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CHANGES IN THE HEART'S CONDUCTION SYSTEM IN HYPERTENSIVE STATES

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It was during the first three decades of the present century that investigations into the heart's conduction system were most numerous and made the greatest progress. Recently ÁBRAHÁM and ERDÉLYI [1] have studied the structure and innervation of the conduction system in mammals. While the anatomical discoveries were followed by hundreds of reports dealing with pathological and physiological problems, the few morphological studies published have been restricted to the description of just a few, or even of isolated, cases. Especially scarce are the studies based on the concurrent evidence of clinical data and morphological examinations.

The first detailed studies of the subject at issue are naturally associated with the names of KEITH, FLACK, ASCHOFF, TAWARA and HIS, as well as with that of MÖNCKEBERG. The subsequent works of GROSS and FRIED [4], MAHAIM [7] and then ZANCHI and LENÈGRE [10], based on a larger material, have considerably contributed to our present knowledge. LEV et al. [5] established a close parallelism between the ECG and the morphological findings, and emphasized that their work was the first to approach the subject from this angle.

The increase in the number of hypertensive patients suffering disturbances of cardiac conduction has prompted us to undertake the present study.

Material and methods

The results of 24 post-mortem examinations will be dicussed. Their grouping is shown in Table I, while the essential data in Table II.

The cases were selected on the evidence of clinical and anatomical data. They all displayed the three morphological manifestations of hypertension, i.e. arteriosclerosis, nephrosclerosis, and cardiac hypertrophy. We endeavoured so to select the material as to survey the widest range of conduction disturbances, heart weights, coronary and myocardial lesions. The control cases represent the vascular diseases usual in each of the age groups.

The conduction system was worked up according to MAHAIM and LEV, except extremely dilated hearts, when the lower fourth of the branches of His' bundle was excised in a horizontal block. From 150 to 200 sections were made in each case. They were stained with Böhmer's haematoxylin-eosin, iron haematoxylin, Crossmon's trichrome, van Gieson's stain, van Gieson + orcein, Gömöri's silver impregnation, Sudan III and Oil Red 0.

Table 1

		No. of cases	Sex	Average age (years)
Cases of hypertension	Malignant and essential benign hypertension	6	18 (9♂, 9♀)	64.1
	Secondary hypertension	2		
	Diabetes mellitus	4		
	Arteriosclerotic hypertension	6		
Control cases	Arteriosclerosis	3	6 (4♂, 2♀)	64.0
	Cor pulmonale and arteriosclerosis	2		
	No vascular lesion	1		

Clinical diagnoses of conduction tissue lesions

	No. of cases
Atrioventricular block affecting both bundle branches	1
Alternating atrioventricular block	1
Block of right bundle branch	2
Block of left bundle branch	3

Table 2

Case No.	Age, years	Clinical diagnosis	ECG findings	Blood pressure mm Hg	Heart weight, g	Autopsy findings	Autopsy after hours
1 10/58 control	80	Cardiorespiratory decompensation, arteriosclerosis	Right branch block	150/90	450	Nephrosclerosis, arteriosclerosis with dilatation of heart	28
2 19/58 control	54	Lung tumour		140/90	390	Bronchial cancer	4
3 23/58	60	Chronic pyelonephritis, hypertension	Left ventricle hypertrophy	250/130	650	Chronic pyelonephritis, generalised arteriosclerosis	15
4 25/58	68	Diabetes	Left branch block	160/90	535	Arteriosclerosis, nephrosclerosis	16
5 39/58	79	Hypertension. cardiac infarction	Myocardial lesion	190/100	500	Benign hypertension, myocardial infarct	24
6 41/58	74	Cardiorespiratory decompensation, WPW syndrome	Shorter PQ interval, WPW syndrome	155/80	420	Nephrosclerosis, arteriosclerosis, cardiac hypertrophy	15

Case No.	Age, years	Clinical diagnosis	ECG findings	Blood pressure mm Hg	Heart weight, g	Autopsy findings	Autopsy after hours
7 42/58	71	Hypertension, coronary sclerosis, pulmonary embolism	Absolute arrhythmia	150/110	540	Nephrosclerosis, arteriosclerosis, dilatation of heart	8
8 129/58 control	27	Acute leucosis	Right deviation of R axis	135/70	285	Acute leucaemic myelosis, subendocardial heamorrhage in Tawara node	6
9 152/58	53	Periarteritis nodosa, hypertension	Left deviation of R axis, repolarisation disturbance	230/120	450	Polyarteritis nodosa, dilatation of heart	20
10 166/58	60	Diabetes, coronary occlusion	Grave repolarisation disturbance	180/100	560	Diabetes, grave generalised arteriosclerosis	7
11 184/58	68	Diabetes, repeated myocardial infarction	Previous infarct in posterior wall, left branch block	150/90	500	Diabetes, grave general arteriosclerosis, coronary sclerosis	13
12 319/58	64	Diabetes, generalised arteriosclerosis	Grave myocardial lesion	160/10	360	Grave generalised arteriosclerosis chiefly in coronary and cerebral vessels	5½
13 322/58	59	Coronary sclerosis, chronic cor pulmonale, decompensation	Grave myocardial lesion	160/10	505	Nephrosclerosis, arteriosclerosis, dilatation of heart ventricles	12
14 336/58	59	Cardiomyopathy, cardiac decompensation, pulmonary embolism	Disturbed repolarisation	165/100	465	Hypertension of arteriosclerotic origin	24
15 370/58	73	Coronary occlusion, generalised arteriosclerosis	3 mm elevation of ST	120/80	500	Hypertension of arteriosclerotic origin. Infarction of septum and anterior wall	5
16 7/59	68	Pulmonary tbc, haemoptysis, heart insufficiency		140/70	275	Pulmonary haemorrhage of bronchiectasic origin, brown atrophy of myocardium	5

Case No.	Age, years	Clinical diagnosis	ECG findings	Blood pressure mm Hg	Heart weight, g	Autopsy findings	Autopsy after hours
17 373/58	63	Essential hypertension decompensation, cerebral embolism	Disturbed repolarisation	180/140	600	Essential hypertension, septal infarcts, bovine heart	6½
18 434/58	72	Arteriosclerosis, myocardial infarction	Myocardial infarction, coronary lesion	110/80	410	Grave general arteriosclerosis, aneurysm of right ventricle	5
19 445/58	68	Sudden death. A—V block, pulmonary embolism	Alternatives A—V, left and right branch block	190/90	475	Arteriosclerotic nephrosclerosis, moderate general arteriosclerosis	23
20 457/58	57	Essential hypertension, absolute arrhythmia, cor bovinum	Absolute tachyarrhythmia elevated ST	240/150	800	Essential hypertension, bovine heart	6½
21 40/59	83	General arteriosclerosis, positive complement fixation	Myocardial lesion, left branch block	180/90	520	Grave aortic and coronary sclerosis, recent infarction of posterior wall	8
22 250/59	58	Hypertension, chronic circulatory failure	Grave myocardial lesion, right branch block	190/100	640	Benign hypertension, arteriolosclerotic nephrosclerosis, bovine heart	
23 604/59	56	Essential hypertension, myocardial infarction	Left deviation of R-axis, disturbed repolarisation	240/140	550	Essential hypertension, coronary stenosis	4
24 638/59	73	Total A—V block, general arteriosclerosis, cardiac decompensation	A—V block, infarct in anterior wall	200/90	390	Essential hypertension, arteriolosclerotic nephrosclerosis	18

Results

I. Atrioventricular node ; Atrioventricular bundle

The ramus septi fibrosi which supplies the atrioventricular node starts, according to MAHAIM [7], immediately in front of the ramus descendens posterior; it originates from the right coronary trunk in 90 per cent and from the left one in 10 per cent of the cases. The artery of the node traverses the bundle of His and supplies the initial portion of the branches of the latter too. MÖNCKEBERG [8] found anastomoses between the ramus septi fibrosi and the ramus descendens anterior.

1. *Changes of vascular origin*

The pathomechanism of the vascularly conditioned lesions of the horizontal part of the conduction system is different according to whether they are due to benign hypertension which affects also the larger coronary branches or whether they consist in arteriolosclerotic changes characteristic of malignant hypertension. In cases of arteriosclerotic coronary stenosis anastomoses are formed in which the ramus septi fibrosi and its side branches are readily participating. For instance, occlusion was observed in the right coronary trunk in Cases 12, 14 and 15, without concomitant necrosis in the corresponding part of the conduction system. Then was necrosis around atrioventricular node in Case 12, but the node itself seemed to be unimpaired. Atheromatous occlusion at the origin of the ramus septi fibrosi was observed in Case 23, and it was only the bundle of His which showed signs of focal necrosis. In association with vascular changes characteristic of malignant hypertension we found, on the other hand, focal necrosis in the atrioventricular node which did not extend to the bundle of His (Case 20). A considerable destruction of fibres was observable around the small vessels in a subject with periarteritis nodosa (Case 9).

Other lesions of vascular origin were as follows:

a) Proliferation of interstitial connective tissue. This is due to ischaemia even if it is a primary phenomenon in persons of advanced age (ZANCHI and LENEGRE, 10), *i. e.* when the degeneration of the conduction fibres is not pronounced. Considerable differences in the size of conduction fibres develop hand in hand with the accumulation of connective tissue, and the connection of the fibres with the atrial muscles is gradually lessened. The relative increase in the number of nuclei, too, points to a destruction of the fibres. The substance of atrioventricular node becomes less (Cases 1, 4, 5, 6, 10, 14, 19, 21, 23) and it is only at the commencement of His bundle that conduction fibres can be found in grave cases (Case 7).

b) Atrophy of the conduction fibres. With the gradual accumulation of interstitial connective tissue, blood supply becomes insufficient and the fibres of both the atrioventricular node and bundle of His undergo degeneration. Degeneration in His' bundle is most pronounced in its peripheral parts, a phenomenon which is due also to dilatation and environmental factors. The brown atrophy of the musculature involves the conduction fibres as well (Case 16).

c) Dilatation of the ramus septi fibrosi and its side branches, caused by the formation of anastomoses, has already been mentioned. If the sclerosis of the coronary trunk assumes grave proportions, the dilated tortuous vessels

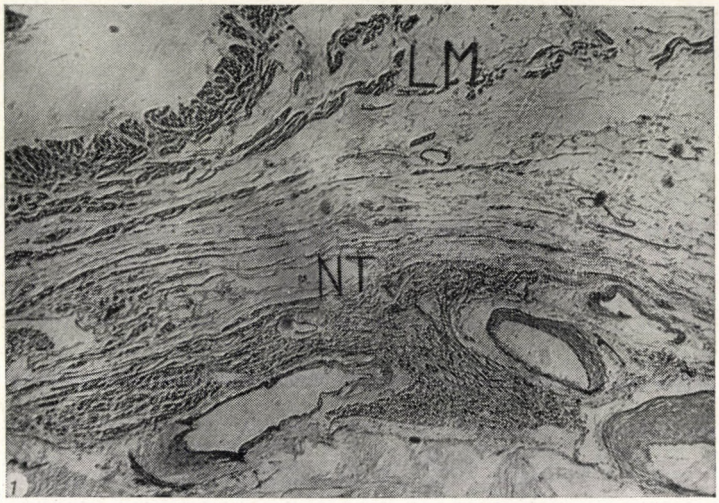

Fig. 1. Case 15. Effect of cardiac and vascular dilatation on Aschoff's node. Frontal section. Conduction fibres extending over large area; reduced connection between muscles of the node and atrium. Note fibrosis around distended vessels. Haematoxylin-eosin

in the atrioventricular node and His' bundle may partially rupture or even destroy the remaining fibres (Fig. 1).

The small vessels become stenosed or obstructed in malignant hypertension so that necrobiotic and necrosed areas will appear in the node (Case 20, Fig. 2), together with a reactive proliferation of the interstitial tissue.

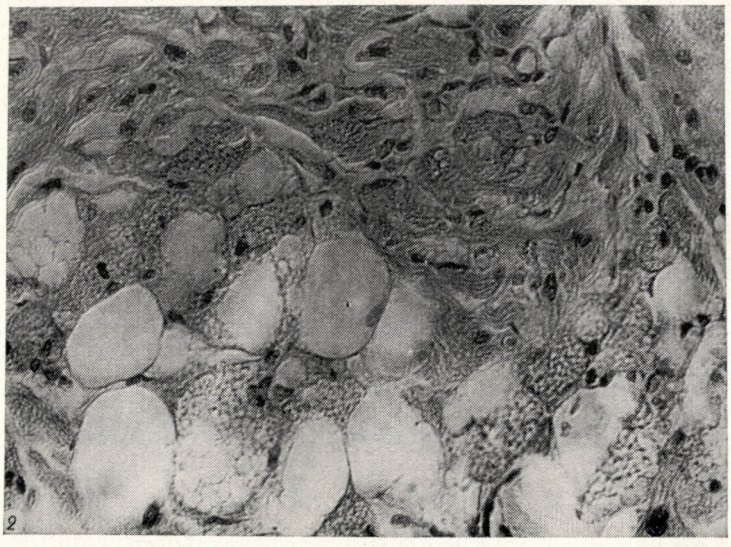

Fig. 2. Case 20. Fresh focal necrosis in Aschoff's node. Many of the necrosed and swollen muscle fibres are depleted of cytoplasm; they resemble fat cells. No infiltration with granulocytes. Haematoxylin-eosin

It accumulates in islets and frequently in the form of a perivascular ring. We saw few granulocytes in the focal necrosis of the conduction tissue while the appearance of lymphocytes seemed to be regular, and their number great, during fibrosis.

2. *Changes of environmental origin*

With advancing age and the progress of hypertension and arteriosclerosis, more or less in proportion with cardiac dilatation, accumulation — sometimes even hyaline degeneration — of the connective tissue around the annulus

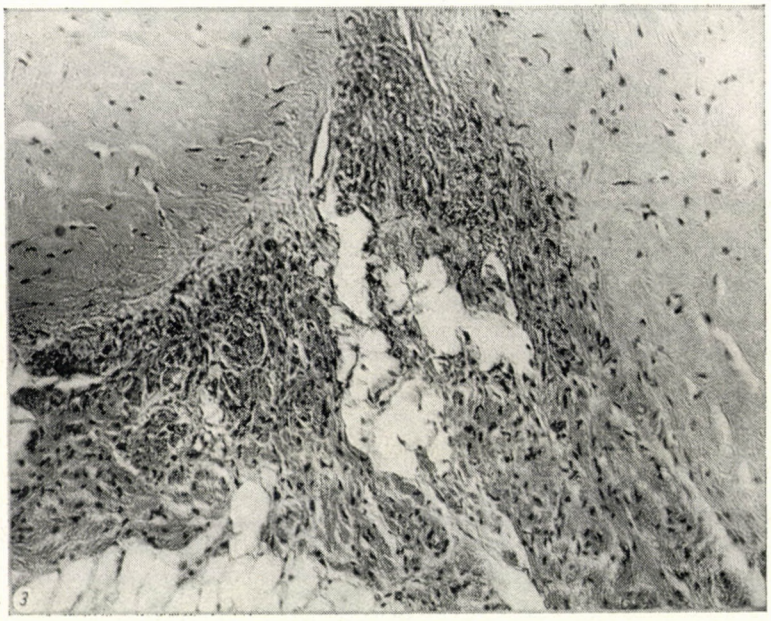

Fig. 3. Case 14. The unevenly enlarged annulus fibrosus and trigonium mediale compressing the fibres of the atrophied Aschoff's node. Haematoxylin-eosin

fibrosus can be observed. The growing mass of coarse-fibred connective tissue invades the atrioventricular node and tears smaller or larger pieces off the specific musculature which takes a veritably pinched appearance (Fig. 3). Perinodal fibrosis often affects the node itself (Cases 9, 12, 19). The connective tissue of the interventricular septum and the collagenous fibres of the membranaceous septum undergo hyaline degeneration. The connective tissue may extend to the interspaces of His' bundle and break up the concurrent fibres into fragments (Fig. 4). The capillaries may also suffer damage, and local ischaemia will induce degeneration of the conduction fibres. Such lesions affect the bifurcation of His' bundle as well, although generally to a lesser

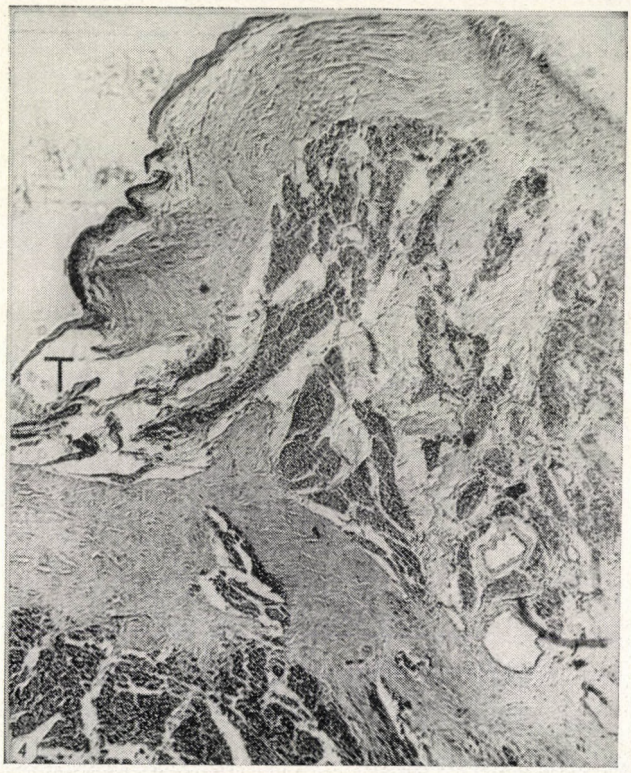

Fig. 4. Case 17. His' bundle fragmented by fibrosis extending to the left bundle branch. Frontal section. Haematoxylin-eosin

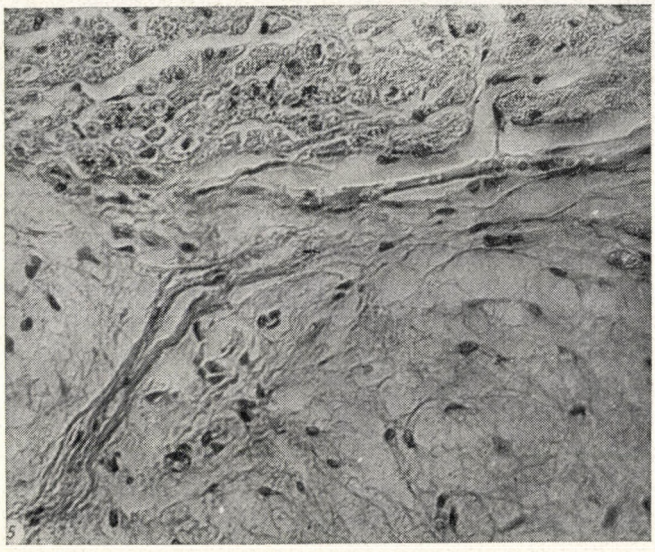

Fig. 5. Case 20. Myxomatous tissue, destruction of specific muscles at the bifurcation of the His' bundle. Haematoxylin-eosin

extent. Septal fibrosis mostly reaches this area and affects the bundle branches. The perivalvular connective tissue may show signs of myxomatous degeneration (Case 12). The myxomatous tissue in the musculature of the bifurcating portion may give rise to grave destruction (Fig. 5).

3. *Changes due to dilatation*

Owing to the dilatation of the heart and the destruction of the fibres, the connection between the atrioventricular node and the atrial muscles is interrupted (Fig. 1), His' bundle becomes thinner, and its fibres are elongated. Since the mass of muscles remains unchanged, the gap due to the dilatation is filled with adipose tissue. Cardiac dilatation usually divides the atrioventricular node into several parts and changes its shape. If there exist earlier adhesions between the atrioventricular connective tissue and the substance of the atrioventricular node the conduction fibres will form a widespread loose plexus (Cases 5, 11, 14, 22, 23). If, however, the adhesions are less close, the original pattern of the fibres will remain unchanged but limited to a relatively small area (Cases 4, 6, 7, 12, 15). Fatty infiltration in His' bundle, as observed in Cases 10, 12 and 13 and — to some extent — the atrophy of fibres, are also due to the dilatation. Fatty infiltration is sometimes due to cardiac lipomatosis (Case 10).

Comment

Destruction of fibres in the horizontal part of the conduction system is a usual concomitant of hypertension. The connection between the conduction tissue and the other myocardial fibres may be damaged even without grave degeneration of the conduction fibres. Each of the changes mentioned causes more or less damage to the fibres, but without simultaneous vascular lesions none of them seems to bring about such destruction as to prevent the transference of impulses. On the other hand, a simultaneous occurrence of the said changes involves fibre-destruction on a large scale. The thinned His' bundle is easily destroyed in cases of vascular lesions, while the extended atrioventricular node is more resistant.

II. Left bundle branch

The commencement of the left branch of His' bundle is supplied by the terminal branches of the ramus septi fibrosi. Of its two bundles, the anterior is situated in the area supplied by the left, the posterior in the area supplied by the right coronary artery. The fibres running on the interventricular septum receive blood through the endocardium and the Thebesian veins. There are few capillaries among Purkinje's fibres.

1. *Changes of vascular origin*

It is due to the peculiar metabolism of the left bundle branch that septal infarctions of lesser extent are, as a rule, not accompanied by the disintegration of the conduction fibres at the initial stage of necrosis. Such disintegration may, however, occur when the infarct becomes organized. Atrophy of the fibres may be prevented by certain compensatory factors. A change in coronary circulation and myocardial blood supply is the most significant

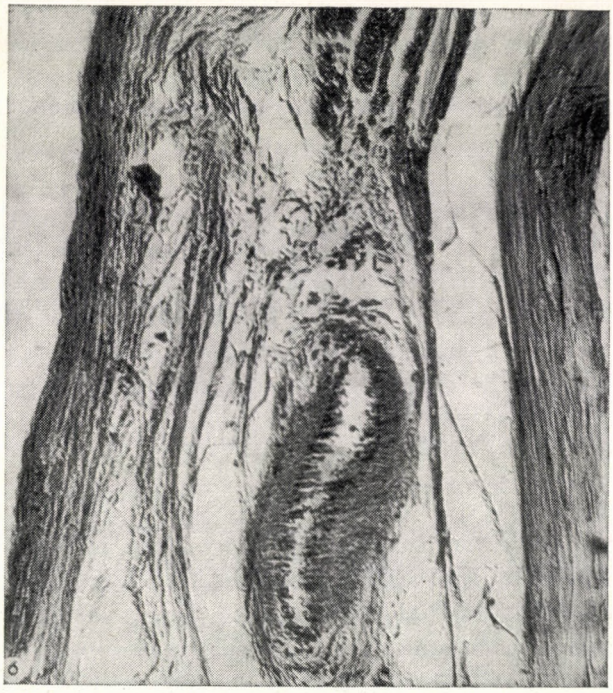

Fig. 6. Case 4. Dilated subendocardial artery; atrophied left bundle branch in scar tissue. Crossmon's trichrome

compensatory factor. These factors are manifested differently in cases of malignant and in those of benign hypertension. Differences in this respect may be summed up as follows:

a) Sclerosis and stenosis of the larger coronary branches cause the subendocardial vessels to distend (Fig. 6). Gradually developing constriction goes hand in hand with the formation of anastomoses. Our histological observations in this respect are supported by FULTON's [3] autoradiographic investigations.

b) Owing to changes in blood supply the musculature between the organizing infarcts and the endocardium, and also the fibres of the bundle branch, may be preserved for long (Cases 13, 14).

c) In cases of malignant hypertension the organizing infarction will soon extend to the conduction fibres (Case 17). The constriction of the larger vessel branches is not compensated by a dilatation of small vessels and capillaries in the subendocardial zone (Case 14).

Irrespective of the necrosis due to malignant or benign hypertension the conduction fibres are destroyed if the septal infarct extends to a surface larger than 2 cm^2.

2. *Changes of environmental origin*

Our observations have confirmed the statement of MAHAIM [7], and ZANCHI and LENÈGRE [10] that the weakest spot of left bundle branch is in its upper third, immediately at its beginning. The fibres of the left bundle branch may, however, be exposed to noxious influences from two directions in its lower portion also: more commonly from the myocardium and less often from the endocardium. Subendocardial fibrosis and infarction have already been mentioned. They are of importance, since the conduction fibres may be invaded by connective tissue, and impended diffusion will induce a deterioration of the fibres. A mural thrombus, due to widespread infarction impairs the blood supply of the bundle branch (Case 14). It is, however, not only owing to mural thrombi but also by the mechanical action of the blood stream (especially in cases of regurgitation) that the endocardium thickens. We observed it in 7 cases of hypertension and in a control case. Rupture of the left bundle branch in Case 4 seemed to have been partially due to a degenerative atrophy of the conduction fibres between two fibrosed layers. Block of the left bundle branch in Cases 11 and 14 had been caused by a rupture of the initial portion where the bundle branch was veritably amputed by the fibrosed muscle tissue and the thickened endocardium. Fig. 4 illustrates a similar, though not so complete, destruction of the fibres.

3. *Changes due to dilatation*

The bundle branches have to adapt themselves to the septal changes caused by cardiac dilatation. The conduction fibres do not perform contractions and are, therefore, saved from becoming hypertrophic [7]. Cardiac dilatation causes the fibres to attenuate. However the parts of the left bundle branch were not uniformly affected by septal dilatation in our cases of hypertension. Attenuation of the fibres was most frequent in the proximal third of the bundle branch, but it must be remembered that fibres in this segment are thin even under physiological conditions. The attenuation in this segment might be one of the following factors.

a) Owing to environmental changes, bundle branches are "fixed" in this area; since they are running almost vertically (the septum is mostly

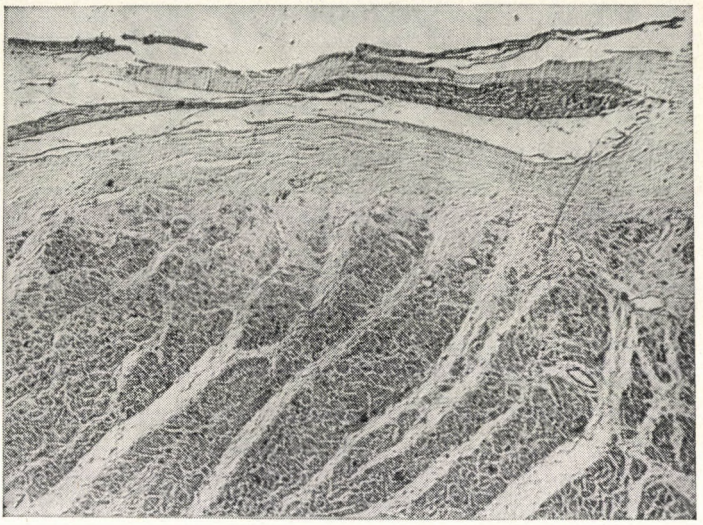

Fig. 7. Case 14. Atrophy of left bundle branch due to cardiac dilatation and fibrosis. Haematoxylin-eosin

elongated in a vertical direction at this point), dilatation affects the fibres severely.

b) The septum becomes not only dilated but hypertrophic as well; its crest on the atrioventricular boundary forms not an acute but an obtuse angle, a veritable shoulder. The conduction fibres are then so much stretched by dilatation that they become atrophied (Fig. 7). The effect of environ-

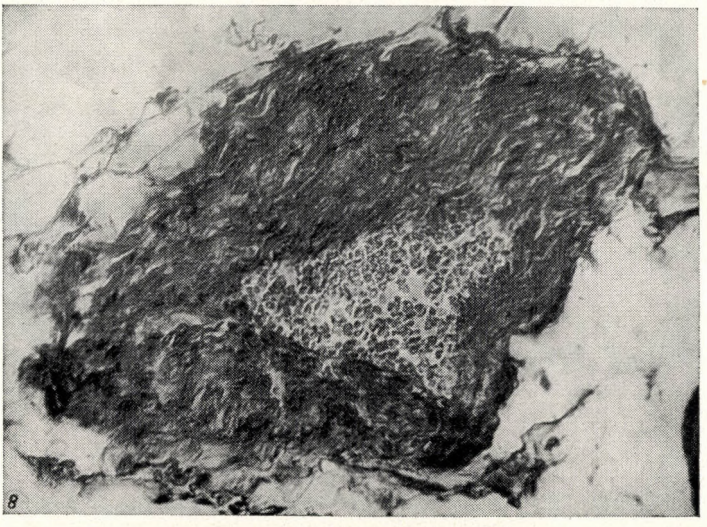

Fig. 8. Case 1. Dilated and ruptured vein in right bundle branch. Horizontal section. Haematoxylin-eosin

mental factors being most marked precisely in the upper third of the bundle branch, it appears doubtful whether atrophy is really due to dilatation. However, in four of our cases—in which cardiac dilatation was pronounced—we found atrophy associated with fatty degeneration in all portions of the left bundle branch.

Comment

The most vulnerable point of the left bundle branch is in its upper third. Conduction fibres in this segment are effected by septal fibrosis, thickening, fibrosis, and hyalinization of the septum membranaceum, further by atrophy due to septal dilatation, and hypertrophy. The lower portion of the left bundle branch may be damaged from two directions: the fibrosis of organizing infarctions may spread from the myocardium to the subendocardial connective tissue, while — from the other side — transendocardial diffusion may be impeded by a thickening of the endocardium, the degeneration of its connective tissue, as well as by mural thrombi. Serious cardiac dilatation induces degenerative atrophy in the lower portion of the bundle branch. If coronary lesions extend to the larger branches, and if conditions are favourable for the formation of collaterals, the fibres of the bundle branch are protected against premature degeneration by the newly-formed subendocardial vascular network. Myocardial infarctions causing ECG-changes achieve importance if the necrotic area lies below the upper or middle third of the left bundle branch where the fibres still form circumscribed bundles.

III. Right bundle branch

Its upper third is supplied by the ramus septi fibrosi, while the vessels in the two lower thirds originate from the ramus descendens anterior. One or two small arteries and veins run along the conduction fibres. Certain authors distinguish the small artery in the moderator band by a special term: ramus limbi dextri.

1. *Changes of vascular origin*

It follows from its anatomy and localization that the right branch of His' bundle is more exposed to the influence of arterial and especially arteriolar changes than other parts of the conduction system. Even small necroses at any point of the right bundle branch may cause rupture without being accompanied by serious alterations in the surrounding muscles (Case 22). Formation of collaterals may prevent the degeneration of the fibres even if the ramus

descendens anterior is occluded (Case 5). Vascular dilatation is nevertheless accompanied by some necrosis of the fibres, and distended veins may burst (Fig. 5).

2. *Changes of environmental origin*

Fibrosis of the muscles around the upper third of the right bundle branch is a frequent occurrence (Cases 4, 6, 13, 15, 19, 24) and it spreads to the bundle branch itself which is anyhow thin in this segment. Progressive fibrosis of the septum fibrosum may invade in the same way as it does the left bundle branch.

In two cases (8 and 9) the arising of fibrous connective tissue was observed around the right bundle branch beside the base of the middle cusp of the tricuspid valve. The peripheral conduction fibres of this annulus showed signs of degeneration, a phenomenon not peculiar to hypertension since it occurs in normal hearts as well.

The frequency of infarcts increases towards the apex of the heart which offers a wider scope for a destruction of the lower third of the right bundle branch.

In three cases of hypertension, where also the right side of the heart was dilated, we observed with the naked eye deposits of fat in the moderator band (Cases 3, 7, 14). There was fatty degeneration in the conduction fibres and fatty infiltration around them. We attributed these alterations to the mechanical action of the anterior papillary muscle: the two muscles must have collided during the heart contractions.

3. *Changes due to dilatation*

The number of fibres in the right bundle branch varies, according to MÖNCKEBERG [8], not only from individual to individual but also from one segment of the branch to the next. Our observations, on the other hand, showed that great segmental differences in the number of fibres are always a pathological sign. If the right half of the heart dilates (Cases 12, 13, 17, 19), attenuation and atrophy of the fibres are more pronounced and (as in the left bundle branch) especially so in the upper third.

Comment

The right bundle branch forms a tenuous band which can be ruptured by necroses of microscopic size caused by arteriolar occlusion. Collateral circulation may ensure the blood supply of the right bundle branch under favourable conditions. Vascular lesions are not necessarily limited to the

arteries: right bundle branch block was due in one of our cases to the rupture of a vein. The upper third of the right bundle branch becomes tenuous and its fibres undergo atrophy in consequence of dilatation. Fibrosis around this segment was found to be more pronounced. A ring of connective tissue around the bundle branch, in the vicinity of the tricuspid valve, was observed in a case of hypertension, as well as in a control case. The mechanic action of the anterior papillary muscle may induce fatty degeneration in the lower third of the right bundle branch.

ECG and morphological changes

It follows from the above that conditions of hypertension are usually accompanied by injury to the septal conduction system. However, partial lesions of the conduction system are not indicated by the electrocardiogram. Although it is generally recognized that a bundle branch block is preceded by the destruction of all fibres in the affected area, we have found, that block may occur in cases of acute damage even if the destruction of fibres is not complete (Cases 1, 22). Block may be clinically diagnosed when the muscles cease to function even if the conduction fibres are more or less unimpaired in the affected area. The fibres above the infarct in the posterior wall had not been damaged in Case 21, whereas the fibres running to the intact muscles were destroyed by fibrosis. This observation agrees with that of LUMB et al. [6] made in the course of experiments in dogs. Our Case 19 justifies the assumption that atrioventricular dissociation and alternating branch block may be caused by coronary sclerosis (insufficient blood supply), myocardial fibrosis, or the organizing infarct of the sinoatrial node. It is evident from Case 14 that even multiple damages of the conduction system are indicated by precordial electrocardiograms.

Our investigations have failed to prove convincingly that a partial interruption of the connection between conduction fibres and atrial muscle-fibres provokes a change in the P Q interval. It may remain normal (Cases 22 and 24) or become shorter (Case 9) in association with focal necroses, or with a fibrosis and vascular dilatation in His' bundle. We observed the said partial interruption and a serious injury to His' bundle in 8 cases; in 3 only did the P Q interval exceed 0.20 sec. We do not attribute the existence of accessory muscular bridges for atrioventricular transmission (Wolff-Parkinson-White syndrome, Case 6) to the presence of the bundle of Kent but to fibrosis affecting the right bundle branch. Although the aberrant fibres known as the bundle of Kent occurred in several subjects (Cases 1, 4, 17, 23), their presence was associated with the Wolff-Parkinson-White syndrome in a single case only. It should be noted that the bundle of Kent may become disabled by the layer of connective tissue beside the middle cusp of the tricuspid valve which separates it from the ventricular muscles.

We have examined our material also with a view to ascertaining the most severely affected point of the conduction system in cases of coronary lesions or repolarization disturbances indicated by the ECG. Such changes occurred in all of our 18 cases with hypertension. It is the left bundle branch which should be regarded as the most vulnerable in conditions of hypertension. We had 8 such cases (one of them among the 6 controls); they included 2 cases where the right bundle branch was similarly affected (33—34 per cent). The next frequent damage was that of the right bundle branch with 5 cases, including one where also the left bundle branch was destroyed (22—27 per cent). Damage of the His' bundle was found in 4 cases, including one in which also the atrioventricular node and one in which also the left bundle branch was similarly impaired (11—22 per cent). Advanced degeneration of the fibres of the atrioventricular node was observed in 2 cases, in one of which His' bundle seemed to be similarly damaged (5—11 per cent).

Since our material consisted of selected cases the results presented do not justify definite conclusions. From the point of view of everyday practice it is nevertheless safe to say that, in cases of hypertension the atrioventricular node and His' bundle show the greatest resistance to injury while a serious lesion of the bundle branches has always to be reckoned with, even if clinical methods do not reveal disturbances in the conduction system.

Summary

The morphological changes in the septal conduction tissue of the heart have been studied in subjects died of hypertension, and 6 non-hypertensive control cases (selected according to age groups). The material included 1 case of alternating atrioventricular block, permanent atrioventricular block and Wolff-Parkinson-White syndrome, 3 cases of left and 2 cases of right bundle-branch block. Especial attention has been paid to the role of vascular, environmental and mechanical factors in the development of morphological changes.

Changes observed in Aschoff-Tawara's node, in His' bundle and the bundle branches have been separately discussed.

A comparison has been made between clinical and morphological changes and the possible conclusions have been discussed.

* * *

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ДАННЫЕ К ПАТОМЕХАНИЗМУ НАРУШЕНИЙ ПРОВОДИМОСТИ СЕРДЦА ПРИ ГИПЕРТОНИЧЕСКИХ СОСТОЯНИЯХ

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У 18 больных, страдающих гипертонией, и у 6 лиц различных возрастных групп без гипертонии, исследовалось развитие морфологических изменений при нарушении межжелудочковой проводимости. В материале обсуждается только один случай переходной атриовентрикулярной блокады сердца, сопровождающейся перемежающей блокадой правой и левой ножек Т., один случай постоянной атриовентрикулярной или двусторонней блокады ножек, один синдром WPW, 3 случая блокады левой ножки Т, и 2 случая блокады правой ножки Т. При изложении развития морфологических изменений авторы уделяют особое внимание роли изменений сосудистой системы и окружающей среды, а также роли механических факторов. После описания отдельных деталей вкратце резюмируются изменения узла Ашофф—Тавара и гисового пучка, и наблюдаемые в правой ножке Т. альтерации. Сопоставляются клинические и патогистологические изменения, и по мере возможности извлекаются из них клинические и морфологические выводы.

BEITRÄGE ZUM PATHOMECHANISMUS DER HYPERTONISCHEN ÜBERLEITUNGS STÖRUNGEN

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Bei 18 hypertonischen und 6 nicht hypertonischen, zu verschiedenen Altersklassen gehörenden Personen wurde die Entwicklung der morphologischen Veränderungen bei septalen Überleitungsstörungen untersucht. Neben einem Fall von vorübergehenden atrio-ventrikulären Block mit weschselndem rechten und linken T-Schenkelblock wurden ein Fall von konstanten atrio-ventrikularem bzw. bilateralem Schenkelblock, ein Fall von WPW-Syndrom, 3 Fälle von linken T-Schenkelblock und 2 Fälle von rechten T-Schenkelblock untersucht.

Der Rolle der vaskulären und Umgebungsveränderungen, sowie der mechanischen Faktoren wurde besondere Aufmerksamkeit gewidmet. Die im Aschoff-Tawaraschen Knoten im Hisschen Bündel, sowie im rechten T-Schenkel beobachteten Veränderungen wurden im einzelnen besprochen, die klinischen und pathohistologischen Veränderungen miteinander verglichen, und manche klinische und morphologische Folgerungen gezogen.

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