This study has shown that, *if* the electrical activity of the human heart *cannot* be represented by the action of a single equivalent dipole, a single spatial vector loop by itself cannot possibly contain all the available information. Indeed, if the evidence that diseased hearts are nondipolar is verified, there would be a strong argument for the necessity of some type of precordial scalar leads.

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References and Notes

1. D. Gabor and C. Nelson, *J. Appl. Physics* 25, 413 (1954).
2. This can be shown by deriving the two-dimensional case from the same hypotheses and retaining individual unit vectors. Thus in place of dy we obtain $(\vec{a}_x \cdot \vec{n})\,dl$ and for dx we obtain $(\vec{a}_y \cdot \vec{n})\,dl$ where $\vec{a}_x$ and $\vec{a}_y$ are unit vectors in the x and y direction, respectively, and $\vec{n}$ is a unit vector normal to the path of integration. For a counterclockwise direction of integration, $(\vec{a}_x \cdot \vec{n})$ is positive for positive dy and negative for negative dy. For the same direction, however, $(\vec{a}_y \cdot \vec{n})$ is positive for negative dx and negative for positive dx. Thus, for the published equation to be correct (assuming a counterclockwise direction of integration), every dx should be replaced by a minus dx.
3. E. Frank, *Am. Heart J.* 46, 364 (1953).
4. This study was supported in part by research grant H-339-C from the U.S. Public Health Service.

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Equipotentiality Versus Cortical Localization

In an otherwise excellent paper, Weinstein and Teuber recently presented (*1*), without taking specific notice of it, clear-cut evidence that the crux of a possible scientific *synthesis* of the opposite sides of the "equipotentiality-versus-cortical-localization" controversy resides in the different kinds of intelligence for which one tests.

These authors correctly infer that Lashley's (*2*) position tends to be substantiated by their findings that "injury to any lobe of the brain, in either hemisphere, can interfere with performance on certain nonlanguage tasks." Furthermore, they state that Lashley's view of equipotentiality is opposed, as is Rylander's [that is, impairment is maximal following injury to the frontal regions (*3*)], by their findings that "performance on a standardized test of 'general intelligence,' such as the AGCT [Army General Classification Test], . . . shows little or no change 10 years after penetrating brain wounds unless the entrance wound included the left parietotemporal region."

They fail, however, to note that this latter statement strongly supports Nielsen's (*4*) views about the validity of the cortical localization concept—that is, that the cortical areas on the *major* side of the brain may be differentiated in terms of their language functions. Moreover, their analysis, as shown in Fig. 1, tends to confound the issue by failing to make specific comparison of the aphasics with the nonaphasics in their experimental sample. Had this been done, the picture of what happens when the left parietal lobe is alone injured would have stood out more clearly.

It appears to me that the results obtained by Weinstein and Teuber could have been predicted from Nielsen's position (*4, 5*), yet no reference is made to him in their article. This is an oversight, because the AGCT seems to be for the most part a test of verbal intelligence (*6*) and, therefore, well suited to testing hypotheses in this area. Their results, which I believe are a solid contribution to the field, happily seem to square nicely with my recent statement (*7*) "that, while there are exceptions on both sides, *animal* experimentation continues to support Lashley's theory of mass action and equipotentiality, but the literature dealing with aphasia in *humans* tends more and more to substantiate Nielsen's confirmation of the classic teaching of cerebral localization. The crux of the disagreement, which by the way is so often overlooked by critics of cerebral localization, is just this: cerebral localization in aphasia deals with language, and language is the most important difference between animals and man"

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4 October 1957

In his comment on our paper (*1*), Holmes emphasizes that our results support a fairly definite localization of language skills in the left hemisphere of the brain. His interpretation is based on a point that we have stressed ourselves, namely that the Army General Classification Test is a verbal instrument. Conceivably even slight traces of aphasia, undetected by other means, might be revealed by inferior performance on the test. We fully agree with Holmes' comment with regard to this possible interpretation.

Holmes further regrets that we fail to compare aphasics with nonaphasics. This comparison was deliberately omitted because of the obvious nature of the results: aphasia is an impairment of language, and the AGCT is a verbal test. However, the comparisons desired by Holmes can be reconstructed from the data contained in our Fig. 1 (*1*). The mean and N of each group are given before and after elimination of aphasics. Thus, the mean drop in score of the group of 10 men (including aphasics) with a left parietotemporal lesion was 18.70. The mean drop in score of the same group after elimination of the four aphasics was 11.17. Arithmetic computation reveals the mean loss of these four aphasics to be 25.01. Incidentally, our Fig. 1 was set up specifically to show "what happens when the left parietal (and other) lobes are alone injured"; what happened was a significant drop in AGCT score after left parietotemporal lesions, and the absence of such a drop after lesions in other parts of the brain.

We find it difficult to follow Holmes when he says that our findings confirm Nielsen's views on localization of function in man. In our brief article we could not enter into the complex history of this field, but it seems clear that a preponderant role of the left hemisphere in man (particularly the left parietotemporal region) for language skills has been observed for nearly a century. In this respect, our findings seem to need no emphasis, since they merely reconfirm what is well known; but just for this reason we cannot see how that laterality difference could specifically support Nielsen's views.

Holmes apparently sees the issues of localization in terms of the classical dichotomy of specific and general effects. He considers Lashley's findings in the rat as an instance of general (nonlocalized) change after cortical removal and invokes Nielsen as an exponent of specificity for man—that is, that every lesion which produces any symptoms produces symptoms of a different kind, depending on its location.

The findings from our laboratory have induced us for some time to reject this dichotomy. Our work has shown rather consistently that brain injuries in man tend to produce twofold effects, "specific" (localizable) and "general" (nonfocal) alterations (*2*). Thus, all subgroups of the population we have studied have shown deficits on certain perceptual

tasks (*3*), regardless of whether the injury was in the right or left hemisphere, or in the frontal, parietal, temporal, or occipital lobes. The deficit on the Army General Classification Test turned out to be comparatively focal (*1*), and so were a number of other changes in performance, such as difficulty with route finding (*4*), which was limited to the group with parietal penetration.

The work of Lashley (*5*) and others on subhuman mammals can be similarly interpreted. For certain complex tasks, such as the maze, Lashley found general (nonlocalized) effects of cortical removals in rats. With other tests, in the same animals, he found focal changes such as alterations in brightness habit after occipital removals, and difficulties on a "double platform box" after anterior removals. Thus, specific and general effects coexist after cerebral lesions in man, as well as in subhuman forms; which of these effects appears to predominate depends on the nature of the tests employed. If the range of the tasks is sufficiently extended, one finds specific and general effects in obligatory association.

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1 November 1957

Thermal Decomposition of 8-Quinolinol Chelates of Silver (I)

The reaction of 8-quinolinol (oxine) with silver (I) has been the subject of a number of investigations. Vis (*1*) found that a green crystalline mass corresponding to the formula, $AgC_9H_6NO \cdot C_9H_6NOH$, was obtained from concentrated solutions of the reactants. Fox (*2*), however, obtained a green precipitate which corresponded to the 1:1 chelate, AgC_9H_6NO. From ionic charge considerations, one would expect the 1:1 chelate to be formed since the 8-quinolinol ion forms a five-membered ring containing one ionic and one covalent point of attachment to the metal ion.

The work of Tzinberg (*3*), which was confirmed by Hein and Regler (*4*), revealed that the formula advocated by Vis was correct. Further confirmation came from Block, Bailar, and Pearce (*5*), who were able to prepare two modifications of the chelate, a yellow form and a green one.

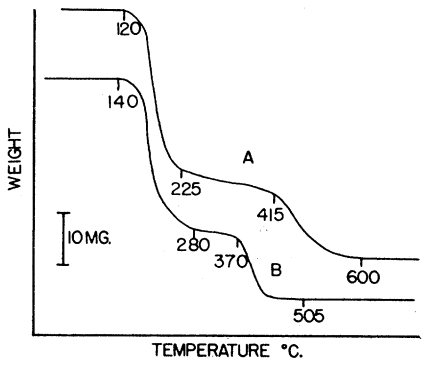

Fig. 1. Thermal decomposition curves of the 8-quinolinol chelates of silver (I). *A*, Green form; *B*, yellow form. Heating rate, 5.4°C per minute.

The nature of the bonding in the metal chelates is still open to question. It was first thought that silver (I) was oxidized to silver (II) and thus would have the normal bonding found in most of the metal 8-quinolinol chelates. If this were the case, the metal chelates would be paramagnetic; however, it was found that they were diamagnetic and hence, that they were silver (I) chelates (*5*). In view of these findings, it was suggested that the silver chelates were addition compounds containing an extra molecule of 8-quinolinol per molecule of silver chelate. This type of chelate, called a lattice chelate because the extra molecule of 8-quinolinol is thought to be held by weak lattice forces, is known for the 8-quinolinol chelates of Sc, Th, U (VI), and Pu (VI).

Now if the silver 8-quinolinol chelate contains solvated 8-quinolinol, it may be possible to prepare the normal 1:1 chelate by thermal decomposition. To find out whether this is possible, the two modifications of the silver 8-quinolinol chelate were prepared as previously described (*5*) and subjected to thermal decomposition on a thermobalance (*6*).

The thermal decomposition curves for the yellow and green forms of the silver 8-quinolinol chelate are given in Fig. 1. The yellow form was the more stable of the two modifications. It was stable up to 140°C when it began slowly to lose weight. The weight loss then became quite rapid, giving a break in the curve at 280°C; however, a constant weight level having the stoichiometry of the 1:1 chelate was not obtained. Beyond 370°C, further rapid weight loss took place to give the metallic silver level beginning at 505°C.

The green form began to lose weight at 120°C, giving a break in the curve at 225°C. Again, a horizontal weight level was not found for the 1:1 chelate. Beyond 415°C, further rapid weight loss took place to give the metallic silver level beginning at 600°C.

The results of these curves reveal that it is not possible to remove thermally the extra solvate molecule of 8-quinolinol without total disruption of the silver chelate. Such behavior is contrary to the thermal decomposition of the 8-quinolinol chelates of thorium and uranium (VI) but similar to that of scandium (*7*).

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14 October 1957

Suggested Revision of Nomenclature—Angiotensin

Concurrent discovery has become commonplace, almost as though a mental sputnik regularly circled the earth, distributing with abandon our most exciting thoughts. The vasoactive peptide resulting from the action of renin on an alpha-globulin was thus discovered by two groups of investigators with the result that the peptide received two trivial names, angiotonin and hypertensin. Synthesis of the octapeptide has now brought a degree of certainty about the identity of this peptide and justifies dropping the double nomenclature. We propose the simplified name, *angiotensin*, and its derivatives *angiotensinase* and *angiotensinogen*. *Angiotensin* is a hybrid word but does, we think, have the advantage of being easy to pronounce even with a variety of accents, and it is euphonious and is understandable despite the most recalcitrant microphone.

There will be many who from habit will want no change, but we hope usage will make the heart grow fonder.

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