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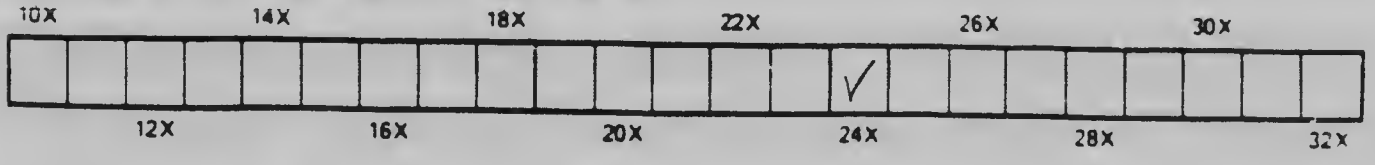

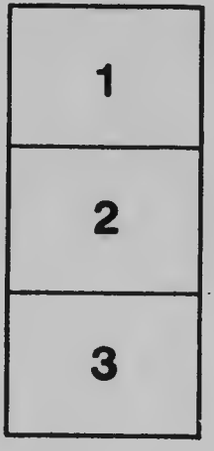

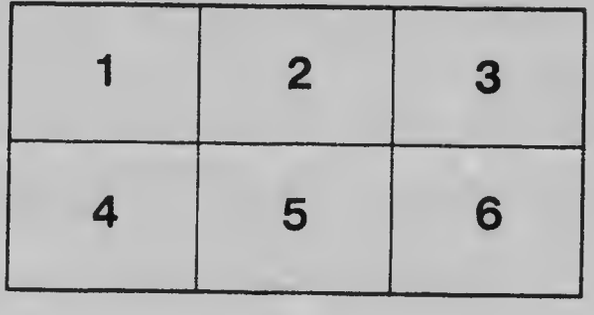

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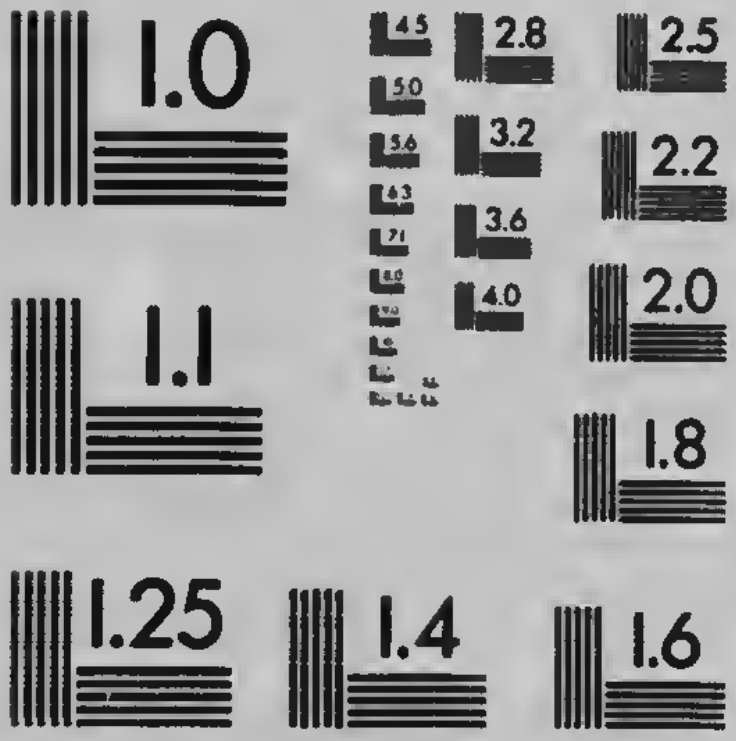

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ON A GIANT-CELLED RHABDOMYO SARCOMA FROM THE TROUT.

BY

J. G. ADAMI, M.A., M.D., F.R.S.
(From the Pathological Laboratory, Royal Victoria Hospital.)

UNILATERAL CONGENITAL ABSENCE OF THE PAIRED GENITO-URINARY ORGANS.

BY

JOHN McCRAE, M.B., M.R.C.P.,
Lecturer in Pathology, McGill University, Montreal.

EXPERIMENTAL "WORK-ARTERIOSCLEROSIS."

AND

TRYPANOSOMES IN MONTREAL RATS.

BY

OSKAR KLOTZ, M.D.,
(From the Pathological Laboratory, of the Royal Victoria Hospital.)

REPRINTED FROM THE MONTREAL MEDICAL JOURNAL, MARCH, 1908.

ON A GIANT-CELLED RHABDOMYO SARCOMA FROM THE TROUT.

BY

J. G. ADAMI, M.A., M.D., F.R.S.

(From the Pathological Laboratory, Royal Victoria Hospital.)

The subject of tumours derived from striated muscle is one that is still involved in considerable uncertainty. French pathologists of the present time, more particularly, report numerous cases of sarcoma which they regard as directly derived from muscle tissue elements, but this view is not by any means universally accepted. It may be laid down as a general rule that the more highly differentiated a tissue, the less is its tendency to afford neoplasms. When we encounter indubitable tumours, containing more or less imperfect but recognizable striated muscle elements, these, with the rare exceptions, are not in association with the ordinary muscles of the body, but are of the nature of mixed tumours, derived, it would seem, by displacement of cells capable of giving rise to striated muscle elements during the course of development. Most often in such tumours there is an admixture of cells of other orders, cells of a sarcomatous type, gland cells and, it may be, bone and cartilage and other tissue elements.

Another feature that we may lay down as characteristic of tumours in general is that the cells composing those tumours represent more or less faithfully some stage of development short of the perfect adult type. If we study the development of striated muscle, we find that there is a pre-existing stage in which the sarcoblasts, the embryonic cells giving rise to this particular tissue, become multinucleate, become, in short, giant cells. In fact, the adult muscle fibre is itself multinucleate. We should expect, therefore, were tumours derived from striated muscle at all common, to find giant-celled growths originating in association with the striated muscle in man. As a matter of fact, in the ordinary rhabdomyoma of man we encounter not infrequent multinucleate cells, but to my knowledge a tumour composed wholly of these,— what may

be termed a pure giant-celled rhabdomyosarcoma is unknown, or, at least, has so far failed to gain recognition as a separate entity. By great good fortune, I am indebted to my colleague, Dr. Hamilton White, for an exquisite example of this very condition in a trout caught by him in October. The fish is the "red trout," and was caught in Balsam Lake, Montfort district, in this province. Save for the tumour, it was a well grown individual, 14 inches long, and weighed about three-quarters of a pound. It will be seen that some 4 cm. behind the main dorsal fin and 1.5 cm. in front of the posterior dorsal fin, there is, on the left side, near the middle line, a very definite tumour. When brought to the laboratory, this was covered by a healthy unbroken skin, and projected some 1.5 cm. above the general surface.

On dissection, the tumour was found to be almost spherical in shape and 3 cm. in diameter lying to the left of the dorsal spines and not attached to these. A layer of muscle appeared to pass over it, and it had a semi-fluctuating feel. It was well defined, and was easily separated from the surrounding tissue.

On section, the tumour is found to be composed almost wholly of giant cells, varying, it is true, greatly in size and shape. The smallest cells may contain but two or three nuclei, the largest, without exaggeration, many hundred. There is no definite capsule, but at the periphery there is a zone exhibiting a moderate grade of small-celled infiltration, in which the tumour cells proper infiltrate between still recognizable striated muscle elements. This infiltration, it is noted, extends between the dorsal spines to the right side to a slight extent. These more normal muscle fibres are easily distinguishable; while shrunken, they exhibit regular striation and well marked longitudinal fibrillation. The interesting part is that in making a careful study of these remarkable giant cells certain of them are of very great length as compared with their breadth, and the nuclei are gathered more particularly at one pole. Such cells recall in a very striking manner the buds or processes projecting from the muscle fibre of a mammal in the process of regeneration after injury and in not a few of them the part of the cell furthest from the grouped nuclei shows well-marked longitudinal fibrillation, while here and there irregular but distinct transverse striation is to be made out. Studying the various transitional stages, there can be no doubt that here we are dealing with a rhabdomyosarcoma, and, as I have already indicated, we have encountered a new form of muscle tumour, but one, which from embryological considerations, is also to be termed "natural" and to be expected. We have found this in one of the lower animals, and it now remains to be seen

whether this form occurs also in man, and whether in man we have to add to the list of giant-celled tumours, a type gaining its origin from voluntary muscle.

Tumours in fish are not unknown. Some twelve years ago, I received from Dr. Deeks a relatively large myxofibroma, which he had removed post mortem from a cod, caught in the Gulf. If I mistake not, I brought the case before the Society. Recently, in connexion with the study of the distribution of malignant growths throughout the animal kingdom, there has been an increased interest in the subject, and several cases have been reported of tumours of different orders found in fi

The majority of these cases, curiously enough, are of adenomatous and even of definitely carcinomatous type (Scott, Gilruth, Plehn, Pick, and Bashford's first case). Judging from Dr. Marianne Plehn and Pick, and Poll's cases, their most common situation in the salmonid is below the lower jaw in the floor of the mouth. This position an their histological structure suggests strongly an origin from thyroid tissue. Bashford records a malignant adenoma of the peritoneal cavity of the Gurnard. The only sarcomatous tumour to which I have found reference is Bashford's second case, that of a spindle-celled sarcoma of the codfish, the figure given by him, with its loose arrangement of cells, shows some similarity to our own specimen of myxofibroma in the same fish. So far, I have been unable to come across the description of any case of a fish tumour at all resembling that here described.

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UNILATERAL CONGENITAL ABSENCE OF THE PAIRED GENITO-URINARY ORGANS.

BY

JOHN McCRAE, M.B., M.R.C.P.,

Lecturer in Pathology, McGill University, Montreal.

The specimen shows an absence of the genital and urinary structures of the left side, in so far as those structures which are bilateral are concerned. The woman was forty-nine years old, and had borne one child and had one miscarriage. There was no kidney, ureter, renal artery, ovary, Fallopian tube or broad ligament on the left side, and the uterus consisted really of but half a uterus—the right half; the organ was finger-shaped, and pointed to the right at about an angle of 30 degrees from the sagittal plane, which is the usual position and appearance of a uterus which has but one cornu. The vagina and urethra were apparently normal; the bladder was median and showed no sign of left ureteral opening. The right kidney was a little larger than usual and was in the normal position. The body of the uterus measured 6.5x2x2 cm. The right ovary was very small and wrinkled, but the right tube appeared normal.

The anomaly has arisen from the fact that on this side there has been no Wolffian body or duct formed, nor any Müllerian duct, and the entire series of structures which arise from them, kidney, ureter, ovary, parovarium, tube and uterine cornu, is missing.

I cannot find how uncommon this combination is; the absence of each one of the structures concerned is noted in nearly all works, and unilateral absence of the genital organs is commented upon; the only case at all parallel that I have found is in the Transactions of the Pathological Society, 1883-4, where Mr. Carrington recorded a case somewhat similar, in which the same abnormality of the genital organs was accompanied by a misplaced left kidney.

EXPERIMENTAL "WORK-ARTERIOSCLEROSIS."

BY

OSKAR KLOTZ, M.D.,

(From the Pathological Laboratory of the Royal Victoria Hospital.)

The experimental work in arteriosclerosis has, up to the present, been mainly of the nature of mechanically injuring the vessels, or else by introducing foreign toxic substances into the animal body. Of the latter type much has been written in the last four years, and it has been shown that substances like adrenalin chloride, barium chloride, digitalin and nicotine, all of which produce high pressure in the arterial system, are capable of bringing about definite arterial lesions. It has also been shown that certain bacterial toxins act on the vessel walls, either by producing degenerative changes, or else in stimulating the proliferation of certain cells.

There has been a considerable controversy as to the nature of these arterial lesions, whether they were the result of the toxic substances, acting directly upon the tissue cells, or whether their mechanical effect of increasing the blood pressure was capable of bringing about these changes.

Clinically, it has been noted that in the adult, the vessels of the more active organs show hypertrophy and sclerosis earlier than in the less active parts. In right-handed persons the radial arteries are considerably more sclerosed than those on the left side, and the reverse is true in left-handed people. Similarly, those whose occupation requires them to be constantly walking around and on their feet show the most advanced arterial changes in the vessels of the legs. These facts point to the prominent part that is played by work, in the production of arterioselerosis, but still the question arises whether in a healthy vessel increased work alone can bring about sclerotic changes, or whether it is necessary to couple the factor with the effect of toxic agents.

It was my endeavour to throw some light on this question by experimental means. I chose healthy, nine months old rabbits to carry on the experiments. The first animal was treated for one hundred and thirty days, by suspending him by the hind legs for three minutes each day. The endeavour was to increase the pressure and the mechanical stress in the arteries, without employing any drugs. By inverting the animal, the pressure in the thoracic aorta and in the arch is decidedly increased over that which normally exists in the animal. At the beginning, the animal did not seem to be worried by treatment, but later

on it showed signs of dyspnœa, and the heart beat was accelerated. Towards the end it was noted that the animal was much fatigued after each treatment.

At autopsy the following was noted:—There were no lesions in the vessels of the brain and no hæmorrhages had occurred in this organ. The carotid vessels had a remarkable appearance; the arteries were enlarged to about twice their size, and looked like sclerosed radials. There were distinct beadings on the vessels, which were most marked just above their origin from the aorta. These beadings were white in colour and encircled the vessels in transverse rings. Similar appearances were also present on the subclavian and brachial vessels. The beadings were distinctly palpable, while the vessels in general were firmer than normal. The amount of change in these arteries diminished after the bifurcation of the common carotids, though it was still apparent in some of the smaller branches.

Thorax and Abdomen.—The lungs were healthy and without change. The heart showed an enlargement of, at least, one and one-half times its normal size. The ascending aorta had its walls thickened, and was larger than normal. This increased size was apparent as far as the middle of the arch, or just beyond the opening of the left brachial. The wall felt firm and nodular, and did not collapse when its contents were removed. Opposite the 6th ri[illegible] the vessel again dilated to twice its size, forming a fusiform aneurysm as far as the diaphragm. This aneurysmal dilatation had firm and brittle walls, in which concentric rings could be distinguished passing about the vessel. Below the diaphragm the aorta again became smaller, but showed thickening of its coat, which was visible as far as the right renal artery. The beginning of the cœliac axis was also sclerosed, though no changes were noted in the branches of this vessel. The renal arteries were normal in appearance, and below them the aorta, too, was without change. There was no change to be noted in the iliac arteries, nor the vessels of the legs, nor did the viscera of the abdomen exhibit any microscopical lesions.

We have, therefore, produced macroscopical changes in the aorta and its branches above the renal vessels. In these changes the aorta is chiefly involved, while the carotids and the vessels of the neck are also sclerosed. Consequent upon the weakening of the aortic wall by sclerosis, a fusiform aneurysm developed in the thoracic aorta.

Microscopical.

Ascending Aorta.—The aortic wall was hypertrophied, the thickening occurring in the intima and possibly in the media. The media, where it was apparently thickened, was normal in structure and showed the

alternating layers of elastic fibres and muscle tissue. The intima, where thickened, showed the hypertrophy to be in the muscle elements (of the musculo-elastic layer). There was no connective tissue proliferation to be found. With the intimal thickening there was everywhere a process of degeneration accompanying it. This degeneration in the mildest form occurred close to the internal elastic lamina, and, in the more advanced types, extended closer to the endothelial surface. The muscle cells themselves were degenerating and disintegrating, leaving areas of non-cellular debris. These areas showed many spicules of crystals like those of calcium salts. Many of the cells were vacuolated as if containing drops of fat. In one area the media, too, showed degeneration where the muscle cells were entirely wanting, while the elastic bands were thrown into prominence by a darker blue staining, due to a calcification. Fractures were occasionally seen in the elastic laminæ. Remarkable cells were found between these calcified elastic bands. These cells were large with a spherical media and lay in a homogeneous looking matrix with vacuoles about them. One was reminded of the appearance of cartilage cells, though definite cartilage was not to be made out.

A study of these sections convinces one that the muscular changes are primary. The rupture and changes in the elastic fibres are secondary.

Descending aorta just above diaphragm.—The vessel wall was in its greatest extent narrowed. Only short stretches of normal looking aortic wall were seen. The rest of the wall showed a hypertrophied intima, in which the musculo-elastic layer was thickened, while the media was much narrowed. The middle zone of the media showed a band of calcification almost encircling the vessel. There was a narrow strip of media on both sides of the calcified band, which showed the muscle cells wanting to a great extent, while the elastic fibres lay more closely together. The adventitia nowhere showed change. In the calcified band of the media no cells were to be made out. This degenerative change in the descending aorta resembled that produced in the aorta by adrenalin chloride.

Carotids.—In the carotids the changes found were principally located in the intima. The media showed no changes in any part, save such as is produced by the compression of the thickened intima and slightly fatty degeneration along the border of the internal elastic lamina.

The intima was in parts normal, consisting of a single layer of endothelium lying upon the internal elastic lamina. In other parts there was a thickening of this membrane to that exceeding the thickness of the media. This thickened portion of the intima was made up of a

superficial and circularly disposed layer of connective tissue (possibly of endothelial origin), while beneath this was a thick layer of longitudinally disposed muscle fibres, with extensive fatty degeneration in them. In this deeper layer of the intima many of the muscle cells had entirely disappeared, leaving behind a granular debris mixed with minute fatty granules. In some places this thickened intima occupied one-half the circumference of the vessel.

We have, therefore, in this experiment been able to reproduce by physical means two kinds of changes in the arterial walls. The one is isolated in the media without intimal change and consists of a purely degenerative process, with death of the muscular elements and calcification of the involved areas, including the elastic fibres. The other change is isolated to the intima, and consists mainly of a proliferation of the tissue, while a secondary fatty degeneration has occurred in the newly formed tissue.

In the lesions of the first type involving the media there has also occurred the production of aneurysm. This, as we have previously pointed out, is the common result of severe degenerative changes in the media.

I believe, therefore, that we may conclude from these experiments that work plays a very important rôle in the production of arteriosclerosis of different characters, and that even in vessels of different histological structure sclerotic changes can be brought about by increasing the work of the artery. And further, as a consequence to certain changes, degenerative in character, taking place as the result of increased work in the media of the vessels, aneurysms may result.

We understand from Professor J. J. Mackenzie that Dr. Harvey of Toronto, working at Cambridge, has by different methods of increasing arterial pressure obtained marked changes in the arteries. As to the character of these changes, we have no information beyond that they are arteriosclerotic. We gather from Professor Mackenzie's letter that this paper has just been presented to the Royal Society of London.

TRYPANOSOMES IN MONTREAL RATS.

BY

OSKAR, KLOTZ, M.D.

(From the Pathological Laboratory of the Royal Victoria Hospital.)

After Dr. Todd's extensive report before this Society on the Trypanosomes of Central Africa Sleeping Sickness, we are apt to associate this organism with diseases in far off lands. There are, however, many varieties of trypanosomes, and it would appear that some of the animal parasites not yet fully worked out will yet be classified amongst the trypanosomes. During the winter of 1906-07, Dr. Ballah and I examined for trypanosomes some forty rats, all obtained in the same locality in Montreal. These examinations all proved negative. Recently I examined two rats from a down-town grocery, and found both of them to harbour trypanosomes in their blood in large numbers. Subsequent to this, Dr. Rankin found trypanosomes in a rat obtained at the Royal Victoria Hospital. No doubt, if an extensive search were made, these trypanosomes would be found in a large percentage of the rats of this city.

This organism, the T. Lewisi, is a very common parasite in the ordinary house rat, some even claim that it is as widely distributed as the rat itself. Novy, of Ann Arbor, was the first to recognize the T. Lewisi in America. Since then, I learn that it has been found in Detroit, San Francisco, Philadelphia, New York, Lincoln, Seattle, Chicago and Ottawa.

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