as large vessel disease with small vessel embolisation.

The anatomical basis of the lesions causing ataxic-hemiparesis have been previously discussed. It is likely that interruption of pontocerebellar fibres from pontine nuclei plus corticospinal tracts in the basis pontis, is responsible for pontine ataxic-hemiparesis. Capsular ataxic-hemiparesis will cause the syndrome by involving the corticospinal tract as well as the frontopontine fibres in the posterior limb of the internal capsule. Although capsule ataxia hemiparesis seems frequently to be associated with sensory loss, sensory loss is not essential for ataxia to be present, as in Iragui and McCutchen's case, and our case 4. Involvement of cerebellar projection to the cortex appears also possible, and underlie perhaps the ataxic hemiparesis when the lesion is above the internal capsule.

References

The Isolated Occlusion of the Angular Gyri Artery
A Correlative Neurological and Anatomical Study —
Case Report
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SUMMARY We examined a patient who had signs of a cerebral hemisphere lesion: right hemiparesis, facial weakness, right hemianopsia, acustico-mnestic dysphasia, "empty speech," acalculia, visuo-spatial agnosia and constructional apraxia, but without changes in consciousness. Taking into account clinical signs, computed tomography and carotid angiography findings, we concluded that our patient had an infarction zone in the left temporo-parieto-occipital region, as a consequence of the isolated angular gyri artery (ANG) occlusion. Some clinical signs were a direct effect of the ANG's occlusion. Namely, this artery supplies the cortical regions of great functional significance: the planum polare and temporale, the transverse temporal gyri, the superior and middle temporal gyri, the angular and supramarginal gyri, as well as the superior, middle and inferior occipital gyri. But the other symptoms and signs could be explained by the pathophysiological effect of the cerebral edema on regions supplied by the non-occluded branches of the middle cerebral artery.

IN THE COURSE of our clinical work we registered a case of an angular gyri artery occlusion. Numerous authors have studied complete or partial occlusion of the middle cerebral artery. However, the isolated occlusion of its single pial branches has rarely been reported. That is the reason that made us examine this case in detail. We carried out two groups of examinations: clinical and anatomical. We studied and compared the collected results in order to find the corresponding correlation among the clinical, neuro-anatomical and functional facts.

Material and Methods
We used the available neurophysiological, neuro-radiological, neuroophthalmological and laboratory methods of examinations.

Thirty-four forebrain hemispheres were used for anatomical study. The hemispheres were fixed for at least two weeks in 10% formaldehyde solution. The main stem and all pial (cortical) branches of the middle cerebral artery were microdissected with complete angular gyri artery examination.

Case Report
The patient M. B., aged 29, a farmer, was admitted into hospital with acute right hemiparesis, facial weak-
ness, hemihypesthesia, anxiety, spontaneous expressing and naming of objects, without changes in consciousness. The disturbances persisted during 40-days of treatment prior to hospitalization.

The right hemianopsy with a homologous pyramidal deficiency dominated the neurological findings. The jaw jerk was increased. Different types of changes such as acustico-mnestic dysphasia, characteristic "empty speech," acalculia, right-left disorientation, visuo-spatial agnosia and constructional apraxia persisted in the course of the treatment.

The left carotid angiography showed a great avascular region in the temporoparietal area (fig. 1). Computed tomography (CT) confirmed the existence of a massive area of decreased density in the caudal part of the temporal area, along the whole parietal region and, to a certain extent, occipitally on the left-hand side of the brain, with a dilated frontal horn of the lateral ventricle on the same side (fig. 2).

Ophthalmological, EEG and laboratory examinations gave results within the physiological values. Heart examination did not show any disorders though cardiac disease cannot be excluded completely.

The patient was re-examined a few times because the higher cortical functions (speech, writing, reading, calculation) delayed recovery. Using gamma-encephalography, the late static scintigrams showed left temporoparietal hyperactive focus, the intensity of which kept on progressing in the course of control examinations, so, in order to exclude the neoplastic nature of the focus, we rehospitalized the patient, whose motor disturbances had completely disappeared, but the mentioned disturbances of the higher cortical functions still persisted. This time the angiographic findings did not show any relevant change, while gamma-encephalography showed regression of the focus. Taking into account the results achieved by all these methods we concluded that the patient suffered from an infarction of the left temporoparietal and, partially, occipital region. Analysing carotid angiograms in lateral and AP projections, i.e. using the corresponding neuroradiological orientation lines for the middle cerebral artery and its branches, we diagnosed an occlusion of the left angular gyri artery.

**Results of Anatomical Examination**

The angular gyri artery (ANG) is the most caudal and, very often, the strongest branch of the middle cerebral artery (MCA).

There is usually one angular gyri artery (88%), and very rarely (12%) two arteries can be detected. In most cases (71%) this vessel is the strongest branch of the middle cerebral artery. In other cases it is of the same caliber as the temporoooccipital artery (11%), the posterior parietal artery (2%) or as the common stem of the temporoooccipital and the posterior temporal artery 3.5%. Finally, the angular gyri artery is sometimes thinner than the temporoooccipital (7%), the posterior temporal (3.5%) or the posterior parietal artery (2%).

The ANG very often has an independent origin (54%) (fig. 3) and rarely arises by a common stem with the posterior parietal (28%) or the temporoooccipital artery (18%). It usually represents the terminal branch of the main trunk (fig. 3) or of the terminal stems of the insular segment of the MCA.

We divided the stem of the angular gyri artery into

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**Figure 1.** Avascular zone in the region of the angular gyri artery supply.

**Figure 2a, 2b.** Massive area of decreased density in the left temporal and parietal region (arrowhead).
FIGURE 3. The left insuloopercular region, with the insular segment of the middle cerebral artery and its branches. The angular gyri (arrowhead) arises at the caudal part of the long insular gyrus, below the dorsocaudal point of the insula (white point). L — limen insula; I — "presylvia," 2 — "suprasylvia," 3 — "postsylvia"; O — orbital, F — frontal, P — parietal, T — temporal operculum; a and b: the anterior and posterior transverse temporal gyri; CT — the central artery; PA — the anterior parietal artery; TO — the temporooccipital artery.

FIGURE 4. The intrasylvian segment of the angular gyri artery (arrowheads). The same case as on Figure 3. PP — the posterior parietal artery; white point: the dorsocaudal point of the insula; a and b: the transverse temporal gyri; PT — planum temporale.

The intrasylvian segment is located in the Sylvian cistern and it extends from the point of the ANG's origin to the Sylvian fissure. The artery arises almost at the level of the dorsocaudal part of the insula (50%) (fig. 3), rarely at the rostroventral part, respectively, immediately above the limen insula (28%), and the least often in the region of the transverse temporal gyri (22%).

The intrasylvian segment runs between the planum polare of the temporal operculum and the long insular gyrus; it passes in fact through the terminal (caudal) part of the "pseudosylvia." In two thirds of cases the artery then runs around the medial half of the transverse temporal gyri (fig. 4), sometimes forming the neuroradiological "Sylvian point." Finally, the artery runs dorsocaudally, to the surface of the hemisphere, passing over the rostrolateral (64%) or the medial and caudal part (5%) of the planum temporale (fig. 4).

The extrasylvian segment (fig. 5) represents the distal part of the ANG, which extends from the Sylvian fissure to the termination of the vessel. This segment leaves the fissure, as a rule, on the border of the Sylvian fissure and its posterior ascending ramus, or in the bifurcation point of the fissure. The artery then has a horizontal or a slightly ascending or, rarely, a descending course. The arterial stem before bifurcation has the form of an S (42%) or a double S (29%), then a tortuous course (24%) or it is like an arc with a dorsal concavity (5%). The extrasylvian segment traverses the caudal part of the superior temporal gyrus, the ventrocaudal part of the supramarginal gyrus and the angular gyrus. In the region of the angular gyrus (or immediately below it) the artery often gives off two terminal branches (63%) (fig. 5), and rarely three, four or more branches (21%). In other cases (16%) no great branches have been detected. The upper and lower terminal branch have different courses and termination.

Two segments: the "intrasyylvian" and the "extrasylvian."

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The upper branch is often located in the parietooccipital region. It usually arises on the angular gyrus and terminates:
- in the caudal portion of the intraparietal sulcus (25%)
- in the transverse occipital sulcus (30%)
- on the superior and middle occipital gyri (40%)
- on the inferior occipital gyrus (5%)

The lower terminal branch runs below the angular gyrus, over the transitional area between it and the middle temporal gyrus. It extends caudally and ventrally, and terminates:
- in the lunate sulcus (25%)
- on the superior occipital gyrus (5%)
- on the middle or inferior occipital gyri (70%)

It is interesting that the lower branch terminates in 42% of cases one to three centimetres dorsally or rostrocaudally from the occipital pole.

Finally, the temporooccipital or the posterior parietal artery sometimes arise from the angular gyri artery.

Discussion

On the basis of our anatomical examination we concluded that the angular gyri artery has a great and considerably constant supplying region.

The intrasylvian segment of the ANG supplies: the caudal part of the planum polare (area 52); the greatest part of the transverse temporal gyri (area 41); and the rostral part of the planum temporale (area 42).

The extrasylvian segment supplies: the caudal part of the superior and middle temporal gyri (area 22 and 37); the ventrocaudal part of the supramarginal gyrus (40); the angular gyrus (39) and, partially, the superior, middle and inferior occipital gyri (area 19, 18 and...
The result of a volition or motivation deficit after parietal damage. However, the remaining signs are not in accordance with the ischemic region of the ANG’s supply. These signs, at least partially, can be explained by the corresponding pathophysiological mechanisms.

Namely, it has been recorded that the decreased blood flow proximally from the site of occlusion occurs in experimental pial artery embolisation.2 This reduction of the circulation has been recorded in the whole supplying area of the middle cerebral artery (the site of occlusion does not play part in it) not only on the side of the lesion, but also contralaterally. The last possibility that can occur with our patient is the branching off of the posterior parietal artery from the occluded angular gyri artery. This possibility, which we have proven by our anatomical investigation, could be the explanation of a slight extension of the avascular zone in a rostrodorsal direction, i.e. in the rostrodorsal part of the supramarginal gyrus. However, this would be of little influence with respect to the symptoms and signs of parietal damage because for the development of the parietal lobule syndrome the angular gyri artery is responsible almost exclusively.

The perifocal brain edema is certainly the very reason for some clinical signs and symptoms. The postocclusive brain edema reaches the maximal level after 3 to 5 days and persists averagely for two weeks, although in certain cases much longer.4 The edema increases peripheral vascular resistance which, in turn, reduces circulation not only in the edematous area, but in the whole hemisphere also.

On the basis of the above mentioned facts it is clear that parietal cortex damage (especially of the left hemisphere) produces: asomatognosia, finger agnosia, disphasia, alexia, agaphia, acalculia, right-left and extrapersonal space disorientation. These signs are the direct effect of the parietal cortex lesion. But some of them are the result of the corticocortical, interhemispheric, corticothalamic (thalamocortical) and other fiber system damage — the so-called “disconnection syndrome.”

Our patient had most of these signs, but also some others: hemianopsia, hemihypesthesia, hemiparesis and central facial weakness. The hemianopsy in this case is the result not only of optic radiations damage, but also of the parietal cortex lesion and consecutive visual inattention and neglect of the contralateral half-field of space.25 Prolonged hemiparesis could also be the result of a volition or motivation deficit after parietal damage. However, the remaining signs are not in accordance with the ischemic region of the ANG’s supply. These signs, at least partially, can be explained by the corresponding pathophysiological mechanisms.

The temporoparietal zone, i.e. the areas 22, 37, 39 and 40, is the central region of the ANG’s supply. These areas are interconnected, but also send and receive fibers from some other ipsilateral and opposite cortical regions and from the basal ganglia, the thalamus and the brain stem. These areas are interconnected, but also send and receive fibers from some other ipsilateral and opposite cortical regions.

The areas 39 and 40 have a “a command apparatus” for operation of the limbs, hands and eyes within extrapersonal space. Finally, the parietal cortex perhaps subserves the decoding of sign language.29

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Analyzing the afferent and efferent connections, it can be concluded that visual, auditory and sensory modalities converge to the mentioned areas, especially to the inferior parietal lobule. The parietal cortex provides the integration of information of those modalities, and then determines extrapersonal spatial orientation. The areas 39 and 40 have a “a command apparatus” for operation of the limbs, hands and eyes within extrapersonal space. Finally, the parietal cortex perhaps subserves the decoding of sign language.

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