THE CEPHALIC POST-TRAUMATIC SYNDROME. PATHOLOGICAL OBSERVATIONS*

By

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I WANT to thank you, Mr. President, and the members of this Society for the privilege of appearing before you.

You are all familiar with the fact that headache, dizziness and mental disturbance occasionally persist for a long time after closed head injuries. These symptoms are usually grouped together under the title, "Post-concussional Syndrome". The question whether or not there is an organic lesion at the root of this syndrome has engaged many minds for many moons. I think the majority of observers would agree with the text which I have taken for this discourse. You will find it in the tenth chapter of the last book of Mayer-Gross, Slater and Roth, page 408. There it is written, "Although predisposition and psychological factors would appear to play a predominant role in its causation, the probability is that some degree of organic change plays its part in most cases."

I want to discuss the site and nature of the organic change postulated in this statement. It is generally taken for granted that if there is an organic lesion it must lie *in* the brain. Denny-Brown (1945) for instance, speaks of "the post-traumatic cerebral syndrome". Guttmann (1946) refers to "hypothetical structural changes" in the brain, while the authors of our text think of some "subtle structural alteration" in the brain. The very word, "post-concussional", implies a cerebral site.

Now there are four facts which suggest that wherever the lesion lies it does not lie *in* the brain: (1) in the majority of cases neurological signs, as distinct from symptoms, are absent, (2) headache, a predominant symptom in this syndrome, is not a symptom of structural damage in the brain; the brain is insensitive, (3) electroencephalography and the most intense neurological investigations have failed to reveal any intrinsic cerebral lesion, and (4) the most significant, the syndrome may occur in patients after a trivial head injury unaccompanied by concussion or suggestion thereof. That is why I've dropped the epithet, "Post-concussional" and adopted the wider one, "Cephalic".

As the search for a purely cerebral lesion has failed, it seems reasonable to consider the possibility of some other intracranial lesion. After all, the brain is not the only thing inside the skull, the only vulnerable occupant. Many years ago I learned that air encephalography and burr-hole exploration yield information about the intracranial state *otherwise unobtainable*, so I used these procedures in cases of the post-traumatic syndrome.

The results of burr-hole exploration are shown in Table I.

A diffuse accumulation of fluid or blood between the skull and the brain is the outstanding feature.

The most common finding was an accumulation of fluid in the subdural

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TABLE I

Subdural effusion	96
Fluid subdural haematoma	4
External hydrocephalus	4
Swollen brain	3
No abnormality found	5
	112

space—a subdural effusion. As this condition is not widely recognized and has not been credited with symptoms of its own, it is necessary to say something

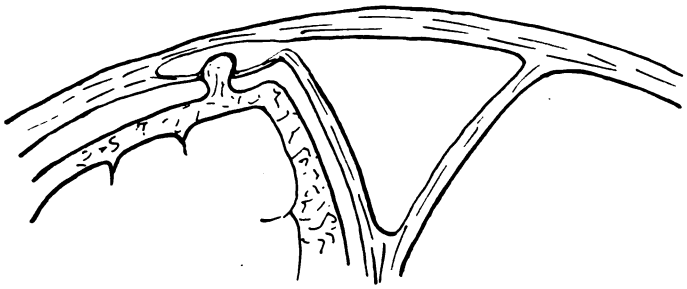


FIG. 1

about it. O'Connell (1953) has given the most satisfying explanation of its origin. Fig. 1 shows diagrammatically the relationships of the subdural and the

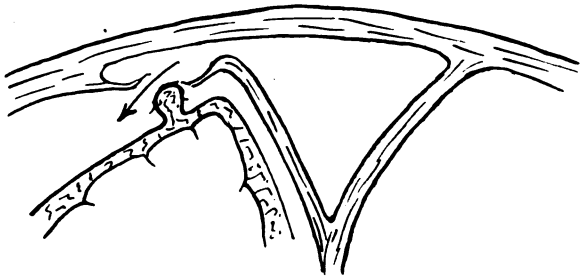


FIG. 2

subarachnoid spaces in the region of the sagittal sinus and the lacunae of that sinus. The arachnoid villi project into the venous channels.

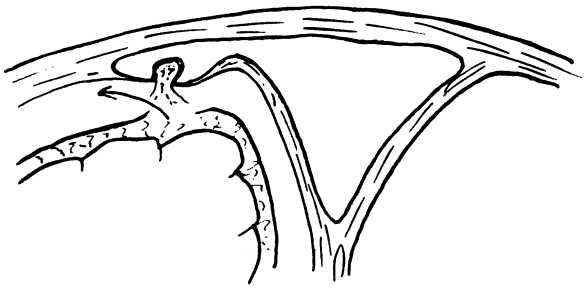


FIG. 3

During any abrupt or forcible movement the dura moves with the skull and the arachnoid moves with the brain. The resulting strain may tear a villus

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out of a lacuna, blood then escapes into the subdural space. The blood clots and the classical subdural haematoma is born.

It is equally possible that the arachnoid villus should be torn at its root



FIG. 4

and a hole left in the arachnoid membrane. This opening allows cerebrospinal fluid to pass into the subdural space—a subdural effusion.

In many cases holes are torn both in the sinus and in the arachnoid at the same time, and the result is a collection of C.S.F. heavily or lightly charged with blood. This is usually described, inaccurately, as a "fluid subdural haematoma" and is much more common than the pure haematoma which clots. Blood in the subdural space, whether pure or mixed with cerebrospinal fluid, indicates an expanding lesion. The pure subdural effusion does not appear to increase in volume with time. It is notorious that these subdural collections of blood, fluid or both can result from trivial injuries.

The hole in the arachnoid membrane which permits the escape of cerebrospinal fluid into the subdural space also permits the passage of air into that space when air encephalography is performed (Fig. 3).

Fig. 4 shows the distribution of air in the subarachnoid spaces and the ventricles immediately after its injection. Fig. 5 taken *twenty-four hours after the injection* shows that the air has passed through the hole and is lying in the

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subdural space where it reveals a fluid level. If this second-day picture is not taken air encephalography is misleading, inconclusive and harmful. The results of air encephalography in 65 cases are set out in Table II.



FIG. 5

TABLE II

Pneumoencephalography

No. of Cases	Normal	Subdural Air
65	13	52

Burr-hole Exploration when Subdural Air Present

Subdural effusion	47
No effusion found	5

That the subdural effusion is not related in any way to air injections is shown by the fact that effusions were found long before encephalography was used. In the present series half the patients with verified effusions had burr-hole exploration without preliminary encephalography.

In Table III the protein content of the subdural effusion is compared with that of the cerebrospinal fluid in six representative cases.

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TABLE III

Protein Content mg. per 100 c.c.

Case	Spinal C.S.F.	Subdural Fluid
1	30	280
2	40	720
3	40	170
4	30	400
5	30	2,200
6	35	5,000

The pathological conditions found in these cases had nothing to do with the patient's pre-traumatic personality, they were found in all sorts of people; in well-adjusted individuals without a care in the world, in the neurotic and in those predisposed to mental illness. Their occurrence had nothing to do with economic, social, or legal embarrassments. Trauma is no respecter of persons.

The mental disturbances in this series of cases so closely resemble those described in every text-book of neurology and psychiatry that it is unnecessary to mention them in detail. In general, each patient reacted to his symptoms according to his personality. The normally integrated individual was often able to carry his additional load, to put up with his disabilities and go on with his work. Sometimes, however, he tended to avoid social contacts and became morose, rude or irritable. In the maladjusted individual the maladjustment was increased; he tended to make the worst of everything and so added to, exaggerated or distorted his symptoms that their real nature was obscured. Anxiety about recovery, employment or compensation, coloured the symptoms in patients of every type.

Removal of the fluid removed the symptoms in 14 out of 21 compensation cases, and in 76 out of 83 of the other cases. In 15 cases fluid collected again and was removed.

CONCLUSION

Our authors postulated "some degree of organic change" as a factor in the causation of the post-traumatic syndrome. Here it is, the subdural effusion.

The simplest way to present the mental symptoms of a *chronic* subdural effusion is to say that they are exactly the same as those of a *chronic* subdural haematoma, with one notable exception; there is no clouding of consciousness. It is well known that a subdural haematoma can produce mental symptoms of any degree; sometimes mild, sometimes suggestive of dementia or cerebral arteriosclerosis. So can a subdural effusion. The haematoma, however, is a progressive lesion with progressive symptoms, however slowly they progress. The effusion is, for all practical purposes, a stationary lesion. If the patient with an effusion were worse he would be better off; if he developed clouding of consciousness someone would open his skull to look for a haematoma. As it is, the symptoms of an effusion are not recognized for what they are. Frustration and hope deferred add their own symptoms and these the psychiatrist tries to treat; he but blows away the smoke and leaves the smouldering fire.

My interpretation of the whole matter is this: the post-traumatic syndrome is compounded of the symptoms of a subdural effusion and the patient's psychological reaction to those symptoms.

The demonstration of a definite pathological condition brings the post-traumatic syndrome into the category of organic syndromes. It becomes an

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integral part of the general neurological tapestry, not a separate piece of embroidery.

In one of his Essays Montaigne remarks, "Nothing can be revealed to him who in some sense knew it not before". The authors of my text use two words which indicate their foreknowledge. The words are, "appear" and "probability". "Although predisposition and psychological factors would *appear* to play a predominant role in its causation, the *probability* is that some degree of organic change plays its part in most cases". Perhaps in the second edition of their book these words will be in italics. I couldn't agree more.

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