Chiari H. Über das Verhalten des Teilungswinkels der Carotis communis bei der Endarteriitis chronica deformans. Verh Dtsch Path Ges 1905; (9) : 326–332.

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Concerning the behavior of the bifurcation of the arteria carotis communis in endarteriitis chronica deformans

It has long been known that the carotid arteries are particularly susceptible to endarteriitis chronica deformans.
Richard Thoma of Heidelberg and his followers were particularly concerned with the study of the localization of endarteriitis chronica deformans, mentioning specifically that in terms of the frequency of occurrence of arteriosclerosis nodosa, the carotis interna ranks second, the carotis communis fourth, and the carotis externa only fourteenth (Mehnert[1]). In Mehnert's study, only the aorta abdominalis ranks above the carotis interna, while the aorta thoracica descendens occupies third place in front of the carotis communis. Thus, according to Mehnert, endarteriitis chronica deformans is especially often localized in the area of the carotid arteries, with the exception of the arteria carotis externa. Not only the frequency of the endarteriitis chronica deformans in the area of the carotid arteries is exceptional, but also its intensity.
Based on my own experience I can, in general, confirm Mehnert's findings. But in the following I will focus on two special points concerning the behavior of the bifurcation angle of the arteria carotis communis in endarteriitis chronica deformans – points that to my knowledge have not been sufficiently considered up to this time. The first is the relative frequency of thrombosis resulting from endarteriitis chronica deformans in the area of the bifurcation of the arteria carotis communis and at the beginning of the arteria carotis interna, quite often the source of embolisms in the brain arteries. The second point is the often very early and also isolated existence of endarteriitis chronica deformans in the bifurcation of the arteria carotis communis and at the beginning of the arteria carotis interna.
The impetus for examining the first point was provided by an autopsy performed on February 14, 1905. The case in question was a 45-year-old woman, a patient in the department of Dr. Alfred Pribram, who had been diagnosed with "hemiplegia d. ex haemorrhagia cerebriante dies II." In the first hemisphere of the cerebrum there was fresh mollification in the posterior third of the lobus frontalis, in the whole lobus parietalis, in the nucleus lenticularis and in the capsula interna. This was caused by embolic obstruction of the centimeter-long

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[1] Mehnert, "Concerning the topographic distribution of angiosclerosis, along with articles on the normal structure of the branches of the aortic arch and some venous stems." Doctoral dissertation at the Institute of Pathology of the Royal University of Dorpat, 1888.

distal end-piece of the arteria carotis interna sin., the centimeter-long beginning of the arteria fossae sylvii sin. and some smaller cortical branches of the same, although these vessels and the remaining brain arteries displayed absolutely no signs of thickening or hardening. The search for the source of embolism initially yielded no results. The heart was normal except for minimal spotted thickening of the valvula mitralis and the valvula aortalis. Only in the ascending part and in the arcus did the aortic valve display isolated and small – up to the size of a lentil - sections of thickening in the intima. Otherwise the vessel showed no signs of thickening or hardening. The branches of the arcus, which, as is customary in autopsies, were cut open three to four centimeters wide, showed a similar behavior. The arteries of the extremities were normal in thickness and hardness. I then suspected a paradox embolism (tooth), because the foramen ovale was patent in a diagonal course, but just large enough to accommodate a common anatomic sound. However, no thrombosis was detected anywhere in the venous system of the body, so I had to give up this idea as well. I subsequently began dissecting the arteria carotis communis sin. with its branches in detail, and I very quickly discovered the source of the embolism in this area: a parietal thrombosis. It was attached to the left wall of the centimeter-long end-piece of the arteria carotis communis and reached one and a half centimeters into the arteria carotis interna. The surface of the thrombus was rough and ragged, as if it had been torn - apparently the result of the detachment of one of its parts. The removal of the thrombus revealed quite severe endarteriitis chronica deformans with partially atheromathous decay in the bifurcation of the arteria carotis communis and at the beginning of the arteria carotis interna. Endarteriitis chronica deformans was as strongly developed on the corresponding location of the right side, but the thrombosis was missing. The arteria carotis interna, as it continued towards the skull base, was normal on both sides, as was the arteria carotis externa. Based on these findings the cause of brain embolism was clear. It developed from endarteriitis chronica deformans, in this case at the bifurcation of the arteria carotis communis sin. and at the beginning of the arteria carotis interna sin. In these places on the right side it was unexpectedly strongly developed and, as was mentioned above, on the left side it was combined with a parietal thrombosis.

From that time until mid-July of this year *[1905]* I dissected in greater detailed the aortic system in approximately 400 subjects with special regard to the carotids and their branches, with the results corresponding to those mentioned above.
With regard to the thrombosis in the bifurcation of the arteria carotis communis and at the beginning of the arteria carotis interna, I found it in seven subjects: a 41-year-old woman, a 67-year-old man, two 70-year-old women, a 71-year-old man, a 79-year-old woman and an 81-year-old woman. One time (in the 41-year-old woman) it was found on both sides, three times (in the 70-year-old women and the 79-year-old woman) on the right side and three times (in the 67-

year-old man, the 71-year-old man and the 81-year-old woman) on the left side. With the exception of one single case (the 67-year-old man), where the thrombus obturated the lumen for almost 3 centimeters at the beginning of the arteria carotis interna sin. and in the upper end of the arteria carotis communis sin., the thrombosis was always only parietal and generally displaying minimal expansion, with the size of the thrombus varying between that of a kernel of millet and that of a pea.

One time (in the 81-year-old woman) the parietal thrombosis surrounded the whole circumference of the ostium of the arteria carotis interna sin. A thrombus with the size of a kernel of hemp was found only once (in the 41-year-old woman) in the ostium of the arteria carotis externa. Otherwise the thrombosis was always found only in the ostium of the arteria carotis interna: three times on the medial edge of this ostium and four times on its lateral edge or on the lateral wall of the upper end of the arteria carotis communis. Corresponding to the location of the thrombi, the bifurcation of the arteria carotis communis showed stronger endarteriitis chronica deformans, with its pillow-shaped thickening of the intima, the bulge of the vascular wall and occasionally (in one of the 70-year-old women and in the 79-year-old woman) with areas of atheromatous decay, where thrombi were attached. In all seven cases the rest of the aortic system was also affected by endarteriitis chronica deformans. But in four cases this disease was predominant in the bifurcation of the arteria carotis communis. This was the case with the 41-year-old woman, where the intima of the arteria thoracica ascendens and of stems of branches of the arcus showed only minimal spotted thickening. Only the ostium of the arteria subclavia dex. showed stronger affection comparable to the changes in the arteria carotis communis. This was also the case with the 67-year-old man, where the intima of the aorta and of stems of branches of the arcus showed minimal spotted thickening. In one of the 70-year-old women there was moderate focal thickening of the intima in the aorta and the arteriae iliacae communes, a parietal thrombosis two and half square centimeters in diameter in the aorta abdominalis on the posterior wall three centimeters above the bifurcation, and a similar thrombosis a half centimeter in diameter on the posterior wall of the arteria iliaca communis sin. Furthermore, there was fairly strong endarteriitis chronica deformans in the stems of the branches of the arcus, the brain arteries and the arteria subclavia, and there was a thrombosis on the inferior wall at the beginning of the arteria subclavia sin. Finally, in the case of the 81-year-old woman there was minimal spotted thickening in the arcus as well as calcification of the intima in the aorta. In three other cases the intensity of endarteriitis chronica deformans in the bifurcation of the arteria carotis communis decreased or was at least not predominant compared to other parts of the aortic system. Thus, in the case of the second 70-year-old woman, the aorta, which carried a parietal thrombus in the arcus at the insertion point of the ligamentum botalli, the arteriae iliacae communes and the brain arteries were strongly affected. In the 71-year-old man and the 79-year-old woman the aorta showed strong thickening of the intima

with adiposis and calcification. In the 71-year-old man there was a parietal thrombosis on the inferior wall of the arcus, on the posterior wall of the aorta thoracica descendens and on two locations of the posterior wall of the aorta abdominalis.

In four of these seven cases, thrombosis in the bifurcation of the arteria carotis communis was the cause of embolism to the brain arteries with consequent encephalomalacia, namely in the 67-year-old man, the 70-year-old woman, the 71-year-old man and the 79-year-old woman. The embolism affected naturally the ramification area of the arteria carotis interna and of the arteria fossae sylvii on the corresponding side, causing partially cortical and partially nuclear malacia. The encephalomalacia was consistently relatively new, and the origin of embolism could sometimes be recognized by the rough, ragged areas on the surface of the thrombi in the bifurcation of the arteria carotis communis. These previously mentioned cases should not, of course, diminish the importance of other causes of encephalomalacia. In the mentioned time period, I myself had the opportunity to investigate encephalomalacia due to other causes in eleven cases. In one of these cases there was a local thrombosis in heavily sclerotic brain arteries. In four cases embolism was caused by endocarditis of the mitral valve. In five cases it was caused by endaortitis chronica deformans in the aorta ascendens with local atheromatous decay and parietal thrombosis. In one case it was caused by mesaortitis productiva. I simply want to point out that the source of embolic encephalomalacia can also be a thrombosis in the bifurcation of the arteria carotis communis, even if the rest of the aortic system shows only minimal pathologic changes. It is therefore recommended that the bifurcation of the arteria carotis communis be more thoroughly inspected in cases of this type.

A second important observation, in my opinion, is the often very early and isolated occurrence of endarteriitis chronica deformans in the bifurcation of the arteria carotis communis and at the beginning of the arteria carotis interna. The 400 autopsies I conducted were – apart from 47 newborns and 15 children under the age of one – of individuals 12 years old and above. I found four significant results, namely in a 12-year-old boy, an 18-year-old man, a 23-year-old woman and a 25-year-old woman. All of them died of widespread chronic tuberculosis. In general the aortic system showed no signs of thickening or hardening. However, there was spotted thickening of the intima – not severe but quite pronounced – in the bifurcation of both arteriae carotides communes, and continuing to the first part of the arteriae carotides internae. It was also present, in a less-developed form, in the ostium of the arteria subclavia dex.

Examined microscopically, the intima revealed a hill-like proliferation of connective tissue with a fairly large amount of round cells and, in some places, fatty degeneration. In the media there were no pathologic changes. In the adventitia there was rather severe microcellular infiltration around several vasa vasorums, a clear indication of endarteriitis chronica deformans. Without this special dissection process, this arterial disease would certainly not have been discovered. These four cases show that endarteriitis chronica deformans can,

indeed, occur very early and completely isolated in the bifurcation of the arteria carotis communis, at the beginning of the arteria carotis interna and, in a less severe form, in the ostium of the arteria subclavia dex.
Particularly in the bifurcation of the arteria carotis communis and at the beginning of the arteria carotis interna, I found that the course of the disease was quite often at a very severe, advanced stage compared to the aortic system affected by endarteriitis chronica deformans. Thus, it is my opinion that on the frequency and intensity scale of endarteriitis chronica deformans, the bifurcation of the arteria carotis communis and the beginning of the arteria carotis interna should be ranked at least equal to the aorta abdominalis and the aorta thoracica. As I have already indicated, this behavior in the mentioned location of the aortic system in endarteriitis chronica deformans is clinically significant due to the relative frequency of parietal thrombosis in this location and its consequences.

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