Calcification of the Carotid Siphon

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Calcification of the internal carotid artery in the region of the cavernous sinus is an extremely common radiologic finding. Although it is the only readily available laboratory criterion of cerebrovascular disease its significance has never been determined by combined clinical, radiologic, and pathologic studies. Whether the arterial changes in this region, which we shall refer to as the siphon (after Moniz), correlate with atherosclerosis locally or of the cerebral arteries or of arteries elsewhere in the body is unknown. Despite the localization of disease within the siphon, occlusive carotid thrombosis is relatively uncommon at this level, originating as a rule in the region of the carotid sinus. Also, bruits appear to arise here infrequently even though stenosis may be demonstrated by carotid angiography. To investigate these matters, a portion of the pathologic material from our study of cerebral atherosclerosis at the Massachusetts General Hospital has been especially examined in regard to the radiologically visible calcification and the corresponding pathologic changes in the carotid siphon. The observations on the atherosclerotic aspects of the study have already been reported.

In the present study the following arteries were selected for scrutiny: the entire carotid systems from their origin up through the neck and petrous bone to the region distal to the siphon, the entire vertebral systems from their origin up through the transverse foramina of the cervical vertebrae to the level of the foramen magnum intradurally, the circle of Willis (including the internal carotid artery stems, anterior cerebral arteries, the communicating vessels, the posterior cerebral arteries, basilar artery, and vertebral arteries), the aorta, and the left coronary artery. The vessels were removed, fixed in formalin, opened, mounted on clear plastic film, and preserved in plastic bags containing formalin.

The extent and severity of the atherosclerotic process were graded by one of us (I.G.) using the “atherosclerotic index” system that he has devised. In this system, the percentage area of the wall involved by atherosclerosis is first estimated and then the proportion of this area occupied by each of four different grades of lesions is determined: grade 1—flat plaque, grade 2—raised plaque, grade 3—ulceration, and grade 4—calcification. The atherosclerotic index (A.I.) ranges from 0 (no atherosclerosis) to 100 (100 per cent involvement by grade 3 or 4 lesions). In addition to grading the atherosclerosis the degree of stenosis was rated as grade 0—0 to 25 per cent reduction in the cross-sectional area of the lumen, grade 1—25 to 49 per cent, grade 2—50 to 75 per cent, grade 3—more than 75 per cent but not occluded, and grade 4—total occlusion.

By the time this part of the study was undertaken only 99 of the arterial specimens from the full study were still adequately preserved, the remainder having dried up because of evaporation of the formalin from the sealed bags. The preserved specimens of the entire carotid and vertebral systems in the 99 cases were x-rayed for evidence of calcification. The degree of calcification seen in the carotid siphon on the x-ray films was then graded by gross inspection into four degrees of severity, 0, 1, 2, and 3, (none, mild, moderate, and severe). These data then served as the principal basis for correlating siphon calcification with various other measures of the patient’s vascular and general disease—carotid atherosclerosis, steno-
Calcification of the Carotid Siphon

The Pathology of Carotid Siphon Calcification

Gross Examination

The siphon comprises the sinuous portion of the internal carotid artery lying between the exit of the artery from the carotid canal of the petrous bone and the region of its terminal division into anterior and middle cerebral branches. Although several different forms have been described, the commonest is a simple hairpin-bend with the convexity directed anteriorly. The siphon is uniquely situated in that much of it lies immediately adjacent to the venous blood of the cavernous sinus, the only such anatomic arrangement in the body.

In the early years of life the siphon is thin-walled and collapsible but with advancing age arterial changes are the rule, mural calcification being particularly prominent. This calcification is commonly regarded as part and parcel of the atherosclerotic process although this has not been conclusively demonstrated.

In our cases with heavily calcified siphons the inner surface of the artery was corrugated showing bony-hard yellowish-brown (honey-colored) excrescences, pits, and shiny smooth areas that formed an irregular, porous-appearing labyrinth through which it was sometimes difficult to trace the lumen of the vessel. The intima was not ulcerated. The appearance of stenosis was in part artifact, for usually on opening of a severely calcified siphon with a scissors the wall was shattered making it almost impossible to determine the natural patency of the lumen. As the vessel was spread open there was a tendency for the hardened subintima to separate from the remainder of the wall. Therefore in another 30 cases not included in the 99 we decalcified the removed siphon in Versene before opening it, trying in this way to avoid shattering the wall. In these cases the lumen of the siphon was only mildly encroached upon even in the most heavily calcified specimens.

The yellowish-brown calcified areas in the intima had a unique appearance not resembling ordinary atherosclerotic plaques seen in arteries elsewhere in the body. This could be either an unusual arteriosclerotic process or ordinary atherosclerosis in which the deposition of calcification is enhanced.

These calcified lesions, however, were not the only type of intimal abnormality found in the siphon; ordinary flat yellowish-white plaques of atherosclerosis of every size and thickness also occurred with little or no associated calcification; most often there was a mixture of calcified honey-colored lesions and ordinary plaques.

Microscopic Examination

In the study of the histologic changes that corresponded to the mural calcification, it was not practical to use the mounted arterial specimens for making microscopic sections. For this purpose therefore we used the removed siphons from 18 routine autopsy cases, and prepared sections after completing the gross examination and x-ray studies. Microscopically, the thin wall of the normal artery shows the three classical layers, intimal endothelium, media, and adventitia. The internal elastic lamina lying just beneath the endothelium is, like the cerebral arteries, especially well-developed compared with arteries elsewhere in the body.

The criterion of calcium deposition was deep blue staining with hematoxylin, and incineration studies were not made. Calcification of the siphon occurred mainly in three sites: (1) the elastic lamina, (2) the thickened subintima (atherosclerotic or arteriosclerotic plaque), and (3) the media, particularly the inner part. These findings are in keeping with an earlier report.4

It was our impression that calcification occurred earliest in the elastic lamina (fig. 1). Commonly only one or more segments of the circumference were involved, but occasionally it formed a complete ring of calcification around the vessel. In some cases calcification in the elastica was present when the intima...
otherwise was almost normal. The elastica, which was often split or frayed, broadened to three or four times its normal width as it became calcified. Microscopically the calcifying elastica no longer showed the typical retracted, undulating or folded appearance of the normal elastic lamina, presumably because of its increased rigidity. Atherosclerotic plaques on the other hand, even thick ones, did not prevent the adjacent normal elastica from assuming its usual wavy form.

The subintimal plaques in the siphon region were fairly typical of fibrous atherosclerosis, consisting usually of stratified or lamellar, syneclial, avascular collagenous connective tissue containing abundant elongated fibroblast nuclei; or the tissue had a hyaline appearance and clear or empty lacunae lay scattered throughout it. Fatty macrophages and cholesterol crystals were seen only occasionally. In these plaques the strands of collagenous material, the contained fibroblast nuclei, and even the endothelial nuclei tended to lie lengthwise along the vessel whereas the muscle nuclei of the media were arranged in the usual circular fashion.

This subintimal tissue was often partly calcified, the calcification involving a highly variable percentage of the cross-sectional area of the plaque, from almost none to approximately 50 per cent. Calcification usually was greatest deep in the plaque near the elastica (fig. 2) but exceptions to this were seen, when the superficial layers of the plaque were more heavily affected and the deeper part relatively spared. Shallow plaques were more heavily calcified than thick ones, and in the same cross-section the thickest segment of a plaque often was not calcified whereas in an adjacent sector a thin rim of atherosclerosis might be uniformly calcified. Calcification of the subintima was nearly always accompanied by calcification of the nearby elastica, as if deposition in the two sites was related. Occasionally the calcification of the elastica and adjacent subintima formed an irregular, broad bluish band extending round the entire circumference of the vessel. There was a striking tendency

*Figure 1*

Siphon wall. *P*, plaque; *E*, dark-staining internal elastic lamina; *M*, media.

*Figure 2*

for atherosclerosis to be thicker where the subjacent elastica was not calcified. No bone formation was seen. Organization of a mural thrombus was seen once microscopically. Old hemorrhage into plaques was not encountered.

The media was frequently calcified, usually the innermost part adjacent to the elastica being most involved (fig. 2). At times this calcification was well developed, transforming the inner one half or even more of the medial coat into a uniformly blue lamellar structure. The resultant thinning of the media possibly accounted for the fact that these lesions could be seen from the external surface of the vessel on gross examination. Medial calcification was much less common and much less severe than calcification of the elastica and plaques, and it never reached the massive proportions that it does in the arteries of the extremities. In the adventitia, calcification was scanty despite the presence of an abundance of elastic fibers.

The calcium deposits in the subintima and media most often consisted of multitudes of tiny blue-walled clear spheres of varying size, but other patterns such as sheaves and needles were also seen. Longitudinal sections of the arteries showed a patchy deposition of calcium along the length of the vessel just as it was patchy in cross-sectional views. Calcification was no more prominent on the convex inner wall of the curves of the siphon, where Samuel found atheroma to be exaggerated, than along the concave outer wall. In longitudinal sections, however, the sharp curves of the siphon could be seen clearly and, in some vessels, the convex inner wall of the curve, formed as the lower arm of the siphon turns upwards and backwards at its forward extremity, showed a calcified shelf jutting forward into the lumen.

Calcification appeared first in the elastica and extended secondarily to involve the arteriosclerotic subintima internally, and the muscle cells of the media, externally. Calcification begets calcification, a process termed renucleation. Siphon calcification, it appears, does not exactly duplicate arterial calcification elsewhere in the body, and for the present it is preferable not to force it into any commonly recognized category.

Calcification of the elastica was described by Mönckeberg and was regarded as distinct from muscle calcification in the media. Blumenthal et al. found that even in muscular arteries the internal elastic lamina was a site of predilection for calcification. A direct relation between calcification of elastic and muscle tissue was reported by Blumenthal et al., who found in calcification of the aortic media that the deposit of calcium occurred in association with the elastic fibrils of the media. This calcification, which was a function of age, was not so gross or homogeneous as in Mönckeberg's medial sclerosis. Hass et al. in experimental vitamin D intoxication noted that degeneration and calcification of the muscle of the media occurred adjacent to the elastica. The finding in our cases of calcification of contiguous regions of the internal elastic lamina and media is consonant with these reports.

The observation that subintimal thickening tended to be greater in areas where the elastica was not calcified and less where calcification of the elastica was greater, i.e., atherosclerosis and calcification of the elastica were inversely related, is in agreement with previous reports of this phenomenon in other arteries. Mönckeberg noted in the lower limb arteries that medial calcification increased distally as atherosclerosis diminished. Lindbom found, in the femoral artery, that medial disease and atherosclerosis were almost mutually exclusive. The author attributed the medial changes to the mechanical effects of bending of the artery where it crosses joints (knee, ankle, etc.) that undergo frequent movement. Harrison reported that in experimental animals when the production of hypervitaminosis D preceded cholesterol feeding, the calcific medial damage produced by the former prevented intimal lipid deposition, and when cholesterol feeding preceded hypervitaminosis D, medial damage was prevented. It is also well known that arteries encased in bone tend not to form atheroma. Willis suggested that calcification of the media by leading to decreased mural tension protected
against local atherosclerosis. It might be argued, however, that the presence of atherosclerotic plaques mechanically hinders or blocks calcification of the overlying elastica, an opinion expressed by Hass et al. In Mönckeberg's series of cases medial calcification in the legs was much more frequent than arteriosclerosis, indicating that if one process influenced the other, medial calcification probably suppressed arteriosclerosis rather than vice versa. Whereas in the arteries of the extremities medial calcification tends to be at a distance from arteriosclerotic plaques, in the carotid siphon both processes occurred in the same segment of vessel, so that the inverse relationship was less striking. Yet it could be detected in different sectors of the same cross-sectional view of the artery. It is a matter of no little theoretical importance to know if elastic calcification limits plaque formation. It should be added here that Blumenthal et al., dissenting from this whole concept, concluded that aortic calcification actually predisposes to local intimal atherosclerosis. Also, Wilens placed metallic bands or cuffs around the femoral and carotid arteries of animals and showed that intimal thickening occurred particularly beneath the section of vessel that was supported in this way.

As already mentioned heavily calcified plaques were shallower than noncalcified or less calcified plaques, suggesting that calcification limits the growth of a plaque. This would be a significant principle to establish, since it raises the possibility of therapeutically influencing human atherosclerosis by altering calcium metabolism. Furthermore, it is remarkable that although there is a special predilection for the development of "atherosclerosis" within the siphon, it is unusual for the deposit to "pile up" with the production of significant stenosis of the lumen.

The Radiologic Picture

Figures 3A and 3B are examples of the x-ray picture obtained in cases of grade 3 calcification of the siphon, the siphon being opened longitudinally before being x-rayed. The vessels are seen from the lateral aspect as in a lateral view of the skull and the calcification appears as map-like plaques of irregular shape and varying density. Figure 4A is a similar picture of a siphon before opening; figure 4B demonstrates the linear double contour of some calcified walls.

![Figure 3](image_url)

Figure 3

A and B show x-ray calcification of the carotid siphon (removed) in two cases; C and D show the corresponding carotid sinuses in the same cases.
CALCIFICATION OF THE CAROTID SIPHON

Figure 4

A and B show x-ray calcification in the unopened siphons (removed), in two cases.

It was not easy to determine with certainty which region of calcification—subintimal atherosclerosis, elastica, or media—was responsible for casting the x-ray shadows of the siphon. However, on comparing the x-ray shadow of a siphon with the appearance of the specimen itself, the irregular-shaped dense opacities on the film seemed to correspond to the hard honey-colored superficial atherosclerotic lesions. These smooth often shiny areas, representing heavy calcification in shallow plaques, did not significantly obstruct the lumen. When the vessel cast dense parallel lines, as in figure 4, the pathologic picture was one of heavy calcification of the subintimal tissue and adjacent elastica—the medial also in some cases—forming a calcified tube, again usually without significant narrowing of the lumen. In one specimen in which calcification was limited microscopically to the elastica the x-ray showed a faint although clear shadow suggesting that isolated calcification of the elastica would be scarcely discernible in ordinary clinical skull x-rays. Slight or finely granular microscopic calcification was not detected in x-rays of the removed specimen.

From a practical point of view it was still necessary to determine to what extent the x-ray picture of the siphon after removal at autopsy corresponded to the lateral and anteroposterior views of the region as seen in routine clinical skull films. It was fortunate that in 21 of the 99 cases x-rays of the skull had been made in life. Comparison of the films of the skull and of the removed specimen showed that calcification as mild as grade 2 in our rough scale was always visible in lateral views of the skull in life. Even in cases with grade 1 calcification a careful examination usually disclosed a faint shadow. Lateral views were more satisfactory. Thus almost all calcification seen in the postmortem x-rays of the removed specimen was visible in ordinary skull films. Nonetheless, in looking for correlations that might be translatable to the clinic, we have relied chiefly on calcification of grades 2 and 3.

In skull films siphon calcification appeared in the lateral view as somewhat fuzzy lines or small faint areas of density overlying the relatively radiolucent pituitary fossa. Since the shadows of the two siphons were superimposed, exact interpretation was often impossible. In the anteroposterior view heavily calcified walls were seen as rings or arcs of density on each side of the sella turcica often partially obscured by the cranial bones. These linear shadows no doubt represent mural calcification lying tangential to the x-ray beam and foci of calcification lying en face to the anteroposterior beam are not dense enough to cast a shadow except in the most severe cases.

In one case a carotid angiogram performed during life showed a distinct stenosis of the siphon in the posterior part of the lower arm of the siphon whereas at postmortem examination after decalcification only a moderate subintimal plaque was found at this site and
the in vivo stenosis was not adequately explained. Calcification of the siphon was minimal in this case and an ocular bruit had not been present in life.

**X-ray Calcification in our 99 Cases**

Of the 99 cases in which the carotid siphons were studied radiographically 50 were male and 49 were female. The number in each decade was fourth 4, fifth 8, sixth 18, seventh 33, eighth 34, ninth 11, and tenth 2.

The cases were almost equally divided among the four grades of calcification as seen by x-ray: grade 0–25 cases, grade 1–24, grade 2–24, grade 3–26, i.e., 75 per cent of cases showed some degree of calcification.

The calcification involved the siphon both in its intracavernous portion, that is the lower arm which runs forward to the region of the clinoid process and in the section distal to the cavernous sinus. In the vessels with heaviest calcification, the deposit extended over a distance of 2 to 5 cm. with most cases in the 25 to 33 mm. range. The cerebral arteries proper and even their atherosclerotic plaques were relatively free from calcification as Buck et al.\(^4\) and Patterson and Cornish\(^5\) have previously reported.

Calcification was approximately equal on the two sides in 77 of the 99 cases. In only three of the remaining cases was there a marked difference between the two sides.

**The Correlative Findings**

1. By inspection it was apparent that an excellent correlation existed between the grade of calcification by x-ray and the presence of the honey-colored lesions within the siphon. Cases with grade 2 or 3 calcification usually showed extensive intimal changes, and the arterial wall was transformed into a brittle shell. Although heavily calcified vessels were often fractured when opened with a scissors, making it difficult to define stenosis of the lumen, it could be reliably concluded that heavy calcification of the siphon by no means indicated significant stenosis of the lumen.

The absence of calcification by x-ray, however, did not exclude local atherosclerosis, for as mentioned previously pale yellowish-white atherosclerotic plaques could be present when calcification was absent or minimal. It is important to keep in mind the two different kinds of intimal lesion, calcified and "ordinary" plaques. Of the noncalcified plaques in this series none caused significant stenosis, i.e., more than 25 per cent narrowing of the lumen. Ratino\(^16\) in a recent clinicopathologic study found that calcification of the intracavernous and intrapetrous segments of the internal carotid artery did not correlate with the occurrence of gross atherosclerosis locally. The type of atherosclerosis referred to was not defined.

2. The relation of siphon calcification to the age of the patient is shown in figure 5. Calcification increased in incidence and severity with age, occurring in 80 per cent in the eighth decade and 90 per cent in the ninth. (As mentioned above grade 2 and 3 calcifi-
3. Regarding the influence of sex, when the groups with grade 0, 1, 2, or 3 calcification were divided into men and women, there was not much difference between the two sexes. Calcification was more commonly absent in males and more commonly severe in females. In the eighth decade the females showed more siphon calcification but less cerebral and aortic atherosclerosis than the males, suggesting a specific relationship to femininity rather than the amount of atherosclerosis. This tendency of the female to calcification outweighed the factors of hypertension and atherosclerosis. Also when the age group 60 to 80 was analyzed according to sex, blood pressure, and cerebral and aortic atherosclerosis, it was found that with approximately the same amount of atherosclerosis and the same blood pressure there was a definite preponderance of calcification in females over males of the same age. In the fifth decade all cases with grade 2 or 3 lesions were males.

4. To determine the effect of the blood pressure on calcification, at the same time controlling the age factor, a special analysis of the cases in the eighth decade was made. In this group, hypertensive males showed more calcification than nonhypertensive males. This increase paralleled greater atherosclerosis in the cerebral vessels but did not parallel greater aortic atherosclerosis. Hypertensive females showed much more calcification than nonhypertensive females and again this paralleled the severity of cerebral atherosclerosis but not of aortic atherosclerosis.

5. Figure 6 shows the relationship of siphon calcification and the atherosclerotic index (A.I.) of the aorta, and the carotid and cerebral arteries. In general there was a parallel, but the amount of scatter was great. For example, in the presence of grade 3 calcification of the siphon, the aortic A.I. ranged from 18 to 74, and with an aortic A.I. of 50 to 60 the calcification ranged almost uniformly from grade 0 to grade 3. In any individual case therefore one can make no reliable generalization concerning the severity of atherosclerosis elsewhere in the body from the degree of calcification in the siphon. The dotted line in each graph is the curve corresponding to the calculated (averaged) A.I. of the various arteries.

6. The x-rays of the siphon included on the same film the region of the carotid sinus (fig. 3C and D) so that an opportunity was provided for comparing calcification at these two sites. The amount of calcification was roughly comparable, 84 of the 99 cases being judged as equal or nearly equal. The siphon showed greater calcification in 13 and the sinus region in two. The calcium was strikingly localized to these two sites. In 19 cases a tiny fleck of calcium could be made out in the petrous portion of the artery, and in two cases there was a slightly greater deposit.

Calcification in the carotid sinus was always associated with atherosclerosis in the sinus but the latter was so common that the association might have been fortuitous. Usually
there was a quantitative parallel between the two but exceptions occurred, and there could be severe stenosing atherosclerosis of the sinus with a rather small amount of calcification or much calcification with relatively little atherosclerosis. Ring and Eddy\textsuperscript{17} in a radiologic study found that carotid sinus calcification as visualized in routine chest films increased with age but no further correlations could be made.

Six of every seven cases (85 per cent) with severe atherosclerosis of the carotid sinus showed grade 3 (severe) calcification of the carotid siphon. In other words if significant carotid sinus stenosis is present, it is likely that the siphon will be heavily calcified and the absence of calcification of the siphon is against severe carotid sinus stenosis.

7. In the 99 cases of the study there were 16 with myocardial infarction. Of these, four had grade 3 calcification of the siphon, six had grade 2, five grade 1, and one grade 0. Those with grade 3 were in the eighth and ninth decades. The cases were too few to warrant a conclusion but myocardial infarction appeared not to be related to a high degree of calcification, and the data might even suggest a negative correlation between stenotic coronary atherosclerosis and siphon calcification. Young et al.\textsuperscript{18} found a parallel between coronary and cerebral atherosclerosis in 37 consecutive autopsy cases, a finding corroborated in the major part of our study.\textsuperscript{5} Oliver et al.\textsuperscript{19} actually found a parallel between coronary calcification and ischemic heart disease, but a quantitative analysis was not undertaken.

8. Regarding cerebral lesions, in the total group of 99 cases, there were eight with état lacunaire and seven of the eight had grade 3 calcification; however the other one had none at all. Lacunes have previously been correlated with atherosclerosis and hypertension.\textsuperscript{20} There were four cases of carotid thrombosis and four with thrombosis of an intracranial artery and all had grade three calcification of the siphon. Thus siphon calcification though not strongly correlated with the cerebral A.I. seems to be related to the occurrence of cerebral thrombosis. There was not such a close parallel with embolic brain infarction or intracerebral hemorrhage. Of the eight embolic cases none had grade 0 calcification, three had grade 1, three grade 2, and two had grade 3. Of the 10 cases of hemorrhage three had grade 0 calcification, one had grade 2, and six grade 3. Of the total of 26 cases with grade 3 calcification 23 had cerebrovascular lesions, a clear indication of the close relationship of the two. Yet exceptions occurred on both sides. Cole and Davis\textsuperscript{21} in a clinicoradiologic study found a higher incidence of clinical cerebral vascular disease in a group of patients with calcification of the siphon than in a group of matched controls without calcification.

From a practical point of view cerebral thrombosis or stenosis with its connotation of actual or threatening brain ischemia is of much greater immediate significance than the A.I. (atherosclerotic index), which is a measure of the extent and ulceration of the plaques. Indeed, in future studies, emphasis might be placed on a stenotic index or thrombotic index rather than the A.I.

That siphon atherosclerosis as evidenced by siphon calcification parallels carotid atherosclerosis and cerebral thrombosis yet itself rarely leads to significant stenosis is an incongruity which, as already mentioned, might suggest that the inordinate amount of calcification limits or checks the growth-in-thickness of siphon plaques. The steady increase in calcification without much increase in bulk would mean that the "atherosclerotic" tissue calcifies soon after its deposition.

9. Since siphon calcification paralleled calcium deposit in the carotid sinus the possibility arose that this kind of calcification reflected a widespread metabolic tendency. With this in mind a scattergram was made to show the relationship of the calcification in the aorta as determined by x-ray and that in the siphon. There was a rough parallel but a great deal of scatter was evident.

10. Calcification in the vertebral artery on one or both sides was found by x-ray in 19 cases. In 15 cases it consisted of a single
fleck or two, approximately 2 mm. in extent while in the other four cases a few flecks were seen. These were situated anywhere along the artery but more were clustered in the lowermost, middle, or uppermost segments. Without exception calcification was found here only in cases with moderate or severe calcification in the carotid system, i.e., there was a distinct parallel.

11. In a review of the x-ray films of the 21 cases in which the skull and other parts of the body had been x-rayed before death no parallel could be established between siphon calcification and calcification of the pineal gland, petroclinoid ligaments, aortic arch, costal cartilages, phleboliths, or hyperostosis frontalis interna. No interrelationship between the calcification at any of these several sites could be discerned.

12. In this series there were 31 cases with cholelithiasis but no correlation was found between cholelithiasis and siphon calcification.

Conclusions and Summary

The gross and microscopic features of the vascular calcification occurring in the carotid siphon have been presented. The degree of calcification found in the siphon is distinctive, and an exact counterpart elsewhere in the body has not been described. Calcification was found in the internal elastic lamina, intimal plaques of "atherosclerosis," the media and very slightly in the adventitia. Calcification of the elastica and the local deposition of atherosclerosis tended to be inversely related. The pathologic lesions corresponding to the calcification seen by x-ray were identified.

The amount of calcification in the carotid siphon was determined by x-ray in 99 pairs of arterial specimens removed at autopsy. The calcium deposit was sharply restricted to the region of the siphon. The x-ray shadow paralleled the presence of flat honey-colored calcified plaques of atherosclerosis. Pale-yellow plaques of atherosclerosis without calcification could be present with a negative x-ray. Calcification increased with age and in the older age groups was more pronounced in women. Hypertension was clearly associated with increased calcification. A parallel existed between siphon calcification and atherosclerosis of the aorta, carotid and cerebral arteries, but the scatter was great and in an individual case, a generalization concerning the severity of atherosclerosis elsewhere is not justified. The best correlation was between severe calcification of the siphon and advanced atherosclerosis of the carotid sinus.

All cases with thrombosis of the extracranial and intracranial cerebral arteries showed severe (grade 3) siphon calcification as did seven of eight cases of état lacunaire. Grade 3 calcification therefore was correlated with cerebrovascular thrombosis but lesser grades were not. There was possibly a negative correlation between siphon calcification and myocardial infarction.

The correlation of siphon and aortic calcification was poor. Siphon calcification did not correlate with calcification of structures elsewhere in the body or with cholelithiasis.

Our findings suggest that calcification of the atherosclerotic tissue may limit the formation or accumulation of atherosclerosis. If this is so, and if myocardial infarction correlates negatively with siphon calcification, the possibility is raised that therapeutically altering calcium metabolism locally or generally might restrict the atherosclerotic deposit.

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Adolf Fick—1829-1901

With a strong early bent toward mathematics, Fick was first educated for a university degree in mathematical physics at Marburg, but changed to medicine on the advice of an older brother. After taking his medical degree, he accepted a prosectorship with Carl Ludwig in Zurich in 1852, and was one of Ludwig's first students. They remained lifelong friends. Fick continued at Zurich for sixteen years, then was appointed to the chair of physiology in Würzburg, succeeding von Bezold. Here he continued for the rest of his active life. The faculty at Würzburg was a distinguished one, and Fick with his broad interests, had many friends: the anatomist Köllicher, the jurist Regelsberger, the pathologist Rindfleisch, the clinicians Bergmann and Gerhardt, the chemist Wielicenus, and the physicists Clausius, Quincke, Kohlrausch, and Röntgen.

Fick achieved distinction as a physicist when he was quite young; his law of diffusion in fluids was published in 1855, when he was twenty-six years old. He was a man, then, who thought about physiology in quantitative terms, who was a good physicist and mathematician, and who was able to bring separate but related phenomena into quantitative formulation.—William F. Hamilton, M.D., and Dickinson W. Richards, M.D. Circulation of the Blood. Edited by Alfred P. Fishman, M.D., and Dickinson W. Richards, M.D. New York, Oxford University Press, 1964, p. 93.
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