IN 1936, in a monograph by Bergstrand, Olivecrona and Tönnis, it was pointed out that the circulatory disturbance in the neighborhood of arteriovenous aneurysms was the probable cause of the clinical signs associated with these lesions. This supposition in the meantime has been confirmed and extended by the gas-analytical methods of Kety and Schmidt, and serial angiographic examinations.

In such vascular anomalies the so-called peripheral portions of the vessels are not in function because arteriovenous fistulas shunt the blood directly into the venous system. Since there is no peripheral vascular resistance in this part of the system, a speedier circulation results within the malformation, and the carotid-jugular blood flow is enlarged without increase in circulation within the brain itself. According to papers of Shenkin et al., Bessman et al., Bernsmeier and Siemons, Gänshirt and Schiefer, and others, gas analysis has proved that there is a circulatory increase up to three to four times the normal blood flow.

However, the value of such gas analysis based on Fick's principle for measuring blood flow in arteriovenous aneurysms may be subject to critique. The blood passing the aneurysm, i.e. the shunt, loses no \( \text{N}_2 \text{O} \) to cerebral tissue; consequently in the jugular vein there prevails a mixture of blood from normal vessels of the brain (with reduced \( \text{N}_2 \text{O} \) tension) and blood that has passed the arteriovenous aneurysm (with practically unchanged \( \text{N}_2 \text{O} \) content). Measuring the difference in arteriovenous \( \text{N}_2 \text{O} \) is therefore handicapped, according to the size of the malformation. As the difference in concentration is reversely proportional to the amount of blood flow, greater circulation is accompanied by greater inaccuracy in measuring. Kety and Schmidt's gas-analytical results therefore may be applied to the arteriovenous aneurysms only with certain reservations.

Comparison with serial angiographic findings likewise reveals that the circulatory increase concerns the arteriovenous aneurysm but not the remainder of the brain. Even a reduction in circulation in the area surrounding the aneurysm may be found frequently in the angiograms together with hypertrophy of the arteries leading to the malformation. This may be shown most clearly by comparing angiograms before and after total extirpation of the vascular anomaly: circulation is back to normal and vessels not belonging to the aneurysm, which were invisible before the operation, may
now be seen. Hypertrophy of the supplying vessels has vanished within a short time.

Serial angiography permits measuring the time of circulation within the arteriovenous aneurysm as well as in the brain proper (Table 1). Generally this time is substantially shorter in the aneurysm than in the brain. Certain exceptions may be observed in very large arteriovenous aneurysms. If the cerebral circulation is increased to double the normal amount—then the circulatory difference between brain and arteriovenous aneurysm is reduced to less than a second. The greater length of vascular pathway in these large malformations cannot account for this fact entirely. Probably increased turbulence of the blood causes the slowing up of the flow of the contrast medium. Schurr and Wickbom\textsuperscript{18} have published a picture demonstrating such a retardation of the contrast fluid.

In the medium-sized arteriovenous aneurysm (circulatory rates between 105.0 and 62.5 ml./100 g./min.) the difference in the circulatory time between brain and the malformation is 1.8–3.0 seconds. Two patients with obvious lengthening of circulatory time-rate (Table 1) had smaller arteriovenous aneurysms accompanied by large intracerebral hemorrhages, combined with a lowering of cerebral circulation down to subnormal values. This was probably caused by the effect of increased intracranial pressure on vascular portions with low blood-pressure rates, i.e. capillaries and veins. As there is no equivalent to capillaries in the arteriovenous aneurysm and intravenous pressure is increased, an influence of higher intracranial pressure

\begin{table}
\centering
\caption{Circulation-time and rate of brain circulation in 17 cases of arteriovenous aneurysm}
\begin{tabular}{|l|c|c|c|}
\hline
Location of Arteriovenous Aneurysm & Circulation-Time (Seconds) & Cerebral Circulation ml./100 g./min. N\textsubscript{2}O Gas Analysis \\
& (Serial Angiography) & Brain & Arteriovenous Aneurysm \\
\hline
Parietal, lt. & 4.8 & 4.9 & 239.0 \\
Temporoparietal, rt. & 5.8 & 4.0 & 180.5 \\
Temporal, lt. & — & 4.0 & 180.0 \\
Frontomedial, rt. & 4.7 & 4.0 & 163.0 \\
Central, rt. & 5.8 & 4.7 & 145.0 \\
Parietal, rt. & 5.1 & 4.3 & 136.0 \\
Frontoparietal, lt. & 6.7 & 4.0 & 105.0 \\
Parietal, lt. & 5.8 & 4.0 & 96.0 \\
Precentral, rt. & 5.8 & 4.0 & 90.3 \\
Occipital, lt. & 5.8 & 4.0 & 90.0 \\
Occipital, rt. & 6.8 & 4.1 & 88.1 \\
Temporal, rt. & 6.8 & 3.8 & 83.0 \\
Side ventricle, rt. & 6.8 & 4.1 & 84.0 \\
Occipital, lt. & 5.8 & 4.0 & 72.8 \\
Occipital, lt. & 5.6 & 3.0 & 62.5 \\
Frontomedial, lt. & 8.6 & 4.9 & 42.9 \\
Occipital, rt. & 12.0 & 7.0 & 37.0 \\
\hline
\end{tabular}
\end{table}