of the body, resulting in an increased production of uric acid. According to the newer theories of uric-acid formation, it must be supposed that under the influence of lead, there results an excessive destruction of nuclein containing substances. This toxic influence is, according to von Jacksch, a decreased oxidation.

CONCLUSIONS.

From the above opinions of the effect of lead on the blood vessels and on those organs which, when diseased, have an associated or resultant thickening of the walls of the vessels, we may conclude that lead may produce a disease of the blood vessels either directly or indirectly.

Directly, as shown by the numerous experiments on animals. Probably this is by the direct influence on the vessel of the lead in the circulating blood. Doubtless the walls of some of the larger arteries may be affected through the alteration in the nutrition, because of the primary toxic endarteritis, with the tendency to thrombosis and obliteration of the vasa vasora.

The undisputed fact that lead may cause gout, and the equally true proposition that in gouty individuals arteriosclerosis occurs early, may explain the influence of lead in producing arteriosclerosis in rare cases.

Experimental and clinical observations prove incontestably that lead will cause nephritis. This is usually the contracted kidney, which sooner or later has associated with it the thickened arteries and left cardiac hypertrophy.

Lead intoxication may lead directly and indirectly to arteriosclerosis.

The recognition of lead intoxication during life, by any of the ordinary symptoms or by the basophilic granulations of the cells of the blood, in an individual suffering with arteriosclerosis, with or without nephritis, would not, perhaps, permit us to say definitely that the lead was the sole causative disease factor. It would, however, be a rational therapeutic measure to safeguard the individual against further intoxication with lead in the treatment of the arteriosclerosis.

100 State Street.

THE RELATION OF ALCOHOL TO ARTERIOSCLEROSIS.*

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LITERATURE.

Most standard text-books[1] assert unconditionally the importance of the excessive use of alcoholic liquors in the production of arteriosclerosis. Standard treatises on the heart and blood vessels[2] are equally emphatic. In Nothnagel's magnificent "Specielle Pathologie und Therapie," Schrötter says, after reviewing the literature: "Alcohol is considered by practically all writers on arteriosclerosis as the chief cause or at least one of the most important causes of the condition." Yet Schrötter himself seems to be somewhat dubious regarding the evidence on which the belief is founded, and there are others, such as Duclos[3] and Ribberts,[4] who are yet more outspoken in their skepticism.

CLINICAL RESEARCH.

1. I investigated first the question:

How many cases of arteriosclerosis not referable to syphilis or to advancing age are to be found among alcoholics?

At the Bridgewater State Farm and the Foxboro Asylum for Dipsomaniacs, I investigated 283 cases of the severest and most chronic forms of alcoholism, excluding those over 50 years of age and those who have had syphilis. A quart of whiskey daily for many years was a not infrequent history in these cases.[5]

Of the total 283 cases, only 18, or 6 per cent., showed arteriosclerosis, so far as could be ascertained by examination of the heart and peripheral vessels, and by questions regarding the functions of the kidney.[6] If the cases over 40 years be excluded also, the percentage showing arteriosclerosis falls to 1.4 per cent.

2. I next endeavored to determine in what proportion of cases of relatively early arteriosclerosis of the peripheral arteries alcoholism was present.

Out of the several hundred cases of arteriosclerosis seen by me at the Massachusetts General Hospital, 45 were under 50 years of age. Only 6 of these, or 13 per cent., gave any history of alcoholism.

POSTMORTEM EVIDENCE.

3. By the kindness of Dr. J. H. Wright of the Massachusetts General Hospital clinicopathological laboratory, and of Dr. F. B. Mallory, pathologist to the Boston City Hospital, I was enabled to examine the records of 656 patients, 95 of whom were under 50 years of age and with no history of syphilis, in which arteriosclerosis formed a part of the pathologic diagnosis at the autopsy. On looking up the clinical records of these 95 cases, it was found that in 57 cases, or 60 per cent., it is expressly stated that the patient took no alcohol. Regarding the remaining 39 cases, in the table given below, I have quoted the actual words of the record. It will be seen that in 8 cases excessive alcoholism is expressly stated to have been present. In 14 cases it is stated or implied that the amount of alcohol consumed was moderate, while in 5 cases the statement may be interpreted as an insignificant amount of alcohol. Subtracting these 19 cases we have left 20, or 21 per cent., in which the consumption of alcohol was excessive.

It will be seen from the table that 8 of the cases among those who took more or less alcohol, were found at the autopsy to have chronic nephritis as well as arteriosclerosis, so that if, as is usually believed, chronic nephritis is a cause of arteriosclerosis, these cases should be subtracted from those which might be supposed to be due to alcoholic excess. This would remove 4 of the cases in whose clinical record excessive alcoholism played a feature.

It should also be borne in mind that as most of these cases were taken from the records of the Boston City Hospital, and as that hospital is patronized very largely by those in whose history alcoholism is a feature, the possibility of a coincidence in the occurrence of alcoholism and arteriosclerosis can not be disregarded.

Finally, I will mention briefly the case of a well-known young Bostonian, who deliberately drank himself to death at the age of 36. For the previous ten years

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*Read at the Fifty-fifth Annual Session of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. J. M. Anders, Frank Jones and W. S. Thayer.

1. For example: Osler, Tyson, Strumpell, Wood & Fitz, Loomis-Thompson, and Krehl (in V. Mering).

2. For example: Huchard, Byrom, Bramwell, and Babcock.

3. Duclos: Du Systeme arteriel chez les alcoholiques. Paris, 1888.

4. Ribbert: Pathol. Anatomie, 1902, p. 612.

5. I desire expressly to thank Dr. Charles A. Drew, Medical Director of the Bridgewater State Farm, for his assistance in securing data on these cases.

6. Patients whose arteries were merely palpable without being tortuous, hard or rough were not counted as cases of arteriosclerosis.

of his life he had been in the habit of taking one or two quarts of whiskey a day for about eight months in the year. At the postmortem examination particular attention was paid to the condition of the arteries, and they were found to be entirely normal.

CONCLUSIONS.

1. Only 6 per cent. of 283 cases of chronic and excessive alcoholism under 50 years of age showed any evidence of arteriosclerosis.

2. Of 45 cases of arteriosclerosis examined by me at the Massachusetts General Hospital, only 13 per cent. gave any history of alcoholism.

3. Of 656 autopsy cases of arteriosclerosis, only 95, or 14.5 per cent., were under the age of 50.

4. Out of these 95 cases under 50, in which arteriosclerosis was found postmortem, only 21 per cent., and if we exclude cases complicated by chronic nephritis, only 17 per cent. appear to have consumed alcohol in any notable excess.

The details of the autopsied cases may be seen from the following table:

TABLE OF POSTMORTEM AND CLINICAL RECORDS IN CASES OF ARTERIOSCLEROSIS.

Case.	Age in Years.	Pathologist's Diagnosis.	Previous Diseases and Present Complications.	Amount of Alcohol Consumed.
1	30	Art.....	Ch. nephritis.....	Five or six whiskies daily for last 15 years.
2	36	Art.....	.....	One or two wines daily; never to excess.
3	41	Art.....	Phthisis.....	Moderate alcohol.
4	24	Sl. art.....	Ch. nephritis.....	Alcohol moderate.
5	43	Sl. scl. of aorta..	.....	Always hard drinker.
6	37	Art.....	Phthisis.....	Four or five whiskies and four or five beers daily.
7	21	Sl. scl. of aorta..	Typhoid fever	Alcohol in moderation, but regularly.
8	35	Gen. art.....	Ch. nephritis.....	Four or five whiskies before breakfast.
9	21	Sl. art.....	Typhoid fever.....	One whisky every other day.
10	38	Sl. art.....	Diphtheria, ch. nephritis.	Four to six beers daily; occasionally whisky.
11	31	Sl. art.....	.....	Two or three whiskies and four or five ales daily.
12	32	Art.....	.....	"Drinks considerably."
13	43	Sl. art.....	.....	Alcohol excessive.
14	30	Art. of aorta.....	.....	Two glasses of whisky a week and two beers daily.
15	43	Art. aorta.....	.....	Bee and whisky occasionally.
16	39	Art.....	Typhoid fever....	Half pint whisky daily.
17	39	Art.....	Typhoid fever....	Occasionally whisky.
18	40	Art.....	.....	Alcohol to excess.
19	44	Art.....	Ch. nephritis.....	Drank heavily of beer.
2	46	Art. of aorta.....	.....	Alcohol to excess.
21	34	Art.....	.....	Alcohol to excess.
22	46	Art.....	.....	Two or three whiskies and two beers daily.
23	46	Art.....	Aortic regurg.....	Alcohol moderate, but steadily for years.
24	25	Sl. art.....	.....	Alcohol in excess.
25	33	Scl. mitral curtain and aortic arch.	.....	Alcohol in great excess.
26	37	Aortic scl.....	.....	Alcohol for last 10 years.
27	24	Coronary scl....	Ch. nephritis; scarlet fever.	Three or four beers daily; occasionally whisky.
28	42	Aortic scl.....	Ch. nephritis.....	Alcohol to excess
29	43	Gen. art.....	Phthisis.....	Four or five beers daily.
30	48	Gen. art.....	Ch. nephritis.....	Alcohol considerable.
31	38	Gen. art.....	Fatty deg. heart..	Diag. "alcoholism" (acute). Amt. of alcohol not known.
32	20	Art.....	Typhoid fever.....	Takes less than one drink a day (beer).
33	44	Gen. art.....	Ch. nephritis.....	Occasional glass of ale, gin or whisky.
34	29	Gen. art.....	Aortic insuf.....	Four beers daily.
35	41	Art.....	.....	Rum, three glasses week, year around.
36	42	Art.....	.....	Two or three bottles of ale a day.
37	39	Art.....	Typhoid fever.....	Drank lots of beer till year ago—four or five glasses a day—and "a certain amount of whisky."
38	40	.....	.....	No alcohol for last 13 years; before then 14 or 15 whiskies a week
39	41	Art.....	Smallpox.....	Two or three beers daily; half pint of whisky a week, off and on.

In Cases 40 to 95 the record expressly states that the patient did not use alcohol.

Art.—Arteriosclerosis; ch.—chronic; sl.—slight; gen.—general; scl.—sclerosis; deg.—degeneration.

ANGINA PECTORIS AND ARTERIOSCLEROSIS.*

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In contrast to the many uncertainties relating to angina pectoris is the circumstance that in a large proportion of cases the attack is only an incident in the history of arteriosclerosis. Since Edward Jenner demonstrated postmortem disease of the coronary arteries, the association of a lesion of these vessels with the disease has been accepted as one of the best attested facts in cardiac pathology. Not that it has helped much to explain the mysterious nature of the pain of the attack, or all of the phenomena of the paroxysm. Pain in arteriosclerosis, as we see it in other parts, deserves a more careful study than it has yet received. In the head there is the association of migraine with arterial disease, the severe and characteristic headaches of arteriosclerosis and high pressure, and the agonizing pain in some cases of embolism of the cerebral arteries, more rarely in thrombosis. Abdominal pain is not often due to vascular disease, though there are cases in which, from the situation and intensity of the paroxysm it might rather be called angina abdominis than pectoris. There may be severe pain in lesions of the mesenteric arteries and in thrombosis of the iliac vessels in typhoid fever. It is in sclerosis of the arteries of the extremities that we meet with the most remarkable disturbances of sensation. The pain in embolism or thrombosis of the femoral or popliteal arteries is very intense, particularly at the site of the lesion. In the ordinary sclerosis, particularly of elderly persons, there may be, first, simple paresthesiæ, the numbness and tingling so commonly complained of; secondly, attacks of painful cramps, usually slight and nocturnal, or recurring paroxysms of extraordinary intensity and deserving the name of angina cruris more than intermittent claudication, which Walton has applied to it; thirdly, the pain, not always present, in intermittent claudication: fourthly, the paroxysms of pain with erythema, etc., the arteriosclerotic type of erythromelalgia.

But the pain in angina pectoris is *sui generis*, unlike in intensity any known variety, and while its association with coronary artery sclerosis is unquestionable, there is something additional, some other element for which, as yet, we have no explanation.

In relation to arteriosclerosis, there are four groups of cases of angina pectoris. First, the neurotic, in which in young persons all the symptoms of the disease may be present and death occur, and autopsy shows normal coronary arteries and neither local nor general disease of the arteries. Such cases are not common, but I have reported an instance in a young man of 28, who had had for years paroxysms of cardiac pain of the most agonizing form, who died in an attack.

Secondly, the angina pectoris of young men associated with syphilitic arteritis, aortic or coronary, or both. This is a very distinctive form, occurring usually in men under 35 years of age. There may be no general arteriosclerosis, but the lesion is either at the root of the aorta, or involves the sigmoid valves, or it may be confined to the coronary arteries. The anginal attacks, while severe, are sometimes relieved or even cured by the iodids. The paroxysms of pain may be the initial symptoms of aneurism of the first portion of the arch.

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