reasonably similar, presumably representative pulse waves have been recorded. *Bilateral* common carotid occlusions are performed *only* on those patients in whom both carotids are easily accessible and easy to occlude. Such occlusions are held *very* briefly, only long enough to obtain 3–5 interpretable pulse waves. Longer single and bilateral occlusions have been performed but only for research purposes in normal subjects who were able to tolerate the procedure without symptoms and who were adequately warned.

3. *Post-Occlusion Tracing:* After satisfactory completion of the occlusions and return of circulatory stability, the tracing is recalibrated and an adequate final sample of reference tracing is obtained. The procedure is usually terminated at this point although other additions may be made at the investigator's option. When included, Queckenstedt and Valsalva maneuvers should be performed *after* the occlusions since these maneuvers significantly alter circulatory dynamics for an appreciable time.

D. *Post-Tracing Procedure:* Bridge balances and interelectrode distances are rechecked and the values recorded. All electrodes are removed from the patient, who receives appropriate cleanup. The electrodes and head-band are carefully washed to remove all traces of paste and dried prior to reuse. All meaningless pages used in the pretracing procedure are removed and discarded after transferring any useful information therein to the remainder. The record is identified with the patient's number, name, address, age, referring physician, and so on. The plethysmographic instrument used, electrodes, interelectrode distances, balance values, and so on, are recorded. Arterial occlusions are listed as are any appropriate remarks or observations. The channels are checked and identified as is the calibration pulse amplitude. The record is finally scanned and the occlusions are identified and clearly marked with any remarks or observations.

E. *Final Comments:* On first reading, the above detailed procedure may seem lengthy and time-consuming; in practice it is neither. The basic procedure is quite simple, easily learned, and rapidly becomes automatic for technician and operator. A complete rheoencephalogram can be obtained in the time required for a routine electroencephalogram, or less if the patient is stable and cooperative. Instrumentation problems most frequently cause delays. They cannot be anticipated, and usually are transient. We have completed tracings in 30 minutes or less although our usual time is about 45 minutes; we schedule an hour per patient.

As is true for any new or unfamiliar procedure, a certain amount of practical experience is required before adequate tracings can be obtained routinely. The technique is tolerant of variations and qualitatively useful tracings may be obtained almost immediately; for example, the tracing diagnosis in our second case[49] is obvious without detailed mathematical analysis. However, if there is to be any hope of determining firm diagnostic criteria from rheoencephalograms, the investigator must accept the requirement of a rigidly standardized technique.

II. Tracing Interpretation:

Our initial evaluation of the diagnostic possibilities of the rheoencephalographic tracing suggested it offered two types of information which were important. Obviously, "abnormal" pulse waveforms should be significant; particular waveforms might be diagnostic of particular diseases as is common in electrocardiography. Further, since rheoencephalography was a potentially quantitative method, tracing amplitudes should be significant; mathematical analysis might give a description of the intracranial circulation in sufficient detail to be diagnostic. We routinely employ both approaches in tracing interpretation. Mathematical analysis has afforded more consistently correct predictions throughout our study and will be presented first.

A. *Mathematical Analysis:* Briefly, the procedure involves three steps:

1. The determination in ohms of tracing amplitudes which represent the right and left sides of the cranial circulation in the unmanipulated state and during arterial occlusions;

2. The calculation of various arterial contributions to the cranial circulation and their comparison; and

3. The formulation of a tentative diagnosis.

1. *Tracing Measurement:*

a. *Equipment:* The equipment required is that customarily used for tracing measurement. In our experience, amplitudes can be scaled off the tracing to 0.1 mm. without difficulty; a millimeter scale readable to this tolerance is advisable.* Since the width of inked lines varies and is appreciable, we measure all calibration and pulse amplitudes from lower edge to lower edge rather than from lower edge to upper edge.

b. *Calibration:* The tracing is rapidly scanned to determine the sequence, location, and interpretability of the various portions and the last set of calibrations before the tracing proper is identified. Sufficient of the final, adequate calibration pulses are measured on each REG channel to give reliable average values and the channel calibration factors in ohms per mm. are calculated. These factors usually remain constant unless the electrodes or bridge balances are disturbed. A few calibration pulses should be measured wherever encountered through the tracing to check for reasonable constancy. If any marked change is noted, new calibration factors should be determined.

c. *Reference Tracing:* The unmanipulated tracing is inspected and a representative artifact-free portion comprising at least ten pulses and two or more complete respiratory cycles is selected for measurement. Pulse amplitudes often vary considerably throughout the respiratory cycle but are reasonably consistent when compared at specific times within successive cycles. Thus, the interpreter may start at any place within a respiratory cycle but must measure in cycle multiples to obtain representative average values. We draw fine, narrow, straight lines from pulse to pulse, tangent to the tracing lower edge at the points at which systolic inflow apparently begins. In an occasional patient, the trace may dip below the pencil line; this is disregarded. Maximum pulse amplitudes are then measured from the pencil line, averaged, and converted to ohms, using the previously determined calibration factors. These values are the reference or baseline pulse amplitudes.

d. *Occlusion Tracings:* All similar occlusions throughout the tracing are inspected and one which appears representative of the group is selected for measurement. Our selection criteria are minimum average amplitude, freedom from artifact, consistency of waveform with other tracings in the occlusion series, and equivalency of the pre-occlusion waveforms with the reference waveform. A typical ipselateral occlusion tracing channel usually shows a brief occlusion artifact for one or two pulses, a series of low-amplitude pulses followed by several of higher amplitude, and finally, throughout the remainder of the occlusion, relatively stable, intermediate amplitude pulses showing only respiratory variations. In young normal patients a trend towards slight amplitude increase may be seen in prolonged occlusions. Prolonged occlusion artifact may obscure some of the above variations. Lesser but analogous changes are seen in the contralateral tracing.

Ideally, measurement should begin with the first pulse after complete occlusion and include sufficient succeeding pulses to give a representative average. We prefer to measure as many pulses per occlusion as were measured for the reference tracing. Interestingly enough, the average of the initial low and high values is

*Paragon Triangular Scale #56-3662, Keuffel & Esser Company, Hoboken, New Jersey.

frequently close to the average of the remaining, more uniform pulses. Measurement should start after the trace has stabilized if the first several pulses cannot be used. Multiple respiratory cycles should be measured if detectable.

Average occlusion amplitudes will always be less than the corresponding average reference amplitudes when occlusions of a significantly functioning artery are performed properly and are complete; the only exception to the above rule occurs when the patient's circulation has altered from the reference state during the tracing. Such higher amplitudes are occasionally encountered in temporal arterial occlusion tracings, particularly in young normal subjects; we have never encountered increased amplitudes during correctly performed carotid occlusions. This will be discussed under error analysis. Occlusion values occasionally may be several milliohms greater than their corresponding reference values. We use the reference values as more representative since the values are probably identical within experimental error.

The above discussions apply quite generally to all tracing measurements. There are several classes of patients on whose tracings such measurements are more than routinely difficult:

First, patients with cardiac arythmias whose stroke output is randomly variable. Such tracings reflect the abnormality as variable pulse amplitudes; representative carotid occlusion amplitudes are, therefore, difficult to obtain, particularly since prolonged occlusion usually cannot be tolerated. The interpreter must devise his own criteria; we have traced one such patient and can make no recommendations.

Second, patients with markedly irregular and deep respiration which cannot be controlled. Again, we have traced but one such patient (the same one mentioned above) and can make no recommendations.

Third, patients with frequent extrasystoles. We consider such pulses to be artifacts and obtain sufficient tracing to ensure artifact-free averages.

Fourth, patients on whom bilateral carotid occlusion results in an almost flat, almost featureless curve. The interpreter frequently has a decided problem in locating the systolic inflow points defining each pulse and in placing his penciled baselines. Here, the EKG tracing offers the best clue and affords intervals which are transferable to the REG channels. Systolic amplitude rise can occur at quite long and often variable intervals after the EKG wave and may be almost unrecognizable. No firm rules can be suggested; each tracing must be individualized from the interpreter's experience.

Bilateral carotid occlusions are difficult to perform, usually cannot be prolonged sufficiently to obtain reliable averages, and may give quite variable amplitudes and waveforms. We select comparison values from either single or average amplitudes.

e. *Error analysis and data selection:* The interpreter obtains average pulse amplitudes for the various temporal and carotid arterial occlusion tracings according to the above general procedure. These and the reference amplitude values are tabulated according to the notation on page 11. The occlusion values are inspected and, if all are less than or equal to the reference values, simultaneity is checked.

Values for the bitemporal compressions are calculated from Equations (22) and (23) and are compared to the corresponding observed values, R_4 and L_4. Initial agreement within 2–3 milliohms is not unusual; the series is acceptable. If disagreement between observed and calculated values exceeds three milliohms, the tracing measurements are re-examined for possible error sources.

Since different interpreters may employ different approaches to error analysis and have different concepts of data selection, no attempt will be made to discuss all possibilities. We will detail those procedures we have found most useful; the interpreter may devise others as necessary. If one or both observed bitemporal occlusion values are larger than their calculated values, the major error is probably in the former; therefore, the bitemporal occlusion tracings are examined first.

A number of possible sources of error in a particular average occlusion amplitude should be considered. First, the measured occlusion may have been incomplete unbeknownst to the operator; measurement of a second, more representative occlusion tracing may be necessary. Second, the sample measured may have been too short; one or more additional respiratory cycles may give a more representative average. Third, particularly in young normals, there may be a

trend towards increasing amplitude as the occlusion continues; measurement of only enough initial post-occlusion pulses to give a representative average may be necessary. Fourth, aberrantly formed pulses may be present which can be discarded. Finally, one or more pulses may have waveforms similar to the remainder but have amplitudes considerably lower or higher than the remainder (usually higher). Such pulses can be discarded unless they recur at regular intervals and must be considered valid observations. A thorough analysis of the data as above will usually result in excellent simultaneity. If not, the interpreter has one further recourse which we have found useful.

The above analysis assumes arterial contributions to the tracing remain constant throughout the tracing period. It is possible that a transient variation may have occurred before a particular occlusion but with later reversion to the reference state; likewise, a permanent change may have occurred after one or more occlusions. These changes can be unilateral or bilateral; they are usually the explanation for the previously mentioned compression values larger than the reference values; and they are easily recognized by associated changes in the pre-occlusion reference pulse waveform and its amplitude. In our experience, the most reliable occlusion tracing before the carotid occlusions is that during bitemporal arterial occlusion.

If any of the unilateral temporal arterial occlusion values are significantly larger than their reference values, or if adequate simultaneity has not been obtained after the above error analysis, we first check intra-tracing calibrations for instrumentation stability. If calibration changes are not a factor, we select the occlusion tracing which appears most likely erroneous and average the immediately preceding reference waveforms on each channel. In practically all such cases, these amplitudes are significantly larger than the initial reference amplitudes. The ratios of the initial reference amplitudes to the pre-occlusion reference amplitudes are used as factors to "normalize" the high occlusion amplitudes to the initial reference state. The resulting simultaneity is usually excellent. These normalization factors frequently agree within several percent between channels and suggest equivalent bilateral changes; the change may also be unilateral. A number of occlusion tracing amplitudes may appear high and have prior reference values agreeing closely for each channel but differing significantly from the initial reference values. Rather than normalize a large number of values to the initial reference amplitudes, we use the later reference values, particularly if they agree with the reference values appearing at the end of the tracing. If the above methods are unsuccessful, the remaining error must be accepted. It has not exceeded six milliohms in our temporal occlusion series.

Error analysis based on observed bilateral carotid arterial occlusion values is considerably more difficult since this maneuver is neither easily performed nor measured. Equations (24) and (25) are used to calculate values which are compared with R_7 and L_7 as with the temporal occlusions. Occasionally, the measured pulse amplitudes are too high; the bilateral occlusions were presumably incomplete and are worthless. Frequently, one pulse or a few pulses from each channel may agree within five milliohms, the others being too high; these lower observed values are accepted. Occasionally, all goes well and a stable, relatively long, low-amplitude tracing is obtained, its averaged values usually agreeing closely with the calculated values. If the observed values are significantly lower than the calculated values, an infrequent occurrance, the unilateral occlusion values are analysed as described for the temporal occlusions. Excellent agreement is usually easily attained. Occasionally, agreement may be within 1–2 milliohms on one channel but less close on the other. We can offer no explanation.

In general, if the tracing has been properly obtained from a stable patient, the initial averages often give excellent simultaneity. The majority of the remaining tracings requires only rejection of aberrant values, enlargement of one or more samples, or inclusion of pulses occurring earlier in the occlusion tracing to achieve simultaneity. The full battery is rarely required and normalization almost never. The latter has been used three times to salvage tracings obtained from young normals who were not available for retracing. Fortunately for its use as a diagnostic tool, older patients with cerebrovascular disease usually give stable and self-consistent tracings; young normal subjects with labile extracranial circulations present the most difficult tracings to analyse. In the latter cases, it is sometimes

easier to obtain a second tracing at another time when the subject is more relaxed, better rested, or has eaten.

Finally, as we have attempted to indicate throughout the above discussions, our concepts of tracing analysis are based on two points: First, values should be obtained from the tracing which are as representative of the tracing as possible. Second, only those methods of analysis should be used which can reasonably be derived from the data itself. While our initial diagnostic successes suggested our approach was valid, the remarkable self-consistency of our method was not suspected. When we dared attempt bilateral carotid occlusions, it was immediately manifest and, in turn, dictated further refinements in analysis.

2. *Calculation of Arterial Components:* Once obtained, the average values are tabulated according to the notation on page 11 and used in the formulae on page 12 to calculate arterial contributions. The calculations are simple and require no discussion. Negative values may be encountered on rare occasions; such values are, of course, impossible. If the data is adequately simultaneous, negative values usually fall within the experimental simultaneity error and may be considered zero. Negative values larger than experimental error were encountered in the initial portion of this study when the possibility of checking simultaneity was unsuspected. Carotid occlusion tracings were brief; the small samples were skewed by the initial low amplitudes.

We next tabulate the arterial contribution values and calculate various percentage ratios to assist diagnosis. We have found the following sets of ratios (or their inverses) valuable:

1. $\frac{RT}{LT}, \frac{RT}{RIC}, \frac{RT}{RCC}, \frac{LT}{LIC}, \frac{LT}{LCC};$

2. $\frac{RP}{LP}, \frac{RIC}{LIC}, \frac{RCC}{LCC};$

3. a. If the NICS is supplied by the right internal carotid artery to the left hemisphere:

$$\frac{PA_R}{RP}, \frac{PA_L}{LP}, \frac{RIC-NICS}{RP}, \frac{NICS}{LP}, \frac{LIC}{LP};$$

or:

b. If the NICS is supplied by the left internal carotid to the right hemisphere:

$$\frac{PA_R}{RP}, \frac{PA_L}{LP}, \frac{RIC}{RP}, \frac{NICS}{LP}, \frac{LIC-NICS}{LP}.$$

Other appropriate ratios can be used to demonstrate particular interrelations.

Reference to the assumptions on which the above method of analysis is based, page 10, indicates that there is some theoretical inaccuracy in directly comparing the temporal with the internal carotid values since their factors for conversion to blood volumes may be different. Similarly, the common carotid values are not strictly analogous to blood volumes but are also useful approximations if this inaccuracy is recognized.

3. *Provisional Diagnosis:* A clinically valid method should yield reliable and objective information about the patient without prior reference to the clinical findings. To be maximally useful, such information should clearly indicate the presence of disease and offer criteria for its diagnosis without clinical reference. Failing this, the information should indicate the diagnosis or permit selection between alternate diagnoses when correlated with the clinical findings. Equivocable or misleading information should be minimal and easily recognized. If reliable and objective information cannot be obtained, the method offers little of value to the clinical situation. We have attempted to derive diagnostic patterns from the tracing itself based on our concept of cranial circulatory physiology and its relation to the tracing; it should particularly be noted that such analysis does *not* require prior knowledge of the clinical history and findings. In our view, clinical information unnecessarily prejudices the interpreter's judgment and may lead to conclusions which, while perhaps correct, are unwarranted from the data.

Detailed analysis of the patient's cranial circulation is now possible. We will discuss the normal pattern, present illustrative patterns we have found associated with various types of vascular disease in this case series, and finally describe our general approach. As stated on pages 6 and 14, disease may or may not alter the cranial circulation sufficiently to alter the tracing. Rheoencephalography, in common with all diagnostic methods, has inherent limitations;

some alterations may fall within the patterns of normal variation; some may be borderline or suspicious; some may be definitive; and some may be uninterpretable or actually misleading.

a. *The normal circulatory pattern:* In theory the ideal normal head should show complete lateral anatomic symmetry in all of its structures; the ideal normal cranial circulation should equally show the same complete lateral symmetry in all of its components. Therefore, the ideal rheoencephalogram should show the same lateral symmetry, within the limits of experimental error, if the calculated arterial components are valid analogs of the contributions of their arteries to the cranial circulation. The ideal normal pattern is obvious and, in terms of the previous ratios, is:

$$\frac{RT}{LT} = \frac{RIC}{LIC} = \frac{RCC}{LCC} = \frac{RP}{LP} = \frac{R_1}{L_1} = 100\%$$

$$NECS = NICS = 0$$

In practice, a completely symmetrical tracing is rarely obtained; as might be expected, tracings obtained from a series of apparently normal subjects give different ranges of values for each of the component amplitudes and their ratios.

b. *Abnormal circulatory patterns:* The successful interpretation of abnormal patterns depends greatly on the interpreter's familiarity with the pathophysiology of the cranial circulation associated with various diseases and his previous experience in this type of analysis and its limitations. Certain patterns are immediately and obviously pathognomonic; others may be obscured by individual variations. Patients with several concurrent diseases may give tracings that can tax the interpreter's ingenuity.

(1). *Common carotid arterial occlusions:* If a common carotid artery is completely occluded at or below the carotid bifurcation and collateral has become established, the pattern is easily recognized. The ipselateral temporal, internal carotid, and common carotid values are zero; the corresponding contralateral values are markedly increased; both transmidline shunts are markedly increased, arise from the contralateral circulation, and supply the ipselateral circulation; one or both posterior values are increased; and the ipselateral perfusion may be slightly smaller, the same, or larger than the contralateral perfusion depending on the effectiveness of collateral compensation.

(2). *Internal carotid arterial occlusions:* If an internal carotid artery is completely occluded at or above the bifurcation, the external carotid artery is not impaired, and adequate collateral is established, the pattern is again clear. It follows the first case except for appreciable temporal values and for the ipselateral common carotid value which is equal to or somewhat larger than its temporal value.

(3). *Bilateral carotid arterial occlusive disease:* This case is more difficult to diagnose. Lesions may be incomplete and may occur above or below the bifurcations; many patterns are possible with or without adequate collateral from the posterior circulation. In general, the posterior values will be markedly increased and reflect the carotid decreases; the carotid values will be decreased significantly below normal while their ratios will indicate the artery with greater involvement; and the shunts will arise from the less involved artery to supply the more ischemic side. If both carotid circulations are relatively equal, their ratios may fall within the normal range; the only clues may be normal or increased posterior values in a set of otherwise uniformly low values.

c. *General Approach:* The first three examples presented above illustrate how analysis patterns are associated with cranial circulatory disease and can be used diagnostically. Unfortunately, the interpreter is frequently faced with an unfamiliar pattern for which he must devise a diagnosis. In such circumstances, we have found a strong sense of logic to be most useful, particularly when coupled with a knowledge of cranial pathophysiology. Further, it is important for the interpreter to be aware that the current field of each electrode pair monitors a relatively constant geometrical volume irrespective of its contents (page 10). Finally, the interpreter should have some familiarity with the normal range of values for each arterial component and ratio.

We will use a hypothetical case to illustrate our reasoning. A tracing from a 56 year old

male has been analysed and the following arterial component values determined:

RT=.032 ohm	LT=.036 ohm
RIC=.067 ohm	LIC=.056 ohm
PA_R=.010 ohm	PA_L=.015 ohm
RP=.068 ohm	LP=.080 ohm
RCC=.099 ohm	LCC=.092 ohm

NECS=.004 ohm left-to-right
NICS=.009 ohm left-to-right

Their ratios are:

1. $\frac{RT}{LT}=89\%$ $\frac{RT}{RIC}=48\%$ $\frac{RT}{RCC}=32\%$

$\frac{LT}{LIC}=64\%$ $\frac{LT}{LCC}=39\%$

2. $\frac{RCC}{LCC}=108\%$ $\frac{RIC}{LIC}=120\%$ $\frac{RP}{LP}=85\%$

3. $\frac{PA_R}{RP}=15\%$ $\frac{RIC-NICS}{RP}=85\%$

$\frac{PA_L}{LP}=19\%$ $\frac{LIC}{LP}=70\%$ $\frac{NICS}{LP}=11\%$

The arterial component values are first examined for abnormality; all appear normal although perhaps somewhat high for the patient's age.

The ratios are next examined for abnormality. The temporal ratios appear normal; therefore, if disease is present, it has either spared the external carotid circulation or is entirely intracranial.

The second set of ratios is next examined. The common carotids are almost equal, which supports extracranial sparing; the left internal carotid is somewhat decreased (or the right increased) but the ratio is not significantly abnormal. Both carotid ratios agree within 12% and are self-consistent; the carotids are reasonably symmetrical. If disease is present, it does not affect the carotids. Therefore, the disease must be not only intracranial but also may lie above the circle of Willis. The perfusion ratio is now examined and is found significantly reduced in opposite symmetry to the carotid ratio. Right hemispheric disease may be present.

The third set of ratios is now examined. The posterior ratios are equivalent and normal. The internal carotid ratios are relatively normal although the right internal carotid not only supplies most of the circulation to the right hemisphere, despite the apparent perfusion decrease, but also supplies a portion of the left hemisphere.

The salient features of this patient's circulatory pattern are now established. The carotid systems and circle of Willis are normally and adequately patent if not slightly increased; the right cerebral perfusion is decreased; and the right internal carotid supplies an appreciable part of the left perfusion. The interpreter now must decide if this pattern suggests a possible diagnosis.

The decreased right perfusion first suggests ischemic or occlusive disease. If true, the right internal and common carotid should be reduced, the left internal carotid should be increased and supply collateral *to* the right perfusion, and the posterior components should be increased. Therefore, primary ischemic vascular disease is unlikely. The interpreter must look to other etiologies for an explanation of the pattern.

The reversed NICS is the important clue. While the intracranial circulation monitored by the right electrode pair is decreased, the intracranial circulation monitored by the left electrode pair has increased. Each electrode pair monitors a relatively constant geometric volume irrespective of its contents; the right decrease and left increase suggest a partial displacement of the brain and its circulation to the left. The diagnosis of relatively avascular, space-occupying lesion on the right is now clear; the slightly increased amplitudes suggest an increase in intracranial pressure.

Unfortunately, patients frequently have a combination of diseases and present difficult patterns to diagnose. In such cases, the interpreter should try various combinations of diseases and anomalies consistent with the pattern. Frequently, a definite diagnosis cannot be reached and a series of probabilities must be listed; occasionally, only lateralization can be determined (which is usually all that is necessary clinically); rarely, no conclusion at all can be reached. In these latter cases, significant information can often be obtained from analysis of the waveform and its timing. We routinely use both approaches and interpret waveform changes in terms of the possibilities suggested by the mathematical patterns.

B. *Waveform Analysis:* In our experience, both the reference and bitemporal arterial occlusion

pulse waveforms are significant; the latter occasionally show changes which are obscured by the extracranial circulation. In analyzing waveforms, we consider three factors: basic waveform, superimposed notching, and timing. The following discussion will assume no cardiac abnormalities are present since these can significantly alter the waveform in an otherwise normal patient.

One or more representative or typical waves are usually studied.

1. *Basic Waveform:* Various REG waveforms are shown in Figure 14.

Theoretical analysis of the plethysmogram[55] indicates arterial inflow and venous outflow are equal at waveform maxima and minima. Arterial inflow exceeds venous outflow during rising portions of the curve; the reverse is true during falling portions of the curve. We assume that the initial portion of the normal curve, Figure 14(a), from end-diastole (1) to peak amplitude (2) primarily reproduces arterial inflow and filling of the vascular bed, while the final portion from (2) to end-diastole (1) primarily reproduces vascular emptying and venous outflow. We designate these phases as "inflow" and "runoff." In the normal case, Figure 14(a), inflow and runoff are rapid.

Deviations from the normal may give some estimate of the underlying disease if the interpreter considers how such deviations can arise by alteration of normal circulatory mechanisms. Thus, the abnormal waveform, Figure 14(b), shows delayed or retarded inflow which may be due to flow restriction in the major arteries or to a more generalized inability of the vascular bed to fill normally. The abnormal waveform, Figure 14(c), shows rapid inflow but delayed runoff and may be due to continued filling, inability of the vascular bed to empty readily, or to venous outflow restriction. These may be combined in generalized or multiple diseases, Figure 14(d). Presystolic waves are often seen and are probably normal.

2. *Notching:* The presence or absence of superimposed notching and its location in the waveform is important. Thus, a well-marked dichrotic notch (3), Figure 14(a), suggests that the cranial arteries are normally elastic and responsive to pressure changes, while flattening of the notch or its absence suggests increased vascular wall rigidity.[56] A well-marked anachrotic notch was seen in a case having markedly increased intracranial pressure[49]; the waveforms were similar to those later presented by McHenry[11] and may be pathognomonic.

3. *Timing:* Jenkner[1] discusses this factor extensively. A marked peak timing delay, when present, is usually significant. Its significance must, however, be determined in combination with the remaining tracing information since delays can arise from asymmetries in the extracranial circulation. A delay on the bitemporal arterial occlusion tracing is almost always significant.

The interpreter can usually reach a diagnosis from synthesis of all the information available from the tracing. Rarely, disease may be missed despite the above analysis. Failure occurred with an arteriovenous fistula suggesting either that further analytic criteria must be developed or that the method or our procedure is insensitive to this disease.

It should be noted that the above analysis is based on the assumption that the patient's circle of Willis originally was widely patent and had the normal or classical pattern; anomalies in an otherwise normal patient may mimic occlusive disease. If both anterior cerebral arteries are solely supplied by the right carotid, both cerebral perfusions will be equal; the net intracranial shunt will be appreciable, flow from right to left, and arise from the right carotid; and the right carotid will be appreciably larger than the left carotid. The left carotid will appear in the analysis as though it were appreciably stenosed although such is not actually the case.

III. Results:

149 tracings have been obtained from 126 patients and normal subjects. Twelve tracings were technically unsatisfactory and could not be salvaged. All tracings were analyzed by the author.

A. *Population:*

1. *Normal Subjects:* A normal subject is defined as a subject examined at our Institute who showed no evidence of significant disease, either by history or on examination, and who was eligible for routine aviation medical certification. Included in this category is a group of male pilots who were studied as part of a special research project.

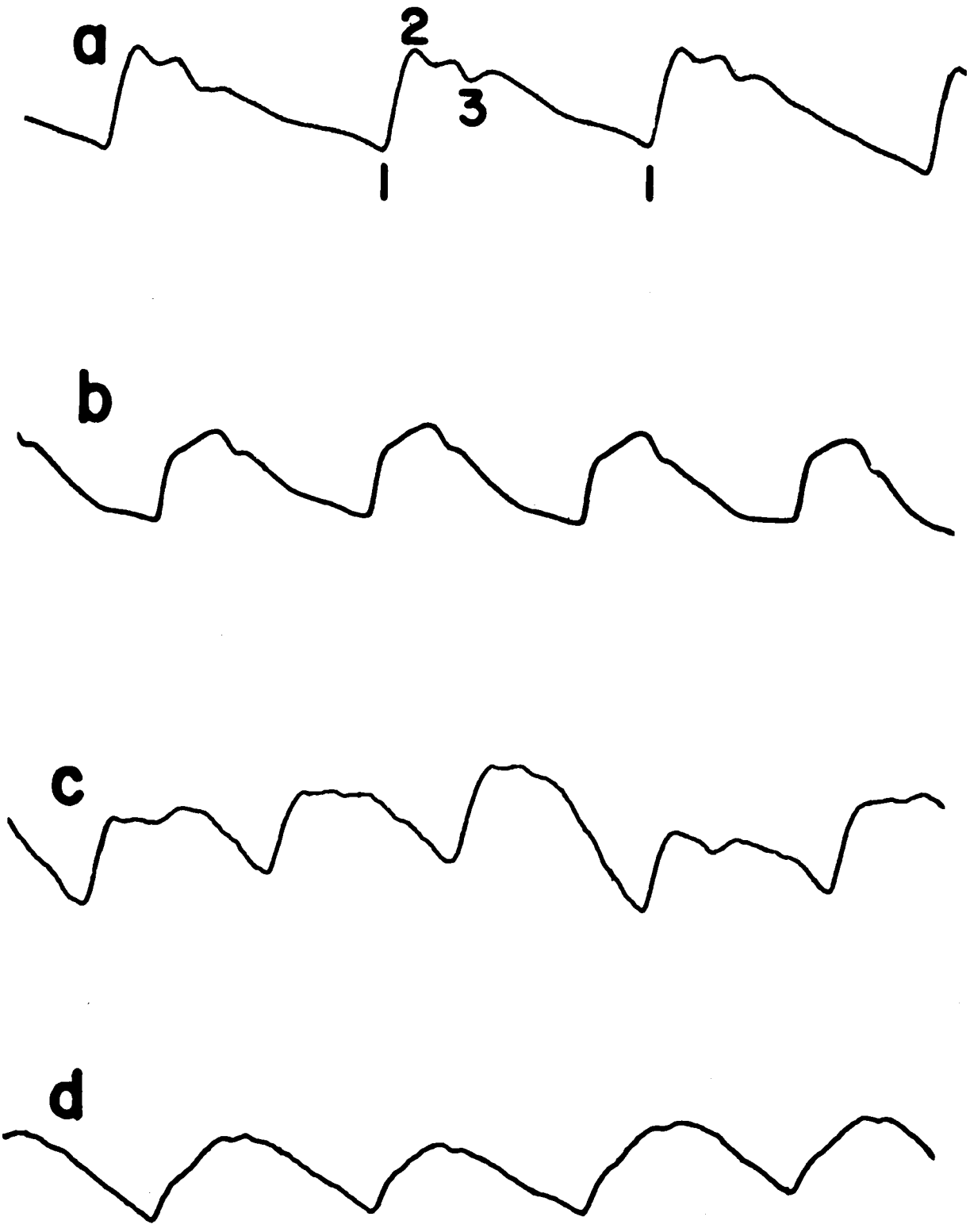

FIGURE 14. Normal and abnormal REG waveforms. (a.) Normal (b.) Delayed inflow (c.) Delayed runoff (d.) Delayed inflow and runoff.

2. *Patients:* The patient group is heterogenous and includes subjects from many sources:

a. Subjects from the Institute's ageing study who showed sufficient cardiovascular or neurological abnormality, by history or examination, to warrant tracing. Most of these subjects were entirely normal except for a slightly less palpable carotid;

b. Subjects referred to the Institute in consultation for evaluation of cardiovascular or neurological disease. Most of these subjects had previously received extensive studies; their diagnoses were known to the tracing interpreter; and

c. Patients referred to the author by local physicians who either wished tracings to assist in their diagnosis or who already had obtained definitive studies and wished to test the interpreter's accuracy. These physicians were aware of the interpreter's interest in developing diagnostic criteria from the tracings alone and therefore carefully withheld clinical information. They were, however, aware of the interpreter's interest in cerebrovascular disease.

Almost all patients were ambulatory and were usually traced as out-patients; neurological disease was not readily apparent on observation. Their tracings were reported without biasing knowledge of the patient's clinical status. Several patients from the interpreter's personal practice were traced prior to definitive diagnostic studies and are included in the series.

B. *Tracing Values:*

1. *Normal Subjects:* Values obtained by measurement of the tracings from 40 normal male subjects are presented in Table I. As our procedure has changed slightly, the data is presented in yearly groupings; the column of reference amplitude ratios is included to simplify later discussion.

Table II presents the results of mathematical analysis of the values from Table I.

2. *Patients:* The patient series is presented in two groups: patients with proved disease whose diagnosis was known by the interpreter prior to the tracing, and patients whose diagnosis was unknown prior to tracing. The latter group is further subdivided into patients who have subsequently come to definitive diagnosis and patients who have received no further studies or on whom no further information is available.

a. *Patients with known disease:* Values obtained by measurement of the tracings from 13 patients who had undergone arteriography prior to referral are presented in Table III; the interpreter was aware of these patients' diagnoses. The tracings from the first two patients were obtained during our initial investigations and were improperly calibrated; the values in ohms are unreliable.

Table IV presents the results of mathematical analysis of the values from Table III.

b. *Patients with subsequently known definitive diagnoses:* We include in this group those patients whose definitive diagnosis was *unknown* to the interpreter until *after* the interpretation. In almost all cases the final diagnoses were determined *after* rheoencephalography.

Table V presents the raw tracing data and Table VI the analytic data.

c. *Patients without definitive studies:* We include in this group all other patients on whom a diagnostic rheoencephalogram was obtained but who have had no further definitive studies or who have been lost to follow-up. Table VII presents the raw tracing data and Table VIII the analytic data.

IV. Discussion:

A. *Diagnostic Accuracy:* If rheoencephalography has any clinical value, diagnostic conclusions derived from its tracings should be in agreement with the disease actually present. Comparison of the data in Table IV with the diagnoses proved in these patients indicates clearly that excellent agreement was attained; even the improperly calibrated tracings gave clear indications of the proved diagnoses. The potential value of rheoencephalography was apparent; an extensive clinical study was therefore undertaken.

As previously stated, patients were referred *without* clinical information. Very early in the study (1962) we set this criterion both to avoid biasing tracing interpretations in favor of obvious clinical diagnoses and to force development of analytic methodology. Since the referring physicians were aware of our belief that rheoencephalography monitored the cerebral circulation, almost all of the patients traced had originally sought medical attention for cerebrovascular insufficiency symptoms and were frequently diagnostic problems. No attempt was made to obtain

TABLE I.
Rheoencephalographic Findings in Normal Male Subjects
Schufried Doppel-Rheograph

No.	Patient No.	Year Traced	Age	Resistance (Ohms)		Capacitance (uf)		Average Reference Amplitudes (Milliohms)		Average Arterial Compression Amplitudes (Milliohms)												Reference Amplitude Ratios (% of larger side)	Peak Timing				Waveforms							
										Right Temporal		Left Temporal		Bilateral Temporal		Right Common Carotid		Left Common Carotid		Bilateral Common Carotids			Reference		Bitemporal		Inflow Delayed				Runoff Delayed			
																											Reference		Bitemporal		Reference		Bitemporal	
				Right	Left	Right	Left	R_1	L_1	R_2	L_2	R_3	L_3	R_4	L_4	R_5	L_5	R_6	L_6	R_7	L_7		Side	Delay (sec)	Side	Delay (sec)	R	L	R	L	R	L	R	L
1.	202	1963	56	110	130	70	70	50	51							23	37	42	25			98L		0			+	+			0	0		
2.	229	"	55	150	140	100	100	137	154							93	142	70	47			89L		0			+	+			+	+		
3.	347	"	50	115	110	100	100	100	99							57	88	62	64			99R		0			0	0			0	0		
4.	668	"	50	130	120	100	100	88	91							40	81	73	46	21	31	97L		0			0	0			0	0		
5.	788	"	58	115	115	100	100	70	70							30	55	54	32	17	19	100	L	.08			0	0			0	0		
6.	1411	1964	51	110	115	250	250	78	85							24	63	58	29			92L		0			0	0			0	0		
7.	405	1965	31	125	120	100	100	96	97	88	97	89	84	81	80	39	64	64	49	11	13	99L		0		0	0	0	+	+	+	+	+	+
8.	706	"	45	120	120	250	250	65	67	51	58	58	56	42	46	35	47	48	34	22	15	97L		0		0	+	+	+	2+	+	+	+	+
9.	855	"	49	120	130	250	250	84	81					73	75	35	62	59	38			96R		0			0	0	0	0	0	0	0	0
10.	1138	"	47	125	125	100	100	69	71	60	71	68	60	59	60	35	48	48	37	15	14	97L	L	.12		0	0	0	0	0	0	0	0	0
11.	1212	"	24	150	160	70	70	80	81	60	74	79	64	59	58	36	52	54	42			99L		0		0	0	0	0	0	0	0	0	0
12.	1703	"	30	135	145	100	100	161	166	132	155	152	128	120	115	67	106	108	68	15	12	97L		0		0	0	0	0	0	0	0	0	0
13.	1713	"	22	140	150	100	100	110	124	99	124	99	94	86	94	45	82	87	57	20	20	89L		0		0	0	0	0	0	0	0	0	0
14.	1715	"	43	120	125	100	100	64	66	46	60	57	44	40	38	30	44	36	25	7	12	97L		0	L	.16	0	0	+	+	0	0	+	+
15.	1717	"	36	120	120	100	100	109	110	85	99	91	78	69	73	48	70	70	42	13	10	99L		0		0	0	0	0	0	0	0	0	0
16.	1719	"	42	140	140	--	--	106	111	85	111	106	87	85	87	60	82	63	38			96L	L	.18	L	.18	0	0	0	0	0	0	0	0
17.	1721	"	33	120	125	100	100	84	80	56	80	83	62	54	50	30	59	60	34	13	13	94R		0		0	0	0	0	0	0	0	0	0
18.	1722	"	42	130	120	70	100	84	79	65	72	70	56	51	48	43	59	58	40	23	27	94R	L	.10	L	.10	0	0	+	+	0	0	0	0
19.	1724	"	41	150	150	70	70	50	52	45	52	44	40	40	40	33	42	34	18			96L	L	.10	L	.10	0	0	0	0	0	+	+	+
20.	1725	"	37	125	125	100	100	83	89	71	87	74	70	60	66	49	60	49	42			93L		0		0	0	0	0	0	0	0	0	0
21.	1726	"	40	110	110	100	100	50	51	43	49	42	39	36	36	20	32	32	18	3	6	98L	L	.13	L	.16	2+	2+	2+	2+	2+	2+	2+	2+
22.	1728	"	38	120	120	100	100	61	63	53	63	61	58	53	55	34	46	42	32	15	13	97L		0		0	0	0	0	0	0	0	0	0
23.	1734	"	41	120	120	100	100	119	125	87	108	110	99	76	80	50	86	82	63	15	24	95L		0		0	0	0	0	0	0	0	0	0
24.	1736	"	36	140	140	100	100	127	128	92	115	115	101	82	86	69	93	98	72	34	36	99L		0		0	0	0	0	0	0	0	0	0
25.	1738	"	47	--	--	--	--	58	67	53	65	57	61	51	57	21	58					87L	L	.09		0	0	+	+	+	0	0	0	0
26.	1740	"	43	115	115	100	100	70	77	63	70	59	62	55	57	43	55	50	45	25	26	91L	L	.13		0	0	+	+	+	0	0	+	+
27.	1742	"	27	120	120	70	70	104	106	82	100	97	83	72	75	44	63	71	51	15	17	98L		0		0	0	0	0	0	0	0	0	0
28.	1743	"	49	115	115	100	100	94	103	86	100	91	92	85	88	62	67	69	64	34	27	91L		0		0	0	0	0	0	0	0	0	0
29.	1744	"	36	105	105	100	100	100	105	78	96	88	75	69	67	55	84	73	47	30	31	95L		0		0	0	0	0	0	0	0	0	0
30.	1745	"	37	125	120	100	100	74	69	60	69	74	58	60	58	26	45	50	26	11	8	93R		0		0	0	0	0	0	0	0	0	0
31.	1748	"	56	125	125	100	100	47	45	42	45	46	40	39	37	23	35	31	23	9	9	96R	L	.10		0	+	+	2+	+	0	0	0	0
32.	1749	"	44	150	150	100	100	107	102	80	94	96	87	73	77	40	72					95R		0		0	2+	2+	+	2+	+	+	+	+
33.	1751	"	42	120	120	100	100	104	105	72	94	99	78	65	68	40	69	74	56	11	24	99L		0		0	+	+	+	2+	+	+	+	+
34.	1752	"	36	120	120	100	100	69	63	49	63	69	49	47	49	28	41	48	25	7	6	91R		0		0	0	0	0	0	0	0	0	0
35.	1754	"	43	125	125	70	70	99	113	80	99	91	99	76	87	53	79	77	71	33	33	92L		0		0	0	0	0	0	0	0	0	0
36.	1758	"	41	100	100	250	250	68	64	51	63	68	47	51	43	26	41	51	36	10	14	94R		0		0	0	0	0	0	+	+	+	+
37.	1759	"	37	110	115	100	100	109	124	84	106	100	99	68	79	51	80	73	64	13	23	88L		0		0	0	0	0	0	0	0	0	0
38.	1761	"	47	115	115	100	100	49	51	39	45	47	41	37	36	25	35	32	21	14	15	96L	L	.11	L	.16	0	+	0	+	0	0	0	0
39.	1762	"	44	125	125	70	70	68	75	49	69	66	56	47	48	33	63	47	40			91L	L	.08	L	.08	0	0	0	+	0	0	0	0
40.	1764	"	43	120	120	100	100	96	88	66	77	90	68	60	58	37	60	66	44	12	15	92R		0		0	0	0	0	0	0	0	0	0
Group Averages:			42	125	125	100	100	86	89	68	83	80	70	62	64	41	64	60	42	17	18	98L												

TABLE II.
Calculated Rheoencephalographic Factors in Normal Male Subjects

Calculated Tracing Components (Milliohms)

No.	Patient No.	Arteries: Temporals R	Temporals L	Temporals Net Shunt To Side	Internal Carotids R	Internal Carotids L	Internal Carotids Net Shunt To Side	Common Carotids R	Common Carotids L	Common Carotids Net Shunt To Side	Posterior-Anterior Shunt: Right Calc.	Right Obs.	Left Calc.	Left Obs.	Cerebral Perfusion: Right Calc.	Right Obs.	Left Calc.	Left Obs.
1.	202							41	34	6L	15		11					
2.	229							56	174	55R	26		35					
3.	347							54	73	27R	19		53					
4.	668							58	60	5R	25	21	36	31				
5.	788							55	54	1R	14	17	17	19				
6.	1411							76	76	2L	4		7					
7.	405	8	20	7R	82	60	8L	90	80	1L	7	11	16	13	81	81	84	80
8.	706	23	18	2L	27	32	1L	50	50	3L	18	22	14	15	44	42	47	46
9.	855	11	6		51	51		62	57	1R	10		19		62	73	69	75
10.	1138	9	12	1R	48	43	3L	57	59	2L	14	15	14	14	59	59	60	60
11.	1212	27	18	6L	46	47	38R	73	65	23L	10		13		59	59	57	58
12.	1703	40	47	2L	116	104	5L	156	151	7L	14	15	8	12	123	120	117	115
13.	1713	11	41	11R	96	49	30L	107	90	19L	22	20	15	20	88	86	94	94
14.	1715	19	29	4R	37	40	2R	56	69	6R	2	7	3	12	41	41	41	39
15.	1717	35	50	7R	66	57	8L	101	107	1L	9	13	13	10	67	69	67	73
16.	1719	21	24	0	54	92	14R	75	116	14R	17		9		85	85	87	87
17.	1721	28	19	1R	47	51	2R	75	70	3R	6	13	13	13	55	54	62	60
18.	1722	26	37	7R	35	28	1L	61	65	6R	17	23	20	27	51	51	49	48
19.	1724	5	18	6R	22	32	0	27	50	6R	17		8		39	40	40	40
20.	1725	14	28	7R	49	53	2L	63	81	5R	15		13		62	60	68	66
21.	1726	9	20	6R	40	31	7L	49	51	1L	2	3	0	6	35	36	38	36
22.	1728	8	5	0	36	45	2R	44	50	2R	15	15	15	13	53	53	58	55
23.	1734	49	35	8L	59	64	6R	108	99	2L	13	15	24	24	78	76	82	80
24.	1736	48	39	1L	45	46	5L	93	85	6L	40	34	37	36	80	82	88	86
25.	1738	7	8	1L	39			46							51	51	57	57
26.	1740	14	26	4R	35	26	6L	49	52	2L	25	25	23	26	54	55	55	57
27.	1742	28	30	1R	75	58	11L	103	88	10L	11	15	8	17	75	72	77	75
28.	1743	11	14	0	57	50	11L	68	64	11L	37	34	28	27	83	85	89	88
29.	1744	31	42	3R	35	43	3R	66	85	6R	28	30	26	31	66	69	66	67
30.	1745	14	11	0	58	56	0	72	67	0	2	11	2	8	60	60	58	58
31.	1748	5	6	1L	29	32	5R	34	38	4R	7	9	13	9	41	39	40	37
32.	1749	35	26	3R	62			97							69	73	79	77
33.	1751	33	32	6L	57	47	0	90	79	6L	10	11	20	24	67	65	67	68
34.	1752	20	14	0	43	45	1L	63	59	1L	7	7	3	6	49	47	49	49
35.	1754	33	22	6L	47	42	6L	80	64	12L	31	33	37	33	72	76	85	87
36.	1758	18	17	1L	47	28	5L	65	45	6L	9	10	13	14	51	51	46	43
37.	1759	43	34	9L	59	62	1R	102	96	8L	15	13	20	23	75	68	81	79
38.	1761	16	12	4L	24	35	5R	40	47	1R	8	14	5	15	37	37	35	36
39.	1762	25	21	4L	22	35	13R	47	56	9R	12		28		47	47	50	48
40.	1764	41	26	5L	46	48	7R	87	74	2R	7	12	16	15	60	60	57	58
Averages:		22	24	0	50	48	0	70	73	1R	14	17	17	18	62	62	65	64

Tracing Component Ratios (Percent)

No.	Temporal Ratios: Larger Temporal	Own Internal Carotid R	Own Internal Carotid L	Own Common Carotid R	Own Common Carotid L	Carotid Ratios: Common	Carotid Ratios: Internal	Cerebral Perfusion Ratios	Cerebral Perfusion Components: Post.-Ant. Shunts R	Post.-Ant. Shunts L	Internal Carotids R	Internal Carotids L	Net Shunt
1.						83R							
2.						32L							
3.						74L							
4.						97L							
5.						98R							
6.						100							
7.	40L	9	33	9	25	89R	73R	95L	9	19	91	71	10L
8.	78R	85	56	46	36	100	84L	94L	41	30	59	68	2L
9.	55R	22	12	18	11	90L	100	92R	16	28	82	72	2R
10.	75L	19	16	28	22	96R	90R	98L	24	23	76	72	5L
11.	67R	59	38	37	28	89R	98L	97R	17	23	78	77	5R
12.	85L	35	45	26	31	97R	90R	95R	11	7	90	89	4L
13.	27L	11	84	10	46	84R	51R	94L	25	16	75	52	32L
14.	66L	51	72	34	42	81L	93L	100	5	7	95	93	5R
15.	70L	53	88	35	47	94L	86R	100	13	3	87	85	12L
16.	88L	39	26	28	21	65L	59L	98L	20	10	64	90	16R
17.	57R	60	37	37	27	93R	92L	89L	11	21	86	79	3R
18.	70L	74	132	43	57	94L	80R	96R	33	41	67	57	2L
19.	28L	23	56	19	36	54L	69L	98L	44	20	56	80	0R
20.	50L	29	53	22	35	78L	93L	91L	24	19	76	78	3L
21.	45L	23	65	18	39	96L	88R	92L	6	0	94	82	18L
22.	63R	23	11	18	10	88L	80L	91L	28	26	68	74	4R
23.	71R	83	55	45	35	92R	92L	95L	17	29	76	71	8R
24.	81R	107	85	52	46	92R	98L	91L	50	42	50	52	6L
25.	88L	18		15				89L					
26.	54L	40	100	29	50	94L	74R	98L	46	42	54	47	11L
27.	93L	37	52	27	34	85R	77R	97L	15	10	85	75	14L
28.	79L	19	28	16	22	94R	88R	93L	45	32	55	56	12L
29.	74L	89	98	47	49	78L	81L	100	42	39	53	61	5R
30.	79R	20	24	16	19	93R	97R	97R	3	3	97	97	0
31.	84L	17	19	15	16	90L	91L	98R	17	33	71	67	12R
32.	74R	57		36				95L					
33.	97R	58	68	37	41	88R	82R	100	15	30	85	70	0
34.	67R	47	31	32	24	94R	96L	100	14	6	86	92	2L
35.	67R	70	52	41	34	80R	89R	85L	43	44	57	49	7L
36.	94R	38	61	28	38	69R	60R	90R	18	28	82	61	11L
37.	79R	73	55	42	35	94R	95L	93L	20	25	79	75	1R
38.	75R	66	34	40	25	85L	69L	95R	22	14	65	86	13R
39.	84R	71	60	53	38	84L	63L	94L	26	56	47	44	27R
40.	64R	89	54	47	35	85R	96L	95R	12	28	77	72	11R
Averages:	95L	47	53	31	33	95L	100	99L	22	23	74	72	2L

TABLE III
Rheoencephalographic Findings in Male Patients with Known Disease

No.	Patient No.	Age	Year Traced	Instru.	Resistance or Impedance (ohms)		Capacitance (nF)		Average Reference Amplitudes (Milliohms)		Average Arterial Compression Amplitudes (Milliohms)						Reference Amplitude Ratios (% of larger side)	Reference Peak Timing		Reference Waveforms			
											Right Common Carotid		Left Common Carotid		Bilateral Common Carotid			Side	Delay (sec)	Inflow Delayed		Runoff Delayed	
					R	L	R	L	R_1	L_1	R_5	L_5	R_6	L_6	R_7	L_7				Right	Left	Right	Left
1.	290*	47	1961	Sch.	Not Recorded				63	91	50	46	37	66	--	--	69L		0	0	0	0	0
2.	583*	51	1962	"	Not Recorded				32	29	12	9	31	28	--	--	91R		0	+	+	+	+
3.	693*	50	"	P.I.	350	350	--	--	78	80	63	45	59	45	--	--	98L		0	+	+	+	+
4.	929	35	"	"	400	370	--	--	173	153	105	139	116	63	--	--	88R		0	0	0	0	0
5.	1097	31	1963	Sch.	180	150	70	70	90	102	80	94	26	12	--	--	88L		0	0	0	0	0
6.	1176	46	"	"	155	150	50	50	90	89	53	75	78	42	--	--	99R		0	+	+	+	+
7.	1333	43	1964	"	115	115	100	100	46	48	24	34	34	20	10	10	98L		0	0	0	0	0
8.	1895	52	"	"	Not Recorded				30	29	21	29	15	8	--	--	97R		0	2+	2+	+	+
9.	1896	73	"	"	135	145	250	250	152	158	83	118	84	48	--	--	96L		0	+	+	0	0
10.	980	52	1963	"	Not Recorded				51	51	14	51	51	22	--	--	100		0	0	0	0	0
11.	"	"	"	"	135	135	100	100	55	57	32	42	43	40	--	--	97L		0	+	+	+	+
12.	"	53	1964	"	140	135	100	100	100	101	41	69	77	41	--	--	99L		0	+	2+	+	+
13.	"	54	1965	"	95	95	250	250	32	33	12	23	21	16	12	8	97L	L	0.11	2+	2+	+	+

*Starred tracings were improperly calibrated; values may be erroneous.

TABLE IV

Calculated Rheoencephalographic Factors in Male Patients with Known Disease

No.	Patient No.	Calculated Tracing Components (Milliohms)							Tracing Component Ratios (Percent)						Clinical and Arteriographic Diagnoses
		Common Carotids			Posterior-Anterior Shunts				Cranial Perfusion Components						
									Posterior-Anterior Shunts		Common Carotids			Common Carotids	
		Right	Left	Net Shunt to Side	Right		Left				Right	Left	Net Shunt to Side		
					Calc.	Obs.	Calc.	Obs.	Right	Left					
1.	290*	58	51	20L	25	--	21	--	40	23	56	56	23L	88R	Congenital kink in left internal carotid; no flow restriction
2.	583*	40	2	19L	11	--	8	--	34	28	66	7	19L	5R	High left internal carotid occlusion
3.	693*	50	53	17L	44	--	10	--	55	13	42	66	21L	94L	Normal right common carotid angiogram with marked basilar filling
4.	929	84	149	41R	48	--	47	--	28	31	49	71	24R	56L	Right common carotid partially occluded at bifurcation; both anterior cerebrals supplied by left carotid
5.	1097	16	154	58R	16	--	4	--	18	4	18	94	65R	10L	Right common carotid ligated below bifurcation for aneurysm
6.	1176	51	59	2L	41	--	28	--	46	32	54	66	2L	86L	Spontaneous subarachnoid hemorrhage Normal arteriograms
7.	1333	36	40	2L	12	10	6	10	26	21	74	83	4L	90L	Left occipital ischemia Normal arteriograms
8.	1895	9	36	15R	6	--	8	--	20	28	30	72	52R	25L	Right internal carotid occlusion sparing external carotid
9.	1896	109	178	28R	15	--	8	--	10	5	72	95	18R	61L	Right common carotid 60% stenotic by arteriography; large left vertebral
10.	980	37	29	0	14	--	22	--	27	43	73	57	0	78R	Normal arteriograms with slightly decreased left carotid
11.	"	38	29	3L	20	--	25	--	36	44	64	51	5L	76R	Ditto
12.	"	91	83	9L	18	--	9	--	18	9	82	82	9L	92R	Ditto
13.	"	31	28	1R	1	12	6	8	3	18	97	82	3R	90R	Ditto

*Starred tracings were improperly calibrated; values may be erroneous.

TABLE V.
Rheoencephalographic Findings in Patients with Subsequently Known Definitive Diagnoses

No.	Patient No.	Age	Year Traced	Instr.	Resistance (Ohms)		Capacitance (nF)		Average Reference Amplitudes (Milliohms)		Average Arterial Compression Amplitudes (Milliohms)											
											Right Temporal		Left Temporal		Bilateral Temporal		Right Common Carotid		Left Common Carotid		Bilateral Common Carotids	
					Right	Left	Right	Left	R_1	L_1	R_2	L_2	R_3	L_3	R_4	L_4	R_5	L_5	R_6	L_6	R_7	L_7
1.	1219*	66	1961	Sch.	90	90	--	--	35	60							20	42				
2.	1886*	37	1962	"	Not Recorded				112	178												
3.	1214	49	"	P.I.	Z = 350 R / 340 L				180	195							28	0	180	195		
4.	1218	62	1963	Sch.	Not Recorded				126	123							77	84	81	56		
5.	1374	51	"	"	150	140	100	100	124	143							42	68	121	82		
6.	1216	52	"	"	150	145	50	50	77	75							48	69	51	33		
7.	1887	60	"	"	95	90	100	100	71	92							71	92	28	32		
8.	1888	49	"	"	105	105	100	100	101	112							37	81	76	57	14	20
9.	1283	53	"	"	135	135	70	70	72	87							40	79	56	44		
10.	"	"	"	"	140	140	50	50	99	109							49	76	70	65		
11.	1286	52	"	"	115	115	100	100	47	47							22	35	34	24	11	14
12.	"	"	"	"	105	105	100	100	53	44							23	31	38	21	9	8
13.	"	"	"	"	115	120	70	70	62	61							30	55	41	27	18	22
14.	1890	55	1964	"	140	135	70	70	50	68							41	68	22	25		
15.	1891	43	"	"	145	140	50	50	83	76							22	32	78	76		
16.	1374	52	"	"	120	115	100	100	98	86							30	34	67	69		
17.	1889	64	"	"	130	125	70	70	68	91							44	66	58	58		
18.	1892	59	"	"	130	130	70	70	71	75							34	65	58	39	19	32
19.	1893	51	"	"	Not Recorded				72	78							29	55	58	36		
20.	1894	49	"	"	145	140	100	100	102	157					74	105	48	81	63	82		
21.	1699	54	1965	"	170	180	70	70	85	94					77	78	29	54	77	78		
22.	1286	55	"	"	130	140	100	100	79	76					62	63	42	60	55	32	18	10
23.	1747	35	"	"	135	145	100	100	142	145	132	142	142	129	128	129	69	101	87	74	40	46
24.	1757	38	"	"	120	130	250	250	34	40	33	40	34	30	33	31	34	40	23	17		
25.	1783	63	"	"	120	105	250	250	82	107	57	106	79	76	60	72	33	93	68	57	26	28
26.	1781	68	"	"	155	150	100	100	202	195	165	191	193	163	150	157	71	131	172	75		
27.	1796	53	"	"	95	95	∞	∞	50	58	43	57	46	47	40	45	26	48	46	30		
28.	1433	56	"	"	130	130	100	100	86	85	80	85	82	78	75	76	45	67	49	27	12	12
29.	"	"	"	"	130	140	100	100	71	83	65	77	71	65	83	59	37	61	48	29	14	12

*Starred tracings were improperly calibrated; values may be erroneous.

TABLE V.--Continued

Rheoencephalographic Findings in Patients with Subsequently Known Definitive Diagnoses

Reference Amplitude Ratios (% of larger side)	Peak Timing				Waveforms								Remarks	No.
					Inflow Delayed				Runoff Delayed					
	Reference		Bitemporal		Reference		Bitemporal		Reference		Bitemporal			
	Side	Delay (sec.)	Side	Delay (sec.)	R	L	R	L	R	L	R	L		
58L	R	0.2			+	0			0	0				1.
63L		0			+	+			+	+			Bedside tracing using portable single-channel EKG	2.
92L	L	0.04			0	0			0	0				3.
98R	R	0.14			+	+			0	0			Possibly unsatisfactory tracing	4.
87L	L	0.11			0	+			0	0				5.
97R		0			0	0			0	0				6.
77L	R	0.10			2+	2+			+	+				7.
90L		0			+	+			+	+				8.
83L	L	0.13			+	+			+	+				9.
91L		0			+	+			0	0				10.
100		0			+	+			+	+				11.
83R		0			+	+			+	+				12.
98R		0			+	+			+	+				13.
73L	R	0.10			2+	2+			+	+				14.
91R		0			2+	2+			0	0				15.
88R		0			0	+			0	0				16.
75L	L	0.20			+	+			+	+				17.
95L		0			2+	2+			0	0				18.
92L		0			2+	2+			+	+				19.
65L		0		0	+	+	+	+	+	+	+	+		20.
90L	L	0.12	L	0.12	0	+	0	0	0	+	0	0		21.
96R	L	0.12	L	0.20	2+	2+	2+	2+	+	+	+	+		22.
98L		0		0	0	0	0	0	0	0	0	0		23.
85L	R	0.18	L	0.21	2+	2+	+	+	+	+	0	0		24.
77L		0		0	+	0	2+	+	0	0	0	0		25.
96R		0		0	+	+	+	+	0	0	0	0		26.
86L		0		0	+	+	+	+	0	0	0	0		27.
99R	L	0.14	L	0.16	+	+	+	+	+	+	+	+		28.
86L	R	0.10	R	0.10	+	2+	+	+	+	2+	+	+		29.

TABLE VI

Calculated Rheoencephalographic Factors in Patients with Subsequently Known Definitive Diagnoses

No.	Patient No.	Calculated Tracing Components (Milliohms)																		Tracing Component Ratios (Percent)				
		Arteries														Cerebral Perfusion				Temporal Ratios				
		Temporals			Internal Carotids			Common Carotids			Posterior-Anterior Shunts									Larger Temporal	Own Internal Carotid		Own Common Carotid	
		R	L	Net Shunt to Side	R	L	Net Shunt to Side	R	L	Net Shunt to Side	Right		Left		Right		Left						
											Calc.	Obs.	Calc.	Obs.	Calc.	Obs.	Calc.	Obs.		R	L	R	L
1.	1219*							33																
2.	1886+																							
3.	1214+							347	0	195L	28		0											
4.	1218							88	112	7R	27		9											
5.	1374+							157	64	72L	39		7											
6.	1216							35	68	20R	22		27											
7.	1887							0	103	43R	28		32											
8.	1888+							95	80	6L	12	14	26	20										
9.	1283							40	59	8R	24		36											
10.	"							83	73	4L	20		32											
11.	1286							37	36	1R	10	11	12	14										
12.	"							43	38	2R	8	9	8	8										
13.	"							39	55	15R	9	18	21	22										
14.	1890+							9	71	28R	13		25											
15.	1891+							105	5	39L	16		39											
16.	1374+							120	48	21L	0		17											
17.	1889+							49	43	15L	34		33											
18.	1892+							47	49	3R	21	19	29	32										
19.	1893+							66	56	9L	15		13											
20.	1894+	28	52		79	10		130	114	37L	9		6			74		105	54L	35	520	22	46
21.	1699+	8	16		88	29		96	45	32L	21		17			77		78	50L	9	55	8	35
22.	1286	19	11	1L	34	57	9R	53	68	8R	18	18	16	10	61	62	64	63	58R	56	19	36	16
23.	1747+	10	16	0	107	110	11R	117	126	11R	14	40	30	46	132	128	129	129	63L	9	15	9	13
24.	1757+	1	10	0	0	24	11R	1	34	11R	23		17		34	33	30	31	10L	∞	42	∞	29
25.	1783	26	31	2R	37	30	2L	63	61	0	19	26	43	28	54	60	75	72	84L	70	41	103	51
26.	1781+	41	41	5R	154	109	39L	195	150	34L	41		11		156	150	159	157	100	27	38	21	27
27.	1796	8	15	3R	26	17	9L	34	32	6L	22		20		39	40	46	45	53L	31	88	24	47
28.	1433	6	11	3R	53	84	15R	59	95	18R	8	12	9	12	76	75	78	76	54L	11	13	10	12
29.	"	12	18	6L	44	59	7R	56	77	1R	14	14	7	12	65	63	59	59	67L	27	20	21	23

*Starred tracings were improperly calibrated; values may be erroneous.
+Diagnosis further confirmed at surgery.

TABLE VI.--Continued

Calculated Rheoencephalographic Factors in Patients with Subsequently Known Definitive Diagnoses

Tracing Component Ratios (Percent)								Reference Amplitude Ratios (% of larger side)	Rheoencephalographic Diagnosis	Definitive Diagnosis	Remarks	No.
Carotid Ratios		Cerebral Perfusion Ratios	Cerebral Perfusion Components									
			Posterior-Anterior Shunts		Internal Carotids							
Common	Internal		R	L	R	L	Net Shunt					
								58L	Partial right carotid occlusion	High right internal carotid occlusion	Arteriography	1.
								63L	Right extravascular space-occupying lesion	Right subdural & subarachnoid hemorrhage	Craniotomy	2.
0R								92L	Left common carotid and vertebral occlusions	Left common carotid occlusion & left subclavian "steal"	Thoracotomy	3.
79L								98R	Slight right common carotid restriction; right cerebral insufficiency	Slight left common carotid insufficiency	Arteriography	4.
41R								87L	Left common carotid and vertebral insufficiency	Left common carotid occlusion; left vertebral stenosis; right carotid stenosis	Arteriography	5.
52L								97R	No signif. intracranial pathology; moderate right carotid stenosis	Slight right cerebral atrophy	Pneumoencephalography	6.
0L								77L	Total occlusion of right common carotid	Total occlusion of right common carotid	1 yr. post-endarterectomy; Thrombosed, pulseless right carotid	7.
84R								90L	No significant disease below circle of Willis	Same (at exploration of left bifurcation)	Aortography showed probable occlusive disease at left bifurcation	8.
68L								83L	Bilateral carotid impairment, more marked on right; funct. impairment of basilar circulation	Bilateral carotid impairment, more marked on right; tortuous vertebrals	Aortography	9.
88R								91L	Normal tracing	Same	2 M on anticoagulation & vasodilators	10.
97R								100	Bilateral carotid occlusive disease	Bilateral internal carotid occlusive disease	Aortography	11.
88R								83R	Bilateral carotid occlusive disease	Same	Aortography	12.
71L								98R	Left carotid has increased markedly	Same	5 weeks on vasodilators and anticoagulation	13.
13L								73L	Right common carotid occluded	Same	Aortography	14.
5R								91R	Left common carotid occluded	Same	Aortography	15.
40R								88R	Left carotid occlusion; improved right carotid circulation	Same	Pulseless left carotid 6M after right carotid endarterectomy. See #5 above	16.
88R								75L	Bilateral carotid occlusive disease, more marked on the left	Same	Aortography	17.
95L								95L	Bilateral carotid occlusive disease; fair vertebral-basilar collateral	Same, plus occluded right vertebral	Aortography	18.
85R								92L	Moderate bilateral carotid occlusive disease, more marked on the left	Diffuse cerebrovascular disease with acute left internal carotid thrombosis 1 yr. later	Aortography	19.
88R	13R	70L	12	6	57	10		65L	Left internal carotid occluded at or above the bifurcation	Left internal carotid 90% stenosed at origin, external carotid spared	Surgery	20.
47R	33R	99L	27	22	73	37		90L	Left internal carotid occluded at or above the bifurcation	Same	Arteriography	21.
78L	60L	94L	30	25	56	75	15R	96R	Right internal carotid stenosis at or above bifurcation, some general improvement	See # 11-13 above		22.
93L	97L	98R	11	23	80	77	8R	98L	Normal tracing ō high amplitudes for age	Left A-V fistula	Arteriography	23.
0L	0L	88L	68	57	0	43	32R	85L	Total occlusion of right common carotid	Same	Right common & internal carotids surgically occluded	24.
97R	81R	72L	35	57	65	40	3L	77L	Normal carotids, ?? space-occupying lesion in posterior circulatory field	Normal carotids, right slightly larger	Carotid arteriography	25.
77R	71R	98L	26	7	74	69	24L	96R	Left common carotid stenosis at or below bifurcation; ?? A-V anomaly	Left common carotid partially occluded at bifurcation	Endarterectomy	26.
94R	69R	85L	56	43	44	37	20L	86L	Bilateral common carotid occlusive disease	Same	Autopsy	27.
62L	63L	97L	11	12	70	88	20R	99R	Significant right common carotid disease	Same	Aortography (1966)	28.
73L	75L	91R	22	12	68	88	11R	86L	Above confirmed	Same		

TABLE VII.
Rheoencephalographic Findings in Patients without Definitive Studies

No.	Patient No.	Sex	Age	Year Traced	Instr.	Resistance or Impedance (ohms)		Capacitance (nF)		Average Reference Amplitudes (Milliohms)		Average Arterial Compression Amplitudes (Milliohms)											
												Right Temporal		Left Temporal		Bilateral Temporal		Right Common Carotid		Left Common Carotid		Bilateral Common Carotid	
						Right	Left	Right	Left	R_1	L_1	R_2	L_2	R_3	L_3	R_4	L_4	R_5	L_5	R_6	L_6	R_7	L_7
1.	305*	M	51	1961	Sch.	Not Recorded				140	64							85	53	87	15		
2.	" *	M	"	"	"	65	65	Not	Rec.	51	46							23	33	29	11		
3.	877*	M	"	"	P.I.	400	360	--	--	203	208							113	138	115	123		
4.	1010	M	42	1963	"	270	265	--	--	216	170							96	111	148	128		
5.	499*	M		"	Sch.	425	365	Not	Rec.	184	156							126	114	86	48		
6.	" *	M		"	"	150	170	50	100	121	97							66	49	66	40		
7.	1206	M	55	"	"	170	150	70	70	88	110							44	94	55	36		
8.	"	M	"	"	"	135	135	100	100	102	80							51	76	92	42		
9.	365	M	49	"	"	140	120	70	70	91	67							42	45	78	37		
10.	1215	F	73	"	"	190	140	50	30	113	95							43	70	83	55		
11.	1897	F	42	"	"	125	150	100	70	91	101							40	87	58	30		
12.	1177	M	45	"	"	150	120	70	70	58	65							18	38	42	28		
13.	1094	M	43	"	"	160	160	70	70	77	58							26	45	54	24		
14.	194	M	51	"	"	150	145	100	100	91	67							29	40	74	30		
15.	1192	M	51	"	"	165	165	100	70	73	67							36	45	48	37		
16.	1898	M	70	"	"	180	170	100	100	71	67							39	60	45	30		
17.	645	M	54	"	"	150	140	70	70	75	75							39	62	51	25		
18.	745	M	52	"	"	160	155	70	70	68	55							39	39	48	22		
19	64	M	27	"	"	140	135	100	100	98	98							40	60	78	42		
20.	1899	M	62	"	"	125	125	100	100	60	77							34	53	57	43		
21.	1900	F	48	"	"	165	155	70	70	102	114							43	69	73	51		
22.	1285	M	26	"	"	135	135	100	100	86	100							35	65	53	38		
23.	1901	M	33	"	"	130	120	100	100	77	83							43	57	50	50		
24.	1322	M	61	"	"	130	125	100	100	86	91							37	71	51	39		
25.	1364	M	48	1964	"	130	130	70	70	75	88							39	65	52	44	21	25
26.	1336	M	58	"	"	135	135	100	70	65	58							36	47	53	35	27	23
27.	1902	F	68	"	"	140	140	70	70	86	107							43	79	46	27		
28.	1385	F	73	"	"	140	140	70	70	81	91							52	84	50	37		
29.	1903	F	34	"	"	140	150	100	100	119	107							--	--	--	--		
30.	1416	M	69	"	"	125	135	250	250	47	41							19	37	31	18		
31.	1904	F	63	"	"	155	145	70	100	67	67							22	40	50	29		
32.	1905	M	55	"	"	130	120	250	250	124	147	101	123	119	123	92	102	61	119	79	50		
33.	1906	F	60	"	"	130	130	100	100	74	94					71	79	51	81	68	67		
34.	1533	M	46	"	"	135	135	100	100	59	73					51	58	39	62	46	37		
35.	1907	F	80	1965	"	140	155	70	70	173	166					143	145	139	158	133	97		
36.	1696	F	54	"	"	145	150	70	70	118	116					98	93	33	70	66	45	31	25
37.	"	F	"	"	"	160	160	70	70	107	98					88	87	37	64	77	43	15	15
38.	1708	F	49	"	"	155	165	70	70	80	92	69	89	79	73	71	74	40	79	63	39	17	20
39.	1710	M	55	"	"	140	150	Not	Rec.	83	93	57	77	80	72	52	58	27	51	76	54		
40.	877	M	53	"	"	140	150	50	70	89	71	65	58	79	62	58	48	50	58	54	24	--	8
41.	1737	M	59	"	"	125	125	100	100	81	83	76	80	81	72	76	73	36	50	45	34		
42.	1908	F	26	"	"	145	150	70	70	68	78	55	78	62	68	53	68	22	58	54	39	10	15
43.	1768	M	57	"	"	105	110	100	250	34	31	33	31	30	25	30	24	27	31	21	10	12	10
44.	1779	M	45	"	"	110	110	250	250	117	118	97	114	108	91	89	88	73	87	90	71	39	37
45.	1801	F	54	"	"	145	145	250	250	97	120	81	113	85	101	72	96	37	90	71	45	13	11
46.	1800	M	74	"	"	110	110	250	250	127	144	88	125	111	94	70	74	62	109	99	53		
47.	1822	M	44	"	"	115	120	250	250	54	69	44	65	50	51	41	48	33	58	43	33	16	24
48.	1841	F	53	"	"	120	120	250	250	142	137	123	131	133	119	113	112	77	114	91	58		
49.	1385	F	52	"	"	120	120	250	250	100	110	94	108	90	99	84	95	76	106	81	77		
50.	1856	F	46	"	"	150	165	250	100	62	79	57	76	55	65	50	64	Not Performed					
51.	1865	F	38	"	"	135	135	250	250	96	86	75	79	84	78	63	70	51	68	51	32		
52.	734	M	44	1963	"	150	140	50	70	63	71							48	65	59	45	23	29
53.	"	M	"	"	"	120	120	100	100	82	88							46	75	58	37	25	23
54.	"	M	46	1965	"	110	110	250	250	82	94	69	86	82	78	67	75	46	70	60	48	20	22

*Starred tracings were improperly calibrated; values may be erroneous.

TABLE VII.--Continued
Rheoencephalographic Findings in Patients without Definitive Studies

Reference Amplitude Ratios (% of larger side)	Peak Timing				Waveforms								Remarks	No.
	Reference		Bitemporal		Inflow Delayed				Runoff Delayed					
					Reference		Bitemporal		Reference		Bitemporal			
	Side	Delay (Sec.)	Side	Delay (Sec.)	Right	Left	Right	Left	Right	Left	Right	Left		
46R		0			+	+			+	+				1.
90R		0			0	0			0	0			Different time constant	2.
92L		0			0	0			+	+				3.
79R		0			0	0			0	0				4.
85R		0			0	0			0	0			Probable instrument failure	5.
53L		0			0	0			0	0			" " "	6.
80L		0			2+	2+			0	0				7.
80R		0			0	0			0	0			4M interval	8.
74R		0			0	0			0	0				9.
84R		0			+	+			0	0				10.
90L		0			+	+			+	+				11.
89L		0			2+	2+			0	+				12.
75R	R	0.18			+	0			0	0				13.
74R		0			+	+			0	0				14.
92R	L	0.20			0	+			0	0				15.
94R		0			+	+			+	+				16.
100	R	.07			2+	2+			+	+				17.
81R	L	.08			+	+			0	0				18.
100		0			0	0			0	0				19.
78L	R	0.15			+	+			0	0				20.
90L		0			+	+			0	0				21.
86L		0			0	0			0	0				22.
93L		0			+	+			0	0				23.
95L		0			2+	2+			0	0				24.
94L		0			0	0			0	0				25.
89R		0			+	+			+	+				26.
80L		0			+	+			0	0				27.
89L	R	0.04			+	+			+	+				28.
90R		0			0	0			0	0				29.
87R		0			0	0			0	0				30.
100		0			+	+			0	0				31.
84L		0		0	+	+	2+	2+	+	+	+	+		32.
79L		0		0	2+	2+	2+	2+	+	+	+	+		33.
81L		0		0	+	+	+	+	+	+	+	+		34.
96R	L	0.12	L	0.12	+	+	+	+	0	0	0	0		35.
98R		0		0	0	0	0	0	0	0	+	+	Very unstable reference	36.
92R		0		0	0	0	0	0	0	0	0	0		37.
87L		0		0	+	2+	2+	2+	+	+	+	+		38.
89L		0	R	0.27	2+	2+	2+	2+	+	+	+	+		39.
80R		0		0	2+	2+	+	+	0	0	0	0		40.
98L		0		0	+	2+	2+	2+	0	0	+	+		41.
87L	L	0.14	L	0.14	+	+	0	+	+	+	+	+		42.
91R		0		0	2+	2+	2+	2+	2+	2+	+	+		43.
99L		0		0	0	0	0	0	0	0	0	0		44.
81L	L	0.06		0	+	+	+	+	+	+	+	+		45.
88L		0		0	+	+	+	+	0	0	0	0		46.
78L	R	0.12	R	0.11	+	+	0	+	+	+	0	+		47.
97R		0		0	2+	2+	2+	2+	+	+	+	+		48.
91L		0		0	+	+	2+	2+	+	+	+	+		49.
79L		0		0	2+	2+	2+	2+	0	0	0	+		50.
90R		0		0	2+	2+	2+	2+	+	+	+	+		51.
89L		0			+	+			0	0				52.
93L		0			+	+			0	0				53.
87L		0		0	+	+	2+	2+	0	0	0	0		54.

TABLE VIII.
Calculated Rheoencephalographic Factors in Patients without Definitive Studies

No.	Patient No.	Calculated Tracing Components (Milliohms)																	Tracing Component Ratios (Percent)				
		Arteries												Cerebral Perfusion					Temporal Ratios				
		Temporals			Internal Carotids			Common Carotids			Posterior-Anterior Shunts								Larger Temporal	Own Internal Carotid		Own Common Carotid	
		R	L	Net Shunt to Side	R	L	Net Shunt to Side	R	L	Net Shunt to Side	Right		Left		Right		Left			R	L	R	L
											Calc.	Obs.	Calc.	Obs.	Calc.	Obs.	Calc.	Obs.					
1.	305*							66	102	42R	22		4										
2.	" *							41	57	9R	1		0										
3.	877*							160	173	18R	25		53										
4.	1010							179	110	9R	28		69										
5.	499*							100	206	56R	28		6										
6.	" *							103	112	7R	11		0										
7.	1206							60	107	17R	11		20										
8.	"							55	48	6R	41	43	38	36									
9.	365							71	43	9L	29		15										
10.	1215							95	70	5R	13		30										
11.	1897							65	104	19R	7		16										
12.	1177							67	53	11L	2		1										
13.	1094							64	54	10R	3		14										
14.	194							89	54	10L	12		3										
15.	1192							59	55	3R	11		15										
16.	1898							39	63	19R	13		23										
17.	645							49	74	11R	15	27	12	20									
18.	745							45	53	4R	19		6										
19.	64							96	76	18L	20		4										
20.	1899							50	37	21L	31		19										
21.	1900							104	92	16L	14		6										
22.	1285							86	95	2L	2		3										
23.	1901							60	60	0	16		24										
24.	1322							69	87	15R	2		19										
25.	1364							59	67	0	16	21	21	25									
26.	1336							40	35	1R	24	27	24	23									
27.	1902							84	119	10R	2		0										
28.	1385							35	85	24R	21		30										
29.	1903																						
30.	1416							32	39	12R	3		14										
31.	1904							72	55	10L	5		2										
32.	1905	47	29	19L	33	102	31R	80	131	12R	24		25		96	92	99	102	62R	142	28	59	22
33.	1906	3	15	---	33	18	---	36	33	7L	45		54			71		79	20L	9	83	8	43

TABLE VIII.--Continued
Calculated Rheoencephalographic Factors in Patients without Definitive Studies

Tracing Component Ratios (Percent)--Continued								Reference Amplitude Ratios (% of larger side)	Rheoencephalographic Diagnosis	Clinical Diagnosis (If Known)	Remarks	No.
Carotid Ratios		Cerebral Perfusion Ratios	Cerebral Perfusion Components				Net Shunt					
			Posterior-Anterior Shunts		Internal Carotids							
Common	Internal		R	L	R	L						
65L								46R	Right carotid insufficiency	Generalized cerebrovascular insufficiency		1.
72L								90R	Right carotid insufficiency c̄ improvement	Generalized cerebrovascular insufficiency	2 M anticoagulation	2.
92L								92L	Moderate cerebrovascular disease	Occipital ischemia	+	3.
61R								79R	Left carotid insufficiency	Seizure, undetermined etiology	+	4.
49L								85R	Right carotid insufficiency	Syncope	+	5.
92L								53L	Normal tracing	Syncope	+	6.
56L								80L	Right carotid insufficiency	Cerebrovascular disease		7.
88L								80R	Bilateral carotid insufficiency	Cerebrovascular disease	4 M anticoagulation	8.
61R								74R	Left carotid insufficiency	Transient cerebral insufficiency	+	9.
74R								84R	Moderate cerebrovascular disease more marked on the left	Basilar insufficiency		10.
62L								90L	Right carotid insufficiency	Same		11.
79R								89L	Normal tracing	Myocardial infarction 1963	+	12.
84R								75R	Slight left carotid insufficiency	Migraine	+	13.
61R								74R	Significant left carotid insufficiency	Generalized severe ASCVD	+	14.
93R								92R	Normal tracing	Migraine	+	15.
62L								94R	Significant right carotid insufficiency	Generalized severe ASCVD, ventricular fibrillation		16.
65L								100	Significant right carotid insufficiency	Seizure, undetermined etiology	+	17.
85L								81R	Moderate cerebrovascular disease	Parkinson's disease		18.
79R								100	Slight left carotid insufficiency with excellent compensation	Normal subject with less pulsatile left carotid	++	19.
74R								78L	Bilateral carotid insufficiency, more marked on left; R/O right space-occupying lesion	Basilar insufficiency		20.
88R								90L	Slight left carotid insufficiency; moderate generalized cerebral ASCVD			21.
90L								86L	Normal tracing	Fainting	+	22.
100								93L	Normal tracing with vasomotor instability	Migraine		23.
79L								95L	Marked generalized cerebral ASCVD; right carotid slightly impaired	Right carotid less palpable; cerebral insufficiency	+	24.
88L								94L	Slight bilateral carotid impairment	Basilar insufficiency	+	25.
88R								89R	Generalized cerebral ASCVD; bilateral carotid impairment more marked on the left	Generalized cerebral ASCVD	+	26.
67L								80L	Occlusive disease above circle of Willis on right	Right middle cerebral insufficiency		27.
42L								89L	Significant right carotid insufficiency	Generalized cerebral ischemia		28.
								90R	Resolving left insufficiency process	Left internal carotid or middle cerebral thrombosis		29.
82L								87R	Bilateral carotid insufficiency, more marked on right	Generalized cerebral ischemia		30.
76R								100	Generalized moderate cerebrovascular disease c̄ slight left carotid impairment	Generalized cerebral ischemia		31.
61L	32L	97L	25	25	34	71	32R	84L	Significant right internal carotid stenosis at or above the bifurcation			32.
92R	55R	90L	63	68	37	23	9L	79L	Bilateral marked carotid stenosis, significantly greater on left	Generalized cerebrovascular disease		33.

TABLE VIII.--Continued
Calculated Rheoencephalographic Factors in Patients without Definitive Studies

No.	Patient No.	Calculated Tracing Components (Milliohms)																Tracing Component Ratios (Percent)				
		Arteries														Cerebral Perfusion				Temporal Ratios				
		Temporals			Internal Carotids			Common Carotids			Posterior-Anterior Shunts									Larger Temporal	Own Internal Carotid		Own Common Carotid	
		R	L	Net Shunt to Side	R	L	Net Shunt to Side	R	L	Net Shunt to Side	Right		Left		Right		Left			R	L	R	L
											Calc.	Obs.	Calc.	Obs.	Calc.	Obs.	Calc.	Obs.					
34.	1533	8	15	---	23	34	---	31	49	2R	26		26			51		58	53L	35	44	26	31
35.	1907	30	21	---	12	88	---	42	109	32R	0		89			143		145	70R	250	24	71	19
36.	1696	20	23	---	114	100	---	134	123	6R	-36	27	23	9		98		93	87L	29	23	15	19
37.	"	19	11	---	84	74	---	103	85	4L	7	15	9	15		88		87	58R	23	15	18	13
38.	1708	14	20	2L	39	50	6R	53	70	4R	23	17	26	20	68	71	70	74	70L	36	40	26	28
39.	1710	42	24	13L	56	22	22L	98	46	35L	20		12		54	52	56	58	57R	109	75	52	43
40.	877	37	19	3L	15	63	25R	52	82	22R	15		11	8	55	58	49	48	51R	247	71	30	23
41.	1737	8	11	3L	70	74	6R	78	85	3R	0		1		76	76	69	73	72L	11	15	10	13
42.	1908	13	16	6R	53	37	12L	66	53	6L	8	10	19	15	49	53	68	68	81L	25	43	20	30
43.	1768	1	10	4R	6	24	9R	7	34	13R	14	12	10	10	29	30	25	24	10L	17	14	42	29
44.	1779	24	36	5R	51	38	9L	75	74	4L	46	39	40	37	88	89	87	88	67L	47	95	32	49
45.	1801	23	31	5R	67	70	9L	90	101	2L	11	13	15	11	69	72	94	96	74L	34	44	26	31
46.	1800	58	66	3L	42	53	4L	100	119	7L	34		18		72	70	75	74	88L	138	124	58	55
47.	1822	14	22	0	18	25	0	32	47	0	22	16	22	24	40	41	47	48	64L	78	88	44	47
48.	1841	25	27	2R	63	103	25R	88	130	27R	26		35		114	113	113	112	93L	40	26	28	21
49.	1385	8	21	8R	20	31	7R	28	52	15R	57		73		84	84	97	95	38L	40	68	29	40
50.	1856	8	21	4R											50	50	62	64	39L				
51.	1865	28	15	5R	35	79	22R	63	94	27R	6		14		63	63	71	70	54R	80	19	44	16
52.	734							21	30	2L	34	23	39	29									
53.	"							49	75	11R	22	25	24	23									
54.	"	21	16	8L	39	52	6R	60	68	2L	24	20	24	22	69	67	70	75	76R	54	31	35	24

*Starred tracings were improperly calibrated; values may be erroneous.
+F.A.A. certification consultation cases.
++F.A.A. normal examinees.

TABLE VIII.--Continued
Calculated Rheoencephalographic Factors in Patients without Definitive Studies

Tracing Component Ratios (Percent)--Continued								Reference Amplitude Ratios (% of larger side)	Rheoencephalographic Diagnosis	Clinical Diagnosis (If Known)	Remarks	
Carotid Ratios		Cerebral Perfusion Ratios	Cerebral Perfusion Components				Net Shunt					
			Posterior-Anterior Shunts		Internal Carotids							
Common	Internal		R	L	R	L						
63L	68L	88L	51	45	45	59	---	81L	Moderate bilateral carotid disease greater on right, c excellent posterior collateral	Generalized cerebrovascular disease and diabetes		34.
38L	14L	99L	0	61	8	61	---	96R	Right internal carotid stenosis at or above bifurcation c marked posterior collateral	Basilar insufficiency	Probably unsatisfactory tracing	35.
92R	88R	95R	X	25	116	108	---	98R	Vasomotor instability, otherwise normal	Hypoglycemic episodes	Fasting tracing	36.
83R	88R	99R	8	11	96	94	---	92R	Normal stable tracing, improved from fasting tracing	Hypoglycemic episodes	30 min. post-prandial	37.
76L	78L	97L	34	37	57	63	9R	87L	Right common carotid decreased slightly compared to left, possibly significant	Chronic brain synd. & alcoholism		38.
47R	39R	96L	37	21	63	39	39L	89L	Significant left common carotid occlusive disease at or below the bifurcation	Left carotid less palpable	+	39.
63L	24L	89R	27	22	27	78	46R	80R	Right internal carotid stenosis of clinical significance	Generalized ASCVD	++	40.
92L	95L	91R	0	1	92	99	8R	98L	No significant carotid occlusive disease; R/O vertebral-basilar disease			41.
80R	70R	72L	16	36	84	54	18L	87L	R/O right space-occupying lesion	Headaches of unknown etiology		42.
21L	25L	86R	48	40	21	60	31R	91R	Significant right common carotid insufficiency			43.
99R	75R	99R	52	46	48	44	10L	99L	Normal tracing with large posterior contributions; excellent collateral potential	Diabetes with hypoglycemic episodes		44.
89L	96L	73L	16	16	84	75	10L	81L	R/O right space-occupying lesion			45.
84L	79L	96L	47	24	53	71	5L	88L	Bilateral carotid occlusive disease more marked on right	Generalized ASCVD		46.
68L	72L	85L	55	47	45	53	0	78L	Bilateral carotid occlusive disease, probably clinically significant on the right			47.
68L	61L	100	23	31	55	69	22R	97R	If right carotid compression values are correct the right internal carotid is significantly stenosed	Possible abdominal A-V fistula; R. carotid bruit		48.
54L	65L	87L	67	75	24	25	8R	91L	Bilateral common carotid occlusive disease more marked on the right			49.
		81L						79L	Decreased right cerebral perfusion	? Intracranial metastatic disease		50.
67L	44L	89L	10	20	56	80	35R	90R	Significant right carotid stenosis			51.
70L								89L	Decreased right common carotid	Right carotid bruit	Normal subject ++	52.
65L								93L	Above confirmed	Right carotid bruit	5 D interval ++	53.
88L	75L	99L	35	34	57	66	9L	87L	Improved right carotid circulation	Bruit still present	24 M interval ++	54.

other types of disease since the author primarily wished to determine if *rheoencephalography was clinically useful* in at least *one* disease and so to stimulate further investigations.

The results of this study are presented in Tables V–VIII. The patients listed in Tables V and VI are, to the best of the author's knowledge, *all* of the patients in this series who have come to definitive diagnosis. The patients in Tables VII and VIII are the remainder who have had no further studies. Some were lost to follow-up, had single certification examinations or consultations, died without autopsy, or are under continuing observation or treatment.

The predictive accuracy of our rheoencephalographic method is clearly demonstrated in Table VI; the 27 (93%) correctly diagnosed tracings require no further comment.

Patient #4 was possibly unsatisfactorily traced; his recording was again analyzed for this report. Re-analysis by our present method gave equivocable findings as before and did not change the final diagnosis. This patient received unilateral left carotid angiography at Georgetown and was later bilaterally studied at a different medical center; to the best of his attending physician's knowledge, he remains a diagnostic problem.

Patient #23 gave suspiciously high amplitudes for his age but clues to the lateralization and diagnosis of his disease could not be obtained. This is the only arteriovenous fistula we have traced and represents a failure of our method.

Tracings #9 and #10 deserve comment. This patient had moderately reduced carotids bilaterally with less filling on the right (as best as can be determined from the rather poor films); there was, however, no evidence of focal occlusive disease. She improved markedly on anticoagulation and vasodilators; anticoagulation was discontinued after four months and all medication after one year without recurrence of symptoms.

Patient #8 is particularly significant when considering the reliability of rheoencephalographic diagnosis. His arteriographic findings suggested marked occlusive disease at the left carotid bifurcation (Figure 15) which was explored.

We quote from the operative note: "A small arteriotomy [at the bifurcation] was carried out which failed to reveal stenotic lesions within the lumen of the vessel. The lumen was adequate." In retrospect, the bifurcation was partially occluded by surrounding tissue when the head was rotated for the lateral films.

The results presented in Table VIII are almost as significant; the diagnostic predictions are quite compatible with the clinical diagnoses given by the attending physicians and consultants. Three otherwise normal subjects who had palpably unequal carotids are included.

The normal subject group presented in Tables I and II may now be reviewed in the light of the above diagnostic accuracy. Six (15%) of these supposed normal males have common or internal carotid arterial contributions which are 65% or less of the corresponding contralateral value; two (5%) have ratios of 51% or less. Asymptomatic at present, these men either have anomalies of the circle of Willis, early disease, or both. They are potential candidates for insufficiency problems if other major arteries later become impaired. Considering the known variability of the cranial vasculature, such findings

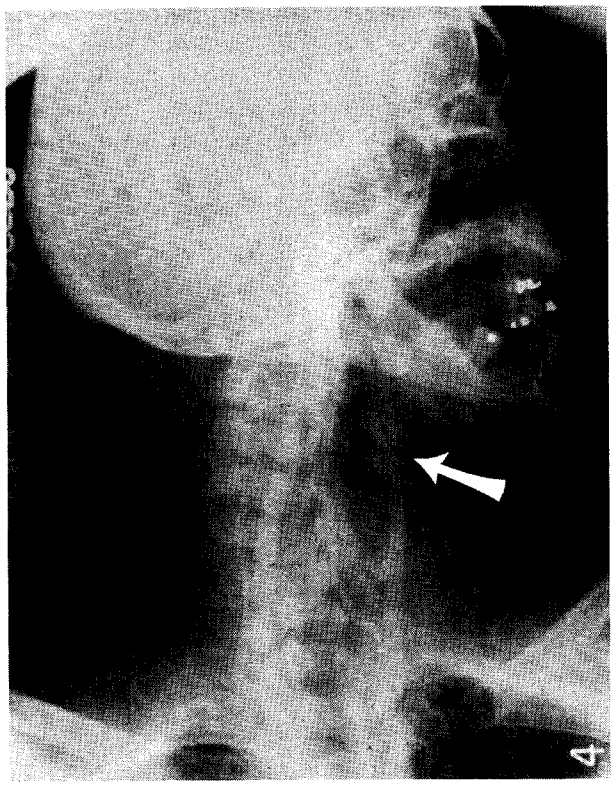

FIGURE 15. Misleading aortography in Patient #8, Tables V and VI.

are not surprising and indicate the potential value of rheoencephalography as a screening procedure.

B. *Criteria for Diagnosis of Carotid Occlusive Disease:* Since this series contains 18 cases with proved and 45 cases with presumptively significant carotid occlusive disease (carotid ratios of 65% or less), a discussion of diagnostic criteria is worthwhile.

Jenkner[1] considers peak timing delays of 0.01 second and longer and differences of amplitude greater than 10% to be significant. While not offering specific criteria, he indicates internal carotid artery occlusions are usually associated with decreased amplitudes and timing delays of 0.10 second or longer on the affected side. Perez-Borja and Meyer[10] and McHenry[11] find no such correlation and note that such criteria frequently give false lateralization to the unaffected side.

1. *Timing Delay:* In this series, 38 tracings showed timing delays of 0.04 seconds or longer on either the reference or bitemporal compression tracings. Five tracings showed no delay during bitemporal compression, while three tracings showed delay only on the latter (see Tables).

The side delayed on the reference tracing was compared with various tracing factor asymmetries, no matter how slight. Random correlation was found with asymmetries in the reference amplitudes, cerebral perfusions, and common carotid, internal carotid, and temporal arterial components. Similarly, *no* correlation was found with delays on the bitemporal compression tracings. Timing delays are, therefore, equally likely to appear on either side and are not reliable indicators of lateralization. Further, at least 47 tracings showed *no* delay despite significant disease; the absence of a delay does *not* reliably indicate an absence of disease. Finally, as might be expected, timing delays correlate well (84%) with abnormal waveforms showing delayed inflow.

Timing delays, either on the reference or bitemporal compression tracings have *no* lateralizing significance and non-specifically indicate the presence of disease.

2. *Reference Amplitude Asymmetry:* Reference amplitude asymmetries of 90% or less were compared to asymmetries in various factors. Agreement with cerebral perfusion asymmetry was excellent in 20 (91%) of 22 tracings. Agreement with temporal artery asymmetries was less satisfactory and was found in 17 (77%) of 22 tracings. Agreement with common carotid asymmetries was poor and was found in 29 (60%) of 48 tracings while agreement with internal carotid asymmetries was equally random in 11 (55%) of 20 tracings.

Reference amplitude asymmetry is an excellent indicator of cerebral perfusion asymmetry, is less reliable in assessing the temporal arteries, and is unreliable in carotid artery asymmetry. There is a similar lack of reliable correlation between cerebral perfusion asymmetries of 95% or less and carotid asymmetries.

3. *Reference Amplitude Asymmetry Plus Timing Delay:* Fifteen tracings combined the two criteria; all tracings showed significant abnormality. Both findings were consistent with lateralization in five tracings (33%); the delay was consistent with lateralization in four tracings (27%); the amplitude asymmetry was consistent in three tracings (20%); and both findings were self-consistent in three tracings (20%) but greater vascular abnormality existed on the opposite side (false lateralization). The two criteria are reliable indicators of disease but not of lateralization.

4. *Cerebral Perfusion Asymmetry Plus Timing Delay:* Thirteen tracings combined the two criteria; all showed significant abnormality. Both findings were consistent with lateralization in four tracings (31%); the delay was consistent with lateralization in three tracings (23%); the amplitude asymmetry was consistent in four tracings (31%); and both criteria were falsely lateralizing in two tracings (15%). The two criteria are reliable indicators of the presence of disease but not of lateralization.

5. *Combination of Criteria:* There is no single criterion or set of criteria which will collect all of the significantly abnormal tracings in this series. A combination of one or more of the following criteria—timing delays in either the reference or bitemporal compression tracings, reference ratios of 90% or less, and cerebral perfusion ratios of 95% or less—loses 16 tracings showing significant disease. Twelve of the latter tracings lack bitemporal compressions and might possibly have been included in the abnormal group. If inflow retardation, either in the reference or bitemporal compression tracings, is added to the above criteria, only the arteriovenous

fistula patient is lost. Waveform retardation alone is as unsatisfactory as are the other single criteria. A combination of all of the above criteria gives information only on the possible presence of disease and gives neither reliable lateralization nor diagnosis. Thus, present information indicates there is no simple and reliable approach to rheoencephalographic diagnosis short of the full method we have presented. The authors must indicate, however, that we have not as yet attempted to correlate specific waveforms with specific diseases. Consideration of the various tables indicates that very few completely normal waveforms are found in the presence of significant disease; such correlation may be possible.

6. *General:* The lack of specific, easily measurable criteria is not unexpected if one remembers that the cerebral circulation is supplied by four relatively independent carotid and vertebral arteries. Significant impairment may be compensated by increases in one or more of the others, often in unexpected patterns and at the expense of other regions. Thus, in Tracing #39, Table II, the right internal carotid is significantly decreased, the left internal carotid is unchanged but supplies the deficit on the opposite side, the left posterior-anterior contribution has increased to compensate for the left perfusion deficit, while the right posterior-anterior contribution has remained essentially normal. In effect, the right cerebral perfusion is supplied at the expense of the left vertebral-basilar system. One is tempted to label this another "steal" syndrome.*

Such phenomena may explain our high incidence of carotid impairment in patients with clinically diagnosed vertebrobasilar insufficiency; such findings strongly suggest that these cases should be screened routinely for carotid occlusive disease since the latter is often amenable to surgery.

C. *Sources and Patterns of Collateral in Carotid Insufficiency:* The tracings showing carotid occlusive disease afford information regarding possible sources of collateral circulation. Sixty-five tracings showing significant insufficiency of one (51%) or both (49%) carotids were reviewed. Sixty tracings (92%) indicated the contralateral carotid had increased and supplied the insufficient side to some degree. In five tracings (8%), the contralateral carotid was not significantly increased; all collateral was supplied at the expense of the posterior circulation.

Of the 60 tracings showing contralateral carotid increase, six (10%) showed collateral supplied only by that carotid, eight (13%) showed collateral additionally supplied only by the ipselateral posterior-anterior shunt, and nine (15%) showed collateral additionally supplied only by the contralateral posterior-anterior shunt. Thirty-seven tracings (62%) showed collateral additionally supplied by both posterior-anterior shunts; in nine tracings (24%), both collateral channels had increased equally; in 18 tracings (49%), the contralateral posterior-anterior shunt had increased more than the ipselateral shunt, while the reverse was true in ten tracings (27%).

Of the five tracings showing only posterior-anterior collateral increases, both channels were equally increased in two cases and both channels were increased with the ipselateral response greater in three cases. *No* patients were traced in whom collateral compensation had not occurred to some degree.

From the above data, the following conclusions can be drawn regarding possible sources and patterns of collateral circulation in unilateral and bilateral carotid insufficiencies. These conclusions are valid to the extent that the tracing analyses reflect the actual circulatory status of the patients and to the extent that this group represents an adequate sampling of the disease.

1. The less involved carotid is an important collateral source in almost all cases (92%), but is the sole source in few (9%);

2. The carotid collateral is assisted by either one of the posterior-anterior shunts on an essentially random basis (12% and 14%);

3. All three less involved sources are increased in the remaining majority (57%). The posterior-anterior shunt *contralateral* to the *lesser* carotid is larger twice as often (28%) as when both shunts are increased equally (14%) or the ipselateral shunt is the larger (15%);

4. In very few cases (8%), the less involved carotid is not increased; the sole collateral source is the posterior circulation.

*We are reminded of a correlation from the evening paper which daily informs us that aims accomplished easily by the wealthy through generosity are accomplished with difficulty by the poor and often through crime, for example theft . . . Of course, here the age correlations are usually reversed!

The high incidence of right carotid involvement is noteworthy and is probably significant ($0.02>p>0.01$). The right carotid is involved unilaterally twice as often (67%) as the left carotid (33%); in bilateral disease, the right carotid is more frequently the site of greater involvement (59%) than the left carotid (41%). As previously noted, our normal series contains 10 men selected at random from our panel of examinees, and 30 men having similar occupations; our patient series includes other Institute normal and consultation examinees and referrals from 14 local physicians. While cerebrovascular insufficiency was frequently suspected, it is difficult to postulate a mechanism capable of biasing so many patient sources toward right carotid disease.

The above data suggest that, as might be anticipated, occlusive disease of the major arteries supplying the circle of Willis is likely to occur simultaneously in all four arteries but progression is usually unequal. The higher incidence of contralateral posterior-anterior collateral increase (rather than the expected ipselateral increase) in single carotid disease may indicate a tendency toward diffuse unilateral progression rather than completely random arterial involvement, at least before generalized involvement becomes severe. Further, the temporal compressions indicate the preferential site of right involvement is below the bifurcation in bilateral carotid disease with major right involvement. On the other hand, when major involvement occurs on the left in bilateral carotid occlusive disease, the preferential site is usually above the bifurcation.

A search of 37 appropriately titled references listed in a recent report on cerebrovascular disease[57] disclosed many papers presenting extensive studies of the incidence of cervicocephalic stenotic and occlusive disease. None of these references report an overall incidence of preferential right carotid involvement as high as in our series. Gurdjian et al[58] find twice as many occlusions at the right bifurcation as at the left but no lateral preference in their total series of carotid studies. Poser et al[59] find the right carotid involved 23% more frequently in their patient series and a ratio similar to ours in their normal series. Their combined series shows 33% more right carotids abnormal than left carotids. Other series show no laterality preference or favor left involvement. For example, Weiner et al[60] find a higher incidence (68%) of complete left carotid occlusion in their 61 cases with complete occlusion of one or both carotids.

As in our series, when one carotid is involved the opposite carotid frequently shows significant disease. Schwartz and Mitchell[61] indicate that, if one carotid is severely affected, 81% of the opposite carotids also will be affected; Gurdjian et al[58] give a 29% incidence of bilateral disease; and Hutchinson and Yates[62] find a 45% incidence of bilateral disease in their overall series and a considerably higher incidence (68%) in their cerebral infarct series.

The incidence of posterior circulatory disease has also been studied. Schwarz and Mitchell[61] find 43% more stenoses in the right vertebral system than in the left, a 23% higher incidence of right rather than left vertebral involvement in right carotid stenosis, and a higher tendency (30%) for right vertebral involvement in left carotid stenosis. In their cases with severe stenosis, there is no preferential vertebral involvement in severe right carotid stenosis but the left vertebral is more frequently involved (64%) than the right in severe left carotid stenosis. Gurdjian et al[58] find equal vertebral arteries in 51% of their vertebral-basilar studies while the right vertebral is larger in 20% and the left is larger in 29%. Poser et al[59] find a slightly increased incidence (15%) of left vertebral involvement in their patient series and a markedly higher incidence (91%) of right involvement in their normal series. Their overall incidence indicates no lateral preference. Hutchinson and Yates[62] and Baker and Iannone[63] also find no lateral preference. In a study of circles of Willis from 350 normal brains, Alpers et al[64] find 52.3% follow the normal or classical pattern while the remainder show anomalies. Stringlike vessels were frequent (27.4%) but 92% of the circles were uninterrupted and potentially patent. In our series, one or both posterior-anterior shunts are increased in 59 patients with no lateral preference (85% on the right, 86% on the left); single posterior-anterior shunts are increased with no lateral preference (14% on the right, 15% on the left); but there is a distinct preference for greater left increase when both posterior-anterior shunts are increased (26% right > left, 26% equal, and 48% left> right). If a lesser or absent increase is an indication of stenosis or occlusion, our findings agree with those of

TABLE IX.
Mean Tracing Amplitudes

Mean Tracing Amplitudes and Standard Deviations (Milliohms)						
Carotid Disease						
	Normal	Unilateral		Bilateral		Combined Group
		Right	Left	Greater Right Involvement	Greater Left Involvement	
R_1	88 ± 17	98 ± 31	96 ± 14	58 ± 19	58 ± 13	81 ± 29
L_1	90 ± 19	103 ± 31	101 ± 33	62 ± 23	60 ± 18	85 ± 29
R_2	74 ± 13	83 ± 23	78 ± 30	51 ± 17	49 ± 16	65 ± 22
L_2	90 ± 16	95 ± 27	101 ± 33	63 ± 22	56 ± 17	78 ± 26
R_3	88 ± 14	97 ± 22	90 ± 13	57 ± 18	52 ± 18	75 ± 25
L_3	78 ± 18	88 ± 24	83 ± 16	53 ± 20	44 ± 14	67 ± 24
R_4	71 ± 13	86 ± 30	72 ± 14	48 ± 20	47 ± 18	64 ± 23
L_4	73 ± 13	88 ± 31	84 ± 20	49 ± 23	48 ± 20	66 ± 24
R_5	38 ± 11	60 ± 24	35 ± 9	35 ± 16	30 ± 12	41 ± 18
L_5	62 ± 14	89 ± 28	54 ± 19	52 ± 20	46 ± 16	63 ± 24
R_6	62 ± 13	59 ± 24	80 ± 17	42 ± 17	47 ± 13	57 ± 20
L_6	43 ± 13	38 ± 17	63 ± 19	30 ± 17	34 ± 15	40 ± 18
R_7	19 ± 9	19 ± 9	20	16 ± 7	15 ± 9	17 ± 8
L_7	20 ± 9	14 ± 8	20	17 ± 8	16 ± 9	18 ± 8
Average age	45	54	47	47	51	48
Number of tracings	36	20	9	19	12	96

Schwarz and Mitchell[61] and Gurdjian et al.[58] We have not as yet analyzed our data considering the known incidence of anomalies of the circle of Willis[63,64]. Such anomalies may in part explain the incidence of apparently significant carotid stenosis in our normal series, as previously discussed (page 48).

D. *Mean Tracing Amplitudes:* The entire Schufried tracing series was reviewed eliminating unsatisfactory or incomplete tracings, cases with proved or suspected disease and cases with common or internal carotid ratios of 75% or less, leaving a remainder of 36 rheoencephalographically normal tracings; similar review of the Schufried tracings in the previously discussed groups leaves 20 tracings of unilateral right carotid disease, nine tracings of unilateral left carotid disease, 19 tracings of bilateral carotid disease more marked on the right, and 12 tracings of bilateral carotid disease more marked on the left. The average amplitudes for each group and the combined group are presented in Table IX. As a variable number of tracings in each group lack the complete series of arterial compressions, the averages are not strictly intercomparable.

TABLE X. Ratios of Average Tracing Components

Tracing Components	*Ratios (Percent of Right or Left Side)*				
	Normal	*Carotid Disease*			
		Unilateral		*Bilateral*	
		Right	*Left*	*Greater Right Involvement*	*Greater Left Involvement*
Temporal Arteries	86%R	144%L	133%R	70%L	169%R
Internal Carotid Arteries	95%R	33%L	33%R	71%L	52%R
Common Carotid Arteries	94%R	50%L	50%R	69%L	88%R
Cerebral Perfusions	105%R	103%L	115%R	94%L	93%R
Posterior-Anterior Shunts	125%R	88%L	89%R	95%L	105%R

Eight tracings (28%) in the unilateral carotid disease group (five right, three left) give amplitudes considerably higher than any encountered in the normal group and raise the group averages and standard deviations; the group reference amplitudes otherwise fall within the normal range and support the previous conclusions regarding the difficulty of diagnosing carotid disease from the reference tracing alone. We have no explanation for the increased amplitudes which do not correlate with youth.

Reference tracing amplitudes in bilateral carotid occlusive disease generally fall well below the normal range and may often be diagnostic; they do not reliably indicate the side of major involvement.

Arterial component ratios calculated from the group averages are presented in Table X.

E. *Temporal Arterial Inversion as an Obscuring Factor in Internal Carotid Occlusive Disease:* The temporal arterial component ratios in unilateral internal carotid occlusive disease suggest that there is a strong tendency for the ipselateral temporal artery (and probably the external carotid artery) to be *larger* than the temporal artery on the *uninvolved* side (Table X). Inversion is also seen in the bilateral disease group having greater left carotid involvement.

The individual tracings were reviewed; 33 tracings include temporal arterial compressions and could be evaluated for temporal arterial inversion. The common carotid is the presumed or actual site of disease in 17 tracings; all 17 tracings show a marked decrease in the ipselateral temporal artery. The internal carotid is the presumed or actual site of disease in 16 tracings; 15 of the ipselateral temporal arteries show marked increases compared to their contralateral arteries, while both arteries are equal in one tracing. Fifteen tracings with bilateral disease greater on the right include temporal arterial compressions; 12 tracings indicate right common rather than internal carotid disease. This group (Table X) is strongly biased by common carotid disease and shows consistent temporal, internal, and common carotid ratios.

This marked tendency for the temporal artery to increase in internal carotid occlusive disease probably is part of the well-established external carotid response;[65] it may also reflect the Horner's syndrome occasionally associated with this disease.[66] This unilaterally increased extracranial circulation may partially explain the difficulty of diagnosing carotid disease from the reference tracings alone. The increased temporal

artery usually supplies appreciable blood to the opposite side; the overall effect is to obscure the evidence of internal carotid disease even though common carotid arterial compressions may have been performed. Fortunately, the inversion effect has not been large enough to hide a significantly diseased carotid completely or to falsely lateralize a diagnosis in our series. Both are possible and may be seen in various tracings. For example, Tracing #39, Tables I and II, gives a relatively normal 84% common carotid ratio which would miss the more significant 63% internal carotid ratio if temporal arterial compressions had not been performed; Tracing #23 in the same tables gives equal and normal common and internal carotid ratios but with a complete laterality reversal between them. Case #20, Tables V and VI, in whom the ipselateral angular and posterior auricular arteries were markedly increased, will be reported separately.

Common carotid arterial compressions are probably sufficient to indicate laterality for further clinical studies in cases of symptomatic cerebrovascular disease or suspected carotid impairment. The complete arterial compression series is recommended for screening purposes, for continued observation of developing disease, or for localization of disease above or below the carotid bifurcation.

F. *Final Remarks:* In conclusion, we again wish to emphasize that our tracing procedures and analytical methods are based on a number of assumptions which may be oversimplifications or which may not be generally applicable. We regard the results reported in this paper as somewhat fortuitous since the referring physicians and the authors were sensitized to and looking for carotid occlusive disease which occurs, of course, in a high percentage of patients with cerebrovascular disease.[59] The significance of these results, in our view, lies less in the successful detection of carotid disease in a preselected high incidence group than it does in other, less obvious demonstrations. Thus, the extracranial and intracranial circulations can be *separated* and studied rheoencephalographically; *both* are important in clinical diagnosis. Contributions from *individual* arteries, whether manually accessible or not, can be determined and are diagnostically useful. Modifications which improve the clinical usefulness of rheoencephalography can be predicted from theory or empirically determined; within its own inherent limitations, the method is flexible and can be tailored for specific applications. Finally and most importantly, we have demonstrated that rheoencephalography *can*, at least in this one disease, accurately lateralize carotid occlusive disease and indicate the major involvement site in reference to the bifurcation. This much maligned technique is worthy of further intensive investigation.

REFERENCES

1. JENKNER, F. L., "Rheoencephalography," C. C Thomas, Springfield, Illinois, 1962.
2. NYBOER, J., "Electrical Impedance Plethysmography," C. C Thomas, Springfield, Illinois, 1959.
3. KEDROV, A. A. and NAUMENKO, A. I., "Problems in the Physiology of Intracranial Blood Circulation and Their Clinical Implications," NASA Technical Translation #F-156.
4. MOSKALENKO, Y. Y. and NAUMENKO, A. L., On the Theory of Electroplethysmography, NASA Technical Translation #F-157.
5. LIFSHITZ, K., Rheoencephalography: I. Review of The Technique, J. Nerv. Ment. Dis., *136*, 388-398 (1963).
6. LIFSHITZ, K., Rheoencephalography: II. Survey of Clinical Applications, ibid, *137*, 285-296 (1963).
7. GEDDES, L. A. and HOFF, H. E., The Measurement of Physiologic Events by Electrical Impedance, Am. J. Med. Elect., *3*, 16-27 (1964).
8. GEDDES, L. A., and HOFF, H. E., The Measurement of Physiological Events by Impedance Change, Proc. San Diego Symp. Biomed. Eng., 115-122 (1963).
9. GEDDES, L. A., et al, Card. Res. Center Bull., II, 112-122 (1964).
10. PEREZ-BORJA, C. and MEYER, J. S., A Critical Evaluation of Rheoencephalography in Control Subjects and in Proven Cases of Cerebrovascular Disease, J. Neurol. Neurosurg, Psychiat., *27*, 66-72 (1964).
11. MCHENRY, L. D., JR., Rheoencephalography: A Clinical Appraisal, Neurology, *15*, 507-517 (1965).
12. NARDI, G .L., and SEIPEL, J. H., The Selective Localization of Alkaloids in Pancreatic Tissue, Surgical Forum, VII, 381-385 (1965).
13. SEIPEL, J. H., and MORROW, R. D., The Magnetic Field Accompanying Neuronal Activity, J. Wash. Acad. Sci., 1-4 (Fall 1960).
14. GLASSTONE, S., "An Introduction to Electrochemistry," D. Van Nostrand Co., New York, N.Y., 1942.
15. ROBINSON, R. A., and STOKES, R. H., "Electrolyte Solutions," Butterworths, London, 1959.
16. SCHWAN, H. P. Determination of Biological Impedances, "Physical Techniques in Biological Research," Volume V, Academic Press, New York, N.Y., 1961.
17. HODGMAN, C. D., "Handbook of Chemistry and Physics," Chemical Rubber Publishing Co., Cleveland, Ohio, 44th edition, 2690-91, (1962-63).
18. SIGMAN, E., et al, Effect of Motion on the Electrical Conductivity of the Blood, Am. J. Physiol, *118*, 708-719 (1937).
19. VELICK, S., and GORIN, M., The Electrical Conductance of Suspensions of Ellipsoids and its Relationship to the study of Avian Erythrocytes, J. Gen. Physiol., *23*, 753-771 (1939-40).
20. COULTER, N. A., and PAPPENHEIMER, J. R., Development of Turbulence in Flowing Blood, Am. J. Physiol., *159*, 401-408 (1949).
21. MOLNER, G. W., NYBOER, JAN, and LEVINE, R. L., "The Effect of Temperature and Flow on the Specific Resistance of Human Venous Blood," Report #127, Army Medical Research Laboratory, 1953.
22. MOSKALENKO, Y. E., and NAUMENKO, A. I., Movement of the Blood and Changes in its Electrical Conductivity, Bull. Exper. Biol. Med. *47*, 211-215 (1959) Translation by Consultants' Bureau, Inc., New York, N.Y.
23. SUGANO, H., and ODA, M., A New Method for Blood Flow Measurement, Jap. J. Pharmacol., *10*, 30-37 (1960).
24. LIEBMAN, F. M., et al, The Electrical Conductance Properties of Blood in Motion, Physics Med. Biol., 7, 177-194 (1963).
25. LIEBMAN, F. M., and COSENZA, F., Study of Blood Flow in the Dental Pulp by an Electrical Impedance Technique, ibid, 7, 167-176 (1962-63).
26. GOLLAN, F., and NAMON, R., Experimental Analysis of the Rheoencephalogram (REG), Proc. Soc. Exper. Biol. Med. *118*, 809-811 (1965).
27. STERN, O., Zur Theorie der Elektrolytischem Doppelschict, Z. Elektrochem., *30*, 508-516 (1924).
28. VERWEY, E. J. W., The Electrical Double Layer and the Stability of Lyophobic Colloids, Chem. Rev., *16*, 363-415 (1935).
29. CRAXFORD, S. R., On the Electrochemistry of Simple Interphases, with Special Reference to That between Mercury and Solutions of Electrolytes, Trans. Far. Soc., *36*, 85-101 (1940).
30. KRUYT, H .R., and OVERBEEK, J. Th. G., Part II.—Theoretical Treatment of the Double Layer and its Implications. Introductory Paper. ibid, *36*, 110-116 (1940).
31. BIKERMAN, J. J., Electrokinetic Equations and Surface Conductance. A Survey of the Diffuse Double Layer Theory of Colloidal Solutions, ibid, *36*, 154-160 (1940).
32. LANDAU, L .D., and LIFSHITZ, E. M., "Electrodynamics of Continuous Media," Pergamon Press, New York, N.Y., 1960.
33. SCHWAN, H. P., Table #279, "Handbook of Biological Data," W. S. Spector, editor, W. B. Saunders Co., Philadelphia, Penna., (1956).
34. CRILE, F. W., HOSMER, H. R. and ROWLAND, A. F., The Electrical Conductivity of Animal Tissues Under Normal and Pathological Conditions, Am. J. Physiol., *60* 59-106 (1922).

35. ZIEMNOWICZ, S. A. R., MCWILLIAMS, J. C., and KUCHARSKI, W. E., Conductivity versus Frequency in Human and Feline Cerebrospinal Fluid, Proc. 17th Annual Conf. Eng. Med. Biol., 108 (1964).
36. FREYGANG, W. H., and LANDAU, W. M., Some Relations Between Resistivity and Electrical Activity in the Cerebral Cortex of the cat, J. Cell. Compar. Physiol., *45*, 377–392 (1955).
37. WEEKS, A. W., and ALEXANDER, L., The Distribution of Electrical Current in the Animal Body: An Experimental Investigation of 60 cycle Alternating Current, J. Indust. Hyg. Toxicol., *21*, 517–525 (1939).
38. GREY WALTER, W., in "Electroencephalography" edited by Hill, D. and Parr, G., Macmillan Co., New York, N.Y., 1963, page 65.
39. VAN HARREVELD, A., and SCHADE, J. P., On the Distribution and Movements of Water and Electrolytes in the Cerebral Cortex, in "Structure and Function of the Cerebral Cortex," Tower, D. B. and Schade, J. P. editors, Elsevier, 1960.
40. SIMONSON, E., in "Cerebral Ischemia," edited by Simonson, E., and McGavack, T. H., C. C Thomas, Springfield, Illinois, 175–179 (1964).
41. SIMONSON, E., Effect of Age on the Changes of Extracranial Circulation During Hypoxia, Circ. Res., IX, 18–22 (1961).
42. DONTAS, A. S., and SIMONSON, E., Carotid Pulses and Peripheral Plethysmograms at Rest, ibid, 450–454 (1961).
43. ZIEMNOWICZ, S. A. R., Techniques of Regional Rheography for Monitoring the Circulation of the Brain and in the Eye, Proc. 16th Annual Conf. Eng. Med. Biol., Baltimore, Md., 96–97 (1963).
44. ZIEMNOWICZ, S. A. R., MCWILLIAMS, J. C., and KUCHARSKI, W. E., Proc. 17th Annual Conf. Eng. Med. Biol., 29 (1964).
45. ZIEMNOWICZ, S. A. R., The Present Status of Standard and Regional Rheoencephalography (REG), in "Cerebral Vascular Diseases" edited by Siekert, R. G., and Whisnant, J. P., Grune & Stratton, New York, 74–80 (1965).
46. ZIEMNOWICZ, S. A. R., Rheographic Regional Method for Evaluation of Cerebral and Ocular Circulation in Cardiac and Cerebrovascular Disease, J. Am. Geriatrics Soc. XIII, 35–43 (1965).
47. BERTA, H., et al, Zur Deutung des Schadelrheogrammes, Z. Neurochir., *15*, 257–266 (1957).
48. LIFSHITZ, K., Rheoencephalography with the Use of Averaging Techniques, Proc. 16th Ann. Conf. Eng. Med. Biol., V, 98–99 (1963).
49. SEIPEL, J. H., ZIEMNOWICZ, S. A. R., and O'DOHERTY, D. S., Cranial Impedance Plethysmography—Rheoencephalography as a Method of Detection of Cerebrovascular Disease in "Cerebral Ischemia," edited by Simonson, E., and McGavack, T. H., C. C Thomas, Springfield, Illinois, 162–180 (1964).
50. JENKNER, F. L., Rheography, Confin. Neurol., *19*, 1–20 (1959).
51. SIMONSON, E., Personal Communication.
52. ZIEMNOWICZ, S. A. R., Personal Communication.
53. JENKNER, F. L., Personal Communication.
54. VAN BUSKIRK, C., Intracerebral Vascular Disease, in "Clinical Neurology," edited by Baker, A. B., Hoeber-Harper, New York, N.Y., 589 and 597 (1962).
55. SEIPEL, J. H., Unpublished Data.
56. MCDONALD, D. A., "Blood Flow in Arteries," The Williams and Wilkins Company, Baltimore, Md., 196 (1960).
57. Survey Report, Joint Council Subcommittee on Cerebrovascular Disease, National Institute Neurological Diseases and Blindness and the National Heart Institute, edited by J. L. O'Leary, July 1, 1965.
58. GURDJIAN, E. S., HARDY, W. G., LINDNER, D. W., and THOMAS, L. M., Four-vessel Angiography: Experience With Three-Hundred Consecutive Cases, Clin. Neurosurg., *10*, 251–274 (1963).
59. POSER, C. M., ZOSA, A. J., GOMEZ, A. J., and HARDIN, H. A., Cervicocephalic Angiography for Cerebrovascular Insufficiency, Acta Neurol. Scand., *40*, 321–336 (1964).
60. WEINER, L. M., BERRY, R. G., and KUNDIN, J., Intracranial Circulation in Carotid Occlusion, Arch. Neurol., *11*, 554–561 (1964).
61. SCHWARTZ, C. J. and MITCHELL, J. R. A., Atheroma of the Carotid and Vertebral Arterial Systems, Brit. Med. J., *2*, 1057–1063 (1961).
62. HUTCHINSON, E. C., and YATES, P. O., Carotico-vertebral Stenosis, Lancet, I, 2–8 (1957).
63. BAKER, A. B., and IANNONE, A., Cerebrovascular Disease. I. The Large Arteries of the Circle of Willis, Neurology, *9*, 321–332 (1959).
64. ALPERS, B, J., BARRY, R. G., and PADDISON, R. M., Anatomical Studies of the Circle of Willis in Normal Brain, Arch. Neurol. Psychiat. *81*, 409–418 (1959).
65. OLIVARIUS, B. DEF., The External Carotid Artery Sign, Acta Neurol. Scand. *41*, 539–50 (1965).
66. O'DOHERTY, D. S., and GREEN, J. B., Diagnostic Value of Horner's Syndrome in Thrombosis of the Carotid Artery, Neurology, *8*, 842–845 (1958).