ON A
DISSEMINATED FIBROSIS OF THE
KIDNEY

FOUND IN
CASES OF INFECTIVE ENDOCARDITIS, AORTITIS, AND
ALLIED DISEASES,
ITS PATHOLOGICAL SIGNIFICANCE, AND CHIEF CLINICAL
FEATURES.

BY
W. AINSLIE HOLLIS, M.D., F.R.C.P.,
PHYSICIAN TO THE SUSSEX COUNTY HOSPITAL.

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THE following kidney disorders, excluding those obviously due to embolism, I have found associated with infective endocarditis :

1. The tough congested kidney, wherein a general venous engorgement of the whole organ is the chief if not the only alteration visible.

2. The large mottled kidney. In this case the organ is tough and misshapen, and its congested surface is relieved by well-defined elevated patches of pale granular tissue. On section, the mottling is found to extend throughout the

thickness of the organ; portions of the pale material protrude into the pelvis, and consist mostly of tubules blocked with swollen and degenerated epithelium. Such kidneys occasionally secrete an inordinate quantity of highly albuminous urine. One of my cases passed upwards of 100 oz. daily for several successive days; and on one occasion the water contained 1.55 per cent. of albumen.¹

3. The small mottled kidney. Here also the organ is misshapen, and usually mottled, pink and white. Extreme toughness, small size, and puckered surface are its chief peculiarities. This type of kidney secretes little water. The urine is of a low specific gravity, and slightly albuminous.

My contention is that these varieties of kidney degeneration, at all events the two last types, are different phases of the same progressive disease.

The distinguishing feature of this disease is the presence of scattered groups of nuclear-like bodies in the loose interstitial tissues of the affected kidney, especially around the smaller arterioles and the Malpighian tufts. These bodies are deeply stained by many aniline and other dyes; and to their presence in the renal tissues the subsequent degeneration of the organ can with some probability, as I shall subsequently show, be traced. Associated with these kidney changes, we frequently find more or less extensive disease of the aorta and of the cardiac valves. We shall also find in these cases, if we search for them aright, groups of similar nuclear-like bodies between the inner and middle coats of that artery or beneath the endocardium. I have also found them, deeply stained as usual, in the adventitia and around the *vasa vasorum* of the aorta.

On the other hand, acute endocardial mischief is sometimes absent in cases of disseminated renal fibrosis, although evidence of old-standing cardiac trouble in the thickened and opaque cusps is mostly present. Aortitis,

¹ Dr. Adolphus Richardson (adopting Brandberg's method) kindly made this estimate for me.

as noticeable to the naked eye in discoloured patches of atheroma, is constantly found, and as often overlooked. In connection with this subject I may remind my audience that the great pathologist whom we have recently seen amongst us, Rudolf Virchow of Berlin, drew attention nearly five and thirty years ago to the relationship which exists between the processes leading to arterial atheroma on the one hand, and to endocardial degeneration on the other ('Cellular Pathology,' Eng. edit., p. 362).

The nuclear bodies, their size and shape; other characters.—The nuclear bodies are small oval or roundish bodies, variable in size, but for the most part less in diameter than is a blood-corpuscle. In the densest groups the smallest sized bodies are seen, usually consorting in twos or threes within apparently one cell, while the larger ones are generally solitary and frequently without any obvious cell membranes around. Under a high power (Zeiss F, oc. 4, Abbé's condenser) they are found to consist, after staining, of two portions, namely of numerous deeply-stained granules embedded in a pale hyaline plasma. Although these bodies are found for the most part in the loose connective tissue surrounding the vessels of the regions they frequent, and although I believe that by their presence in this tissue they are directly responsible for the kidney degeneration to be more fully described elsewhere, yet they are rarely seen in the thickened fibrous tissue of Bowman's capsules or in the hypertrophied walls of the arterioles. They appear to require elbow-room to grow and thrive in, as do their unfortunate hosts.

The kidney degeneration.—The morbid changes observable in the renal structures as the outcome of this disease may be conveniently placed under two headings, according as they are apparently the result of mechanical or of purely pathological processes. In considering the mechanism of urinary secretion, prominence must necessarily be given to the renal arteries and their branches for the part they take in regulating the supply of blood by means of their muscular coats to the varying require-

ments of the organ. But the alternate dilatation and contraction of a tubular vessel necessitate a more or less rapid change of its calibre. The renal arterioles in order to permit this variation in their sectional area are embedded in a delicate elastic tissue which readily yields to any change in size or shape. When, however, the resiliency of the perivascular tissue is impaired at various points by the introduction of rapidly proliferating corpuscles amongst its meshes, an early result must be the development of corresponding points of muscular weariness in the embedded vessel, followed by a more or less continuous diastole. As a consequence of the collapse in the system which regulates the local blood-supply, we find areas of vascular engorgement developed frequently throughout this disease. After a time in many cases the affected vessels undoubtedly recover their regulating powers at the expense of their muscular coats. Meantime, however, other groups of nuclear bodies are heaped around the necks of the Malpighian corpuscles, or are scattered over the membrane covering a tuft, or, again, pervade a Bowman's capsule. This sudden invasion of the kidney substance—for, I take it, the evidence, both clinical and pathological, points to a succession of explosive outbursts in this disease followed by periods of comparative calm—this sudden invasion of the kidney substance, I repeat, by these pathological entities must, and speedily does, entail secondary changes, both organic and functional. And so we find districts of fibrous hyperplasia developed, containing thickened capsules and shrunken tufts, and adjoining others wherein the glomerules are apparently healthy. Hypertrophy of the muscular walls of the interlobular arteries and their branches follows, partly in consequence possibly of these vascular changes, partly from the increasing thickness of the adventitia. These abnormal circulatory conditions are subversive of the orderly adjustment of the blood-supply of the kidney to the demands made upon it, and as a result we meet with evidences of over-stimulation of the epithelium, clinically

in an excessive secretion of albuminous urine, pathologically in considerable masses of pale parenchymatous tissue, consisting mostly of swollen and dilated tubes choked with epithelial *débris*. As the disease advances, or whenever the nuclear bodies become generally dispersed throughout the interstitial tissues, a condition arises similar to that which leads to the contracted granular kidney of Bright. I need not here recapitulate the minute changes of the organ under such circumstances, as they have been repeatedly discussed at the Society's meetings by many illustrious Fellows, notably by Sir George Johnson. I may, however, be permitted to mention that the twenty-eighth volume of the 'Transactions' contains a paper by Sir James Paget on the coincidence of granular degeneration of the kidneys with old clots in the pulmonary arteries.

The disease of the endocardium and aorta which so often accompanies disseminated fibrosis of the kidney consists essentially of nests of nuclear bodies similar in size, in shape, and in physical properties, as far as I can ascertain, to those located in the kidneys. They are found beneath the inner coats of the heart and artery respectively, and they undoubtedly give rise to the structural degenerations so fully described by Virchow (*loc. cit.*). I have, as before mentioned, found the nuclear bodies in the adventitia of the aorta.

The question how these morbid units are conveyed, say from the aortic intima to the renal tissues, if we admit these pathological processes to be dependent the one upon the other, is a difficult one to answer. I imagine that they are conveyed directly by the blood either within a leucocyte or separately, and that they subsequently select a favorable site to penetrate the walls of the renal vessel. I feel assured that the nuclear body is quite capable of passing through the comparatively thin-walled arteriole, as I have found them occasionally between the layers of the tough elastic coat of the aorta. Are the nuclear bodies when present in kidney disorders the *fons et origo*

malorum? If we are to believe the essence of this disease to be particulate, I think that they are so. But whether the virus lodges within the nuclear bodies themselves, or only in the highly-stained granules, I shall not venture to decide. I have been unable to find any bacilli, either stained or unstained, among the affected tissues, although fibrillar irregularities in a certain light can produce rod-like appearances which might readily be mistaken for the organisms mentioned.

Finally, as regards the chief symptoms observable in the disease. One of the most important is hæmorrhage from a mucous surface: bleeding from the nose or mouth, hæmoptysis, hæmaturia, and rectal hæmorrhage are commonly met with. The pulse tension is usually *plus*, and the heart is hypertrophied. Valvular incompetence can possibly modify this condition. Anæmia is generally severe. One of my eleven cases had only 24 per cent. of the normal number of blood-corpuscles in his vessels. Repeated short attacks of unconsciousness, with and without rigidity of the muscles, occurred in three cases. Clots in the veins occurred in two, in one of which the right subclavian was blocked. Temperature was variable.

APPENDIX.

In the following notes the cases have been arranged, as near as may be, with regard to the kidney-changes seen in the organs after death.

CASE 1. *Congestive mottling of kidneys; early stage.*—Arthur H—, æt. 11, a schoolboy residing at Brighton, was admitted to the Sussex County Hospital on July 14th, 1893, with symptoms of recurrent appendicitis. Present attack began a fortnight previously with pain in the abdomen. Had a similar attack at the beginning of the year, necessitating his absence from school for several days. On admission patient was collapsed; face pinched; abdomen distended; tongue dry and furred; bowels not

relieved for four days ; constantly vomiting faecal matter. In the afternoon Dr. Furner opened the abdomen and discovered adhesions in the neighbourhood of the appendix with strangulation of a loop of intestine. This was relieved, and the bowels were with difficulty replaced. The patient died about fourteen hours afterwards. At the autopsy the appendix was found to be ulcerated through, about half an inch from the caecum. The free end was the seat of an abscess, behind the caecum and shut off from the peritoneal cavity by adhesions. The aorta was striated just beyond the valves with patches of atheroma. The kidneys were somewhat tougher in texture than natural. Capsules separated readily. There was much congestive mottling of the surfaces of the organs.

I have added the above case to those already collected before writing this paper as the microscopic sections of the aorta and kidney show the nuclear bodies very distinctly, and what I take to be the earliest changes in the renal structures due to their presence.

CASE 2. (This case has also been added since the paper was written.)—H. R—, a barber, æt. 21, residing at Brighton, was admitted on June 6th last, also suffering from abdominal symptoms. Five days ago, shortly after eating some whelks, he was seized with pain in the right iliac fossa, which soon spread over the whole abdomen. Tenderness was well marked. For two days before admission he had frequent vomiting. Constipation had persisted during the attack, notwithstanding doses of jalap and senna. He had a similar attack last January, which lasted five weeks. Family history was good. On admission the temperature was normal. Tongue dry and furred. Abdomen distended and tympanitic, with well-marked tenderness generally. Respirations thoracic in character. Heart's apex-beat one inch outside nipple-line in the fourth space ; some roughness with the second sound. Basal aortic sounds feeble. Urine sp. gr. 1035, acid, no albumen.

During the next two days the youth remained mostly in the same condition. The abdominal tenderness, however, became localised over the right iliac fossa, and the temperature was usually a degree or two above normal. The bowels were not moved, although he passed flatus *per rectum*. On the evening of the 9th he was suddenly seized with severe pain about the navel, with vomiting. The bowels were opened twice, motions formed and dark coloured, no blood. The pain gradually became more severe; the next morning collapse set in. The pulse rose to 152, thready in character. Temperature 103·2°. The patient's intellect continued clear throughout; he did not consent to an exploratory operation recommended by the staff. He died that afternoon. At the autopsy pus was found in the abdominal cavity, and there were numerous adhesions between omentum and the coils of intestines. The extremity of the appendix had sloughed away, and an opening was found passing into the cæcum, and partly blocked by an intestinal concretion. The left ventricle of the heart was hypertrophied. No valve disease noted. There was a considerable patch of atheroma in the aorta. The capsules of the kidneys were somewhat adherent, and their substance congested and tough.

Nuclear bodies could be seen between the inner layers of the elastic coat of the aorta forcing it inwards.

CASE 3. *Tough congested kidneys*.—I. W. B—, æt. 67, a retired soldier, had been ailing for many months and on two previous occasions an inmate of the Sussex County Hospital with similar symptoms. Admitted February 1st, 1893. He had been almost continuously in bed for the last five months with shortness of breath, cough, and swollen legs. No history of syphilis or of acute rheumatism. On admission the patient's temperature was subnormal. Face and extremities cyanosed. Urine acid, no albumen. Heart's apex in fifth space, half an inch outside the nipple line. Cardiac dulness absent. No bruits audible. Lungs emphysematous; rhonchi general

over the back ; both bases fairly clear. Some œdema of the dorsal integument. Abdomen tense and resonant. No visceral enlargement detected. Legs hard, red, and œdematous. Penis much distended with œdema. Passed urine with difficulty, and frequently. During his stay in hospital the lividity of his face increased, the œdema became more marked, and the temperature remained subnormal. The heart's sounds meanwhile were very feeble and indistinct, and the dyspnoea was distressing. The patient died somewhat suddenly twelve days after admission.

At the autopsy the lungs were found to be emphysematous, and overlapped the heart. The latter organ was both dilated and hypertrophied. The valves of the right cavities were thickened. On the left side the mitral was greatly thickened, and the aortic cusp calcified at its attachment. The aortic valves were converted into a calcareous ring with a slit in the centre, through which water could pass in either direction with the same facility. On the endocardium below, and on the aorta above, were atheromatous patches covered with vegetations. The kidney cortex was diminished in amount, and the capsules stripped fairly well. On section the whole organ was seen to be intensely congested, and it was tougher than natural.

Numerous groups of nuclear bodies were visible in the section of the aorta.

CASE 4. *Large mottled kidneys*.—Fred K—, an errand boy, æt. 16, was admitted to the Sussex County Hospital on November 8th, 1892. Two years ago had a small impacted stone removed from the urethra ; subsequently had rheumatic fever with pericarditis (?). Had been short of breath since that time. On admission he was found to be suffering from pains in the joints without perceptible swelling, and from much sweating. Heart's apex felt half an inch outside nipple line in fifth space. A soft systolic murmur at apex, heard towards axilla ; and a to-and-fro

sound at base over aortic cusps. During his stay in hospital he had several attacks of epistaxis. The temperature was mostly 100° and upwards. The urine was very albuminous, sp. gr. about 1020; he passed from 60 to 80 oz. daily for some weeks. He had general œdema of the integuments, and was tapped twice for ascites. He died on February 15th, 1893.

At the subsequent examination the following conditions were noted: The body was fairly developed. There was general anasarca. Pleuræ contained two or three pints of clear serum, and the lungs were much collapsed. Heart weighed 17 oz. Pericardium universally adherent; cardiac cavities both hypertrophied and dilated. On one of the tricuspid flaps was a small white nodule with roughened surface, and the adjacent ventricular surface was coated with a small rough fibro-cartilaginous patch. On the left side both sets of valves were roughened and ulcerated along their free borders; there were also patches of roughened endocardium at the position of the foramen ovale and along the ventricular walls. The kidneys together weighed 7 oz. They were much mottled. The left was very small.¹ Cortex narrowed and remarkably white in parts. Capsule stripped readily. The right was considerably enlarged. Capsule peeled readily. The section presented several white patches of tissue extending from the cortex to the pelvis, into which some of them projected, seemingly distinct from the rest of the substance. These patches consisted mainly of dilated tubules filled with swollen epithelium and other detritus.

CASE 5.—Wm. S. T—, æt. 37, was admitted into the hospital on November 23rd, 1892, in a semi-conscious state. He had suffered from pain in the head and back, with hæmaturia and vomiting, for five weeks. Present illness came on after exposure. Previous health good. On admission the temperature was subnormal. Pulse 72, cord-like. Respirations 16, long and deep. He answered a question

¹ Probably a congenital condition.

when roused. Colour of skin good, no cyanosis. Pupils equal, somewhat dilated. He could move his limbs. No muscular twitching. Heart's apex half an inch outside nipple line in fifth space. Prolonged systolic murmur at apex. Reduplicated second sound at base. Lungs clear throughout. Urine sp. gr. 1012, alkaline, very albuminous; on one occasion it contained 1.55 per cent. albumen. An excessive quantity was passed; once it reached 116 oz. in the twenty-four hours. The patient complained of severe occipital headache with increasing dimness of sight. There were recurrent attacks of vomiting, and of rectal hæmorrhage. During his stay the head symptoms became more prominent. There was loss of sight in left eye with atrophy of disc, and paresis of rectus externus. The blood contained at this time only 24 per cent. of red corpuscles. Lung symptoms were subsequently developed; a patch of consolidation appeared at the upper part of left, and another at lower part of right lung. No tubercle bacilli were met with in sputa. The legs became œdematous through blocking of the saphenous veins a few days before death, which occurred on March 6th following. At the autopsy discrete glistening white bodies about the size of pins' heads were scattered over the pia mater of hemispheres and choroid plexuses. The left upper lobe was sharply marked off from the rest of the lung; it was of a light grey colour, consolidated and airless; bands of fibrous tissue could be traced from the surface spreading through the lobe. The substance was, notwithstanding, somewhat soft and friable. In the right lower lobe were two or three cavities. Heart weighed 22 oz.; cavities both hypertrophied and dilated. The tricuspid orifice admitted five fingers. Valves healthy. The mitral curtains contained patches of thickening, and their edges were also thickened. Aortic valves were healthy. There were many patches of atheroma along the commencement of aorta. The kidneys weighed together about 12 oz. Their contour was misshapen. Their surfaces were mottled with pale and congested

patches. The capsules stripped easily. The pale blotches were raised above the surrounding surface, and they extended into the substance of the organs. The bases of pyramids were frayed out. The kidneys were very tough and fibrous. The omentum and the capsules of the liver and spleen were adherent to the abdominal walls. Groups of nuclear bodies are disposed around the thickened renal vessels. The uriniferous tubules were in various stages of degeneration.

CASE 6. *Large pale kidneys*.—James B—, æt. 62, a labourer, residing at Brighton, was admitted September 2nd, 1892. He was then suffering from diarrhœa, vomiting, and swollen legs. The illness began two months ago. Had recently passed a large quantity of water daily. Eyesight good. Both his previous and his family histories were good. On admission very chloræmic; hæmoglobin 20 per cent. Some dyspnœa; legs cedematous. No hæmorrhages. Heart's apex in normal position. Systolic murmur heard at apex, conducted towards axilla; also a systolic murmur heard over the aortic orifice and upwards. Breathing heard faintly at lung bases. Bowels open four or five times daily. Urine sp. gr. 1012, no albumen. Passed rather more than usual quantity; on one occasion 43 oz. in the twenty-four hours. Temperature about 99·0°. He was discharged on September 26th, 1892, improved in health. On October 24th last he was readmitted to the hospital. For three months his breath had become shorter, and the legs and arms had swollen more than usual. Sight was failing. No history of rheumatism; he stated that he had had two or three attacks of jaundice (?). On admission, very sallow and chloræmic; no jaundice. Temperature normal. Pulse regular, of high tension. Tongue pale and flabby. Heart's apex in fifth space in nipple line. Cardiac dullness absent. Prolonged apical systolic murmur, conducted outwards. Rough systolic murmur over pulmonary cusps. Bases of lungs clear; some emphysema and rhonchi.

No enlargement of liver or spleen. Legs slightly œdematous. Urine contained no albumen. He died two days subsequently. At the post-mortem examination on October 28th a considerable thickness of subcutaneous fat was found. There was some fluid in either pleura; and two or three roundish bodies, very hard and calcareous, and about the size of small peas, were attached to the membrane. The central tendon of diaphragm contained a plate of bone about three inches in diameter. The cartilage rings of the lowest fourth of trachea and at the commencement of large bronchi were calcified. Heart weighed 20 oz. It was much dilated, especially the right ventricle. The left was also much hypertrophied. Muscular tissue pale and friable. Tricuspid, mitral, and aortic valves were all thickened. The chordæ tendineæ of left ventricle were also thick and short. There were many patches of atheroma on the endocardium and along the aorta, mostly of the circinate variety. At the upper part of the aortic sinus was a concavity about half an inch deep, with atheromatous walls. Liver small; capsule thickened. Kidneys together weighed 1 lb. Pale and very tough. Capsules thick and adherent, left the surface granular; many cysts in cortex. Papillæ shortened. A large amount of fat surrounding pelvis. Nuclear bodies were to be seen in the aortic intima, and in the renal connective tissue.

CASE 7. *Small granular kidneys*.—James K—, stableman, æt. 54, was admitted to hospital February 17th last. He had suffered for six months from vomiting and indigestion, and from frontal headache. Eyelids and face had swollen in the morning recently. Eyesight had become dim. Some cough. Previous history good. No history of syphilis. When first seen the temperature was subnormal, and the pulse tension high. Heart's apex-beat in sixth space in nipple line. Soft systolic murmur at apex; second sound accentuated at base. Cardiac dullness absent. Scattered rhonchi over anterior chest; crepi-

tation at the left pulmonary base behind. Edge of liver almost down to navel. Urine sp. gr. 1012, acid, albumen 0·12 per cent.; passed 24 oz. during first twenty-four hours. During his stay in the institution he had several short fits of unconsciousness, muscles limp, no stertor or convulsions. The urine diminished in quantity as time progressed; once only 6 oz. were passed in twenty-four hours. Bleeding from the mucous membrane of mouth continued, with short intermissions, for the last ten days of life. About the same time lung symptoms became urgent. Crackling crepitant sounds, possibly due to friction, were heard over lower half of right lung. The day before death, on March 10th, the temperature rose to 100·1°, otherwise it was mostly subnormal. The autopsy revealed consolidation of the upper lobe of the right lung, its surface covered with shreds of lymph. On section a cavity, the size of a pigeon's egg, was discovered at the right apex, containing whitish friable material. The fibrous tissue was much increased throughout the lobe. Its texture was tough and airless; and its colour, a light grey, separated it sharply from the middle lobe. The rest of the lungs were œdematous and congested; some collapse at bases. Heart weighed 13 oz. Very flabby, pale, and dilated. Muscular fibres fatty. Both auricles much dilated. Tricuspid and mitral valves thickened and granular along their free edges. Aortic valve thickened, and a small vegetation on one cusp. Coronary arteries atheromatous. Just beyond the central cusp of aorta, and impinging on the orifice of coronary artery, was an atheromatous ulcer, the size of a shilling. It presented a fatty-looking base, with calcareous deposit just beneath the surface. The ulcer seriously obstructed the entrance of the artery. There was a second smaller ulcer about an inch onwards, and a number of calcareous plates, about the size of threepenny bits, for some inches along the arch. The kidneys were very small. Mottled pink and white. Capsules thickened. Surface pitted and granular. The granules were large and of a lighter colour than the

intermediate portions. Numerous cysts were scattered over both organs.

Nuclear bodies were seen in the aortic adventitia. In kidney sections compressed tubules adjoined dilated ones. Many nuclear bodies were duplicate.

CASE 8.—X. Y—, æt. about 40, a ticket collector on the Electric Railway, was brought to the hospital in a comatose state. He died shortly after admission. There was a history of a sudden fit. The brain was extensively lacerated by a large hæmorrhage into its substance. The arteries at the base were very atheromatous. Small patches of atheroma at commencement of aorta. Heart muscle hypertrophied. Valves thickened in parts, no growths along edges. Kidneys small and granular. Capsules adherent. Tissues tough.

Nuclear bodies can be seen scattered through the aortic coats, at the seat of an atheromatous patch. Some bodies are apparently about to escape from the inner surface of the intima into the blood-stream.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 25.)

DESCRIPTION OF PLATE II.

A Disseminated Fibrosis of the Kidney found in cases of Infective Endocarditis, Aortitis, and allied disease (W. AINSLIE HOLLIS).

FIG. 1.—A portion of an aortic cusp, adjacent to a *corpus Arantii*, affected with atheroma circinatum, early stage. Case 6. Magnified about 5 diameters.

FIG. 2.—A portion of descending aorta, with atheromatous ulcer. The light parts were raised above the general surface of the endothelium. Case 7. Magnified about 4 diameters.

FIG. 3.—The upper part of the great aortic sinus, affected with ulceration due to atheroma circinatum. The endothelium was absent from the deeply-shaded portions of the ulcers, the edges of which were raised and everted. Case 6.

FIG. 4.—The adventitia of aorta with the openings of some vessels, showing the grouping of nuclear bodies around the vasa vasorum in atheroma. $\times 540$.

FIG. 5.—Section of small mottled kidney, associated with multiple aortic ulceration. Case 7. A A, Tubules with epithelium swollen and granular. B, Malpighian tuft, with numerous nuclear bodies scattered over its surface. C C, Thickened interstitial tissue, with deeply-stained nuclear bodies within its meshes; a group of the latter at D. $\times$ about 600.

FIG. 6.—Section of Malpighian body in congestive mottling of kidney; early stage. Nuclear bodies, mostly apple-pip in shape, are grouped within the membranes of the tuft. Two or three are visible in the space between the tuft and capsule. Case 1. $\times 540$.

FIG. 7.—Sections of atheromatous aortæ, showing nuclear bodies *in situ*. A A', Inner surface of the aorta in Cases 1 and 8 respectively. At B a nuclear body is to be seen partially within the lumen of the vessel. In Case 1, that of a boy, the nuclear bodies are apparently more rounded in shape than they are in the adult, Case 8. The greater resiliency of the younger tissues possibly explains this difference of shape. $\times 540$.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.