IV. *The Excitatory Process in the Dog's Heart.** Part II.—*The Ventricles.*

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[Plates 8–12.]

Historical.

In describing the results of experiments of a physiological nature it is the natural custom to discuss the observations of previous workers in the same field.

Our present subject is the excitatory process in the mammalian ventricle, and the past publications to which we shall at present refer are those of Waller and Reid†, Bayliss and Starling,‡ and lastly Gotch.§

Believing, as we do, that the spread of the excitatory process in the mammalian ventricle is far more complex than any of these writers had reason to suspect, and of so special a kind as to render the methods which they adopted almost abortive, we do not feel justified in discussing these papers in any detail. With a single exception the experiments named date back many years, and antedate recent and remarkably progressive developments of cardiac physiology.

It is necessary to emphasise the fact that recent discoveries have increased our knowledge to such extent and so altered our conception of processes in the ventricle, as to place the whole question upon a different footing and to make it abundantly clear that the older methods of investigation were incapable of analysing the progress of the natural excitation wave. In this connection they become almost purely historical.

The observations of Waller and Reid, and the later observations of Waller,‖ were undertaken upon the dying heart, either excised or beating *in situ* after decapitation of the animal. Such records as were obtained were from the heart muscle deprived of its circulation. Upon this score alone we should be compelled to rule out the conclusions of these experiments while discussing the cause of the natural excitation wave.

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† 'Phil. Trans.,' vol. 178, p. 215 (1887).

‡ 'Monthly Internat. Jour. of Anat. and Physiol.,' vol. 9, p. 256 (1892).

§ 'Heart,' vol. 1, p. 235 (1910).

‖ 'Phil. Trans.,' vol. 180, p. 169 (1889).

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Such records as have been taken in the past have been obtained with the capillary electrometer; the curves in no case having been analysed to eliminate instrumental defects. The recent introduction of the string galvanometer has very greatly simplified our procedure, but has shown the current changes of direct leads to be far more complex than was formerly believed.

The chief question which has engaged the attention of past workers has been as to whether the base or apex of the ventricle passes first into the excitatory state. Different observers have reported different findings; WALLER concluding that the apex usually precedes the base; BAYLISS and STARLING believing "that the ventricular contraction is a single wave, starting from the base, at the auriculo-ventricular groove and spreading thence to the apex"; GOTCH believing his results to show that the contraction begins at the base, travels to the apex, and returns to the arterial base. The relative unimportance of this question, hitherto considered fundamental, will become clearer as our own results are described. The assumption, usually tacit, in all these papers is that the excitation wave travels as a simple wave propagated from muscle element to muscle element. It is this assumption which has led to a comparison of base and apex of the heart, regions not too easily defined.*

The essential question of propagation in the ventricle is, as we purpose to show, not a question of the order of base and apex, and the assumption which brings this question into prominence is, in all probability, erroneous; for the wave of contraction is not a simple and uninterrupted spread from a single region. The possible significance of the Purkinje system has been unrecognised by past workers.

Lastly, the workers in question have failed to appreciate the essential distinction between currents emanating from two sources, *intrinsic* and *extrinsic*, as we term them, according as the origin is (*a*) in excited muscle beneath the contacts, or (*b*) in excited muscle at a distance from the contacts.

As we have shown in the case of the auricle, the initial appearance of relative negativity at a given contact, which is one of a pair upon the heart, does not necessarily indicate that the muscle beneath this contact first passes into the excitatory condition; such conclusions as have been based upon this assumption are open to very serious objections. In estimating the order in which the excitation wave appears at a series of given points, it is evidently necessary that we should be able positively to identify those deflections of our curves which correspond to the arrival of the excitation beneath the individual contacts; this is not so simple a matter as might at first appear. In looking back at the records of our predecessors in this field,

* To attempt to bring our own results into complete harmony with those of previous workers would require extensive comparisons of electrometric and galvanometric records; a laborious task which we deem unprofitable, especially as we are unaware of the exact sites of contact used in their experiments, a very essential matter. After full consideration we are of opinion that such discordances as exist are to be explained by the different plans upon which the investigations have been carried out, and especially to fallacious assumptions by former workers, to which we shall refer in more detail.

we are confident that extrinsic effects have been read repeatedly as intrinsic effects; in other instances, it is impossible for us to recognise the all important intrinsic effects with any degree of certainty in their curves.

Since our experiments have been completed our attention has been called to the recent publication of ERFMANN.* This worker has used methods not dissimilar to our own; he employed two recorders and utilised one as a standard for measurement;† his observations are much less extensive than our own, and are often open to the objection that extrinsic and intrinsic effects are not differentiated. ERFMANN'S work will be referred to from time to time in our report.

Anatomy.

As an introduction to our paper it will be convenient briefly to describe certain anatomical observations.

The Front of the Dog's Heart.—When the dog's heart is laid bare by splitting the sternum, a large area of the right ventricle and a smaller area of the left ventricle is exposed. Of the right ventricle we see the whole conus, the base of the ventricle where it merges with the fat of the auriculo-ventricular groove, a portion of the blunt apex of this chamber, and almost the whole extent of the anterior interventricular furrow (fig. 1). The latter is marked with accuracy throughout the greater part of its course by the descending branch of the left coronary artery and its accompanying veins, and forms almost a straight line. An area of heart wall is exposed to the right (in the animal) of this line in its lower reaches, which is of considerable importance for purposes of orientation. It is bounded on the left by the descending branch of the left coronary vessel and on the right by a line convex to the right. At first this curved line may be mistaken for the interventricular junction; it is not properly so, but forms the outermost attachment of fibres of the right ventricle to the septum; it is up to this line, and not beyond it, that the right ventricle swells when distended. The enclosed area now described is relatively flat, though its surface is dimpled, for a reason which will appear subsequently. Beneath it is a sponge-like tissue, the pores of which communicate with the cavity of the right ventricle. This area is in reality a continuation of the wall of the right ventricle, but closely bound to the septum by numerous and strong trabeculæ which ascend to the septum and anterior papillary muscle of the right side; we term it the *trabeculated region* for descriptive purposes (fig. 1).

We have made numerous dissections of the whole musculature in the dog's heart and find it to be arranged on the same general plan as described for the pig and

* 'Zeitschr. f. Biol.,' vol. 61, p. 155 (1913).

† ERFMANN employed two galvanometers side by side. His method of measurement is not stated, and errors of measurement are not estimated, but we judge it probable, both from the small amplitude of his curves and from the photographic method employed, that they were considerably greater than seems to us expedient in the estimation of small time differences.

human heart by MacCallum and Mall.* We shall refer only to those features of the architecture which are essential to our thesis.

The superficial fibres over the ventral surface of the ventricle sweep from right to left. Starting in the conus tendon and at the auriculo-ventricular ring a broad and thin sheet of fibres passes obliquely over the upper conus and crossing the inter-ventricular groove (Plate 8, figs. 20 and 21), sweeps around the left heart margin and eventually terminates in the vortex of the left ventricle. Further to the right, fibres belonging to the same system pass to the trabeculated region, but these do not cross; they interlace in an intricate manner with fibres ascending from the left apex. The general distribution of these superficial fibres may be seen in Plate 8, figs. 20 and 21. Thus, of the superficial fibres which start at the base of the right ventricle and appear over the front, the upper ones pass to the left ventricle and sweep around it in a continuous sheet, while the lower ones are lost

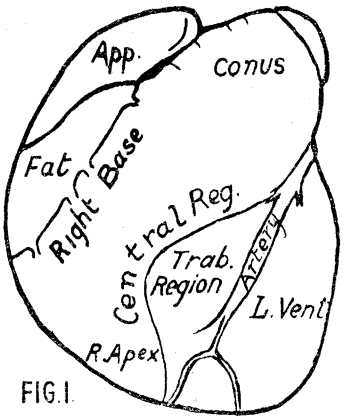


FIG. 1.

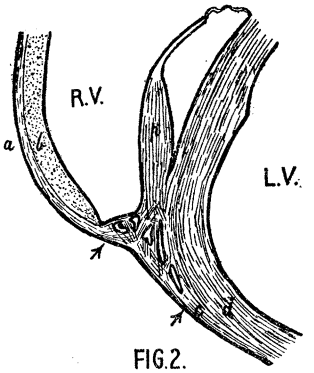


FIG. 2.

Fig. 1.—A diagram of the ventral surface of the heart of a dog.

Fig. 2.—A diagram (natural size.) illustrating the architecture of the trabeculated region, as seen in section. p = papillary muscle.

in the interlacement of the trabeculated region; on the surface, the change in distribution defines the upper limit of the trabeculated area. The deep distribution of the fibres of the trabeculated region may be most easily understood with the aid of a diagram (fig. 2). This diagram represents an oblique coronal section taken through this region of the heart, so arranged as to cut the large anterior papillary muscle in its length. The superficial fibres of the right ventricle are cut longitudinally and are seen at (a); they sweep into the trabeculated region, the limits of which are indicated by arrows, and, decussating, pass into the papillary muscle and septum through strong trabeculæ. Beneath them lies a second and thicker layer of fibres (b), which takes a more transverse course over the front of the heart and sweeps forward to form the main thickness of the conus proper; it enters the septum at a much higher level and takes little or no part in the formation of the trabeculæ. The fibres a decussate with fibres c which sweep over the

* 'Amer. Journ. Anat.,' vol. 11, p. 211 (1910–11).

left apex in one direction and form the right wall of the septum in the other. A complete and clean separation may be easily effected between the layers *c* and *d*, the latter comprising the chief fibres of the left ventricular wall. That portion of the trabeculated region which lies opposite the anterior papillary muscle may be regarded as the root of this muscle, and the dimpled surface of the heart in this region, especially prominent in the active organ, is due to this attachment of the papillary muscle. It is used as the chief landmark in estimating the position of the papillary muscle and right branch of the $A-V$ bundle in the intact organ when observations upon these structures are desired.

Distribution of the Purkinje System of Fibres.—A complete and accurate account of the course of the auriculo-ventricular bundle and its two chief branches to right and left ventricles in the dog is to be found in TAWARA's important monograph.* Some idea of the distribution of the arborisation and subendocardial network of the junctional system, of which this bundle is the beginning, is to be gathered from the same publication. TAWARA's description is based upon reconstruction in serial sections, and this writer refers to the difficulty of identifying the branches of the network in his sections, on account of their close resemblance to ordinary ventricular muscle fibres. His description of the network is therefore imperfect.

The extent of the Purkinje system and the general plan of its ramifications has recently been studied in the ungulate heart by the injection of the sheaths which everywhere enclose the main strands, a method introduced by LHAMON† and since employed by a number of workers. We have also used this method extensively in the ungulate heart, for, by its means, we are able to form a very exact idea not only of the form and distribution of the chief branches, but also of the extent of the network. To certain of these observations we shall allude. In the ox heart, as has been demonstrated by TAWARA, and by LHAMON, COHN,‡ and others using the method of injection, the right division of the main bundle stem courses from the membranaceous septum, along the right side of the muscular septum to the base of the moderator band. At this point, according to COHN, branches flow to an extensive network which covers the dorsal part of the septum. The arrangement of the network is well displayed in Plate 11, fig. 24, a photograph of the opened right ventricle of an ox. The sheaths have been injected. The course of the right division of the bundle is seen as a white streak over the moderator band (*c*–*b*). This division subdivides at the base of the chief papillary muscle (*b*), but early outgoing branches are visible at *c*. That there is a definite connection in this region with the network covering the septum of the ox, we can show clearly. In an occasional ox heart the muscular moderator band does not include the right bundle division, but the latter traverses the chamber on the conus side of it, and has a separate sheath of endocardium.

* 'Das Reizleitungssystem des Säugetierherzens,' Jena (1906).

† 'Amer. Journ. of Anat.,' vol. 13, p. 55 (1912).

‡ 'Heart,' vol. 4, p. 225 (1912–13).

An example is shown in outline in fig. 3 (*m.b.* = moderator band ; *c–b*, the separate sheathed branch of the bundle). The injection fluid was allowed to flow up the sheath of this bundle (from the point marked *inj*) ; it quickly spread along the whole length of the right division (*d*) and, flowing out through the free branches (*a*, *a*), filled a network whose extent is indicated by the arrows on the chart. We emphasise these early outgoing branches in the ox heart because we have failed to find them in the dog. The actual network of the right ventricle in the ox is found throughout almost the whole extent of the subendocardial space (see Plate 11, fig. 24). The apical portions are everywhere lined with it, and it extends throughout the whole conus to within a

FIG.3.

FIG. 3.—An outline drawing ($\frac{2}{3}$ natural size) of the opened right ventricle of an ox heart. *p.v.* = pulmonary valves ; *t.v.* = septal segment of tricuspid valve ; *p.m.* = papillary muscle ; *m.b.* = moderator band ; *c–b* = right division of bundle specially ensheathed with endocardium. Colouring matter was injected upwards at the point marked *inj.* ; it flowed in the sheaths of the Purkinje tissue over the areas indicated by arrows.

few millimetres of the bases of the pulmonary valves. It extends towards the tricuspid valves on the free and septal wall of the ventricle, but on the latter not more than a few slender offshoots extend so far as to be concealed by the septal segment of the valve. The subendocardial space deep to this valve segment is poorly supplied.

In the dog's heart the injection method has been found to be impracticable, though it has been attempted frequently. We have used another method of displaying the

system. We have taken advantage of BEST's* method of glycogen staining, knowing from NAGAYO's studies† that the glycogen content of the special tissues is high. At the end of an experiment the animal is kept alive, and 150–300 c.c. of a saturated solution of dextrose is slowly run into the bloodstream. After one or two hours the dog is killed, and the heart quickly removed and washed in a saturated dextrose solution. It is distended with 35 per cent. formalin solution also saturated with sugar and left until hardened. After being again washed in saturated dextrose solution it is stained in bulk with Best's alkaline carmine for from 12 hours to a week, and differentiated in his alcoholic fluid. After 12 or more hours the whole Purkinje network and the chief branches of the bundle are well displayed *in situ* as dark red lines upon a pink background.

In the dog, the right bundle division passes deep to the conus edge of the septal segment of the tricuspid valve (Plate 8, fig. 19), and is almost always superficial in its course from this point onwards; it follows a curved course over the septum, reaching and embracing the base of the chief papillary muscle. Arriving at the right and dorsal border of this muscle it breaks up into a number of branches, and many of these then proceed across the cavity of the ventricle to the free wall, each isolated and sheathed by endocardium (Plates 8 and 10, figs. 20, 21, 23). Frequently a complex interlacement extends dorsally from these free branches, and twigs are sent to the apical portions of the endocardium in many directions. Reaching the wall of the ventricle, the branches and twigs break up and are lost in the network proper. In our experience every free strand of considerable size in this region is a carrier of Purkinje substance. But these are not the first branches of the right division. The earliest outgoing fibres which we can discover come from the convexity of the right division, as it sweeps around the papillary muscle (see diagrams below, figs. 12 and 13, and Plate 8, fig. 19). These course along the trabeculæ from septum to free wall. We are unable to find early outgoing fibres to the septum towards the base of the heart, either in studying macroscopic specimens, or in the histological preparations similarly stained.‡ In a solitary heart a single branch of early origin was discovered, but this ran, seemingly, as an unbranching strand, almost to the apex of the ventricle along the septum dorsally to the chief papillary muscle. The network has a very similar distribution to that found in the ox, though our method displays it more fully than it can be seen in the last named animal. It may be said to line the subendocardial space universally with the exception of areas to be mentioned presently. Over the free wall of the right ventricle, including the conus, it forms an almost continuous sheet; like dense lace it forms an almost imperforate sheet, the denser parts of which

* BEST, 'Zeitschr. f. wissensch. Mikroskopie,' vol. 23, p. 319 (1906); see also SCHMORL, 'Untersuchungen Methoden,' 7th Ed., p. 183.

† 'Verhandl. d. deutsch. pathol. Gesell.,' vol. 12, p. 150 (1908).

‡ Experimental evidence has since been obtained, however, that earlier outgoing fibres exist; the right division of the bundle does not pass entire and unbranched to the region of the papillary muscle.

cover over the raised trabeculæ, the fibres in the sheet being arranged side by side and following faithfully the directions of the smallest trabeculæ Thus every depression is encircled by a broad band of parallel fibres. The depressions themselves are lined more sparsely, but the whole subendocardial space is supplied. If strips are torn from the endocardium and examined under a microscope, a finer network is seen filling the interstices in the coarser network. Where, as in the upper reaches of the conus, depressions are less defined or absent, the same general arrangement holds good ; broad bands of the tissue are looped together and encircle spaces in which a flimsier network is discovered. It seems as though this region were also once trabeculated, but that with the expansion of the conus the irregularities of the surface have been lost. The special tissue runs into the deep sulci of the freely trabeculated space along the ventral attachment of the right ventricular wall (the trabecular region already described). The septal subendocardial region is less richly supplied ; the network does not appear to be well developed towards the tricuspid valve, once the papillary muscle is passed. The base of this muscle is richly supplied, but above it, strands visible to the naked eye are rarely seen, and under the microscope only occasional traces of a flimsy network are discovered. No fibres have been seen deep to the septal valve segment itself. There appears, therefore, to be an area of subendocardial tissue in the region of this valve segment, poorly supplied by the network or actually devoid of it. On the free wall and towards the tricuspid valve the extension is higher, though it falls short of the auriculo-ventricular ring itself.

The general arrangement in the left heart is similar to the recognised distribution in ungulate hearts. The left division breaks into two chief strands, and these proceed, completely enwrapped by endocardium, to basketworks around the anterior and posterior muscles (Plate 9, fig. 22). Other and smaller branches run down the septum branching and anastomosing freely. The network over the whole apical half of the ventricle is extremely dense and similar in its general constitution to that found in the other ventricle. A sheet of interlacing strands passes deep to the unattached portion of each papillary muscle ; the whole subendocardial space is richly lined except for a small area immediately beneath the aortic valves on each side of the main left division.

It is most important for our purposes that the general and wide distribution of the Purkinje system should be grasped, and that certain special features should be remembered, for these features serve to explain some of the experimental facts presently to be detailed. In particular we would emphasise the course and relation of the right division to the chief papillary muscle of the right ventricle (see Plates 8–10, figs. 21, 22, and 23); the presence of a complex system of large free or bridging strands in the right chamber ; the fan-shaped distribution in the left chamber ; and lastly, the presence of two areas which are comparatively free from fibres, the first and largest being on the right side of the septum in the region of the septal segment of the tricuspid valve, the second immediately beneath the aortic valves. It is also of importance to

point out that in the dog, as in the ox, the distribution is not confined to the sub-endocardial space; for sections clearly demonstrate that here and there offshoots pass into the muscle substance, following the course of vessels or connective tissue strands. Such penetration is not deep so far as we can ascertain; it is of one or more millimetres extent; neither is it frequent; but where the wall of the ventricle is thin, it may bring the special tissue very near to the pericardial surface.

Experimental Method.

Our methods are described in detail in the section of our reports which deals with the auricle, consequently it is necessary to record only those variations of technique which have been necessitated by the transference of observation from auricle to ventricle. In all experiments the vagi have been divided. We expose the heart in one of three ways: by splitting the sternum, the animal lying on its back; by removing a large area of the right chest wall, the animal lying on its left side; or, finally, by removing a large area of the left chest wall, the animal lying on its right side. We adopt one or other procedure in a given experiment, maintaining the attitude throughout that experiment. The several exposures permit us to examine the whole superficies of the ventricle.

The electrodes for direct leads which we have been employing have been of three classes:—

1. Paired contacts such as were used for the auricle.

2. Solitary contacts of the same pattern, though smaller in bore (*i.e.* 1½ mm.), and used in conjunction with non-polarisable contacts stitched permanently to right or left chest wall.

3. All-glass electrodes, with suitable curvatures for observation inside the cavities of the ventricles. At first these electrodes were double; eventually, and for the majority of the observations, we have employed single contacts, and have led from these to a contact on the chest wall.

Our standard signal has been Lead *II*, from right shoulder to left groin, and the sensitivity of the recorder for this lead has been such that 3 millivolts introduced give an excursion of 3 cm. For direct leads the sensitivity has varied according to circumstances; but as a general rule it has been such that 6 millivolts give 3 mm. (scale divisions in our figures) excursion.

The experiments have been so arranged that all the observations upon a given animal have been made with as little displacement of the heart as possible. For external leads there is no displacement; for internal leads there is little; the latter have been undertaken for the most part by introducing contacts through one or other auricular appendix and the corresponding auriculo-ventricular orifice. Internal leads have always been the last in an experiment.

At the time of the experiment careful drawings, so far as possible to scale, have been made of the exposed heart, a number of the vessels being charted to aid the

subsequent identification of contact points. In some instances it has been necessary to mark the epicardium at the time by staining it with a small spot of carmine. The heart has been excised at the end of the experiment, distended, and fixed. Subsequently the contact points are identified and an accurate drawing to scale has been made either by measurement or by optical projection. Certain of the hearts were prepared in the special manner already described to display the Purkinje network.

Our standard, in estimating with the comparator the time at which the excitation wave appears at a given point, is *R* in Lead *II*; this deflection starts sharply, and as a rule requires no check. Its beginning is subject to far less error in estimation than is *P*, the auricular summit, adopted in our previous observations.

Recognition of Intrinsic Deflections.

It will be remembered that our observations upon the auricle depended upon our ability to isolate and recognise that deflection of our galvanometric fibre which, in records from direct leads, corresponds to the arrival of the excitation process beneath one of the contacts placed upon the muscle. In a direct lead the first deflection of the string, as we were able to show, does not usually correspond to the passage of the muscle beneath the contacts into the excitatory state. We divided the deflections obtained in direct leads into two classes for purposes of description, terming them intrinsic and extrinsic respectively. In the case of the auricle, to recognise the intrinsic deflection, or that which signals the arrival of the excitation at the contact area, is not difficult; for the spread of the excitation wave takes place in a radial fashion from the sino-auricular node, and so if the paired contacts are arranged radially to this node, and the same contact is maintained as the proximal contact throughout, the intrinsic deflections are all in the same direction upon our plates. Further, the time of a given intrinsic deflection may be taken as the time at which the excitation process arrives at the corresponding proximal contact. When we came to study the ventricle we were met by an initial difficulty. We had no preliminary guide to the direction taken by the excitation wave; so, when we placed a pair of contacts upon the ventricle and obtained a complex curve consisting of a number of upward and downward deflections, we might be unable to state from its appearance which contact had first received the excitation wave; it not infrequently happened that we were unable to identify our intrinsic deflection with any degree of certainty. We performed a large number of experiments in which various areas on the surface of the ventricle were examined by means of paired contacts and, although these experiments gave us a general idea of the events, the method did not provide us with data of sufficient accuracy; and, ultimately, we were obliged to discard it. If closely paired contacts are placed upon the surface of the ventricle and connected to the galvanometer, the direction of the intrinsic deflection is usually obvious, for this deflection is the only one which is of considerable amplitude as a general rule. In our first studies we were able to map out the general order of the muscular activity

by watching the moving fibre, and by using the direction of the chief deflection as a guide to the initial negativity of one contact as compared to the other. If this method is used for the front of the heart it is found that the earliest region to show the excitatory state is a point or band on the ventral surface of the right ventricle where its wall becomes attached to the septum and near the middle of this line of junction; that is to say, where the free wall of the ventricle borders the middle of what we term the trabeculated region. For descriptive purposes we name this region, which shows the first sign of passing into the excitatory state, the *central region* (fig. 1). If the plane of the contacts is maintained across the heart, and the contacts are moved over the ventral surface of the right ventricle (speaking of the free wall), a contact on the left border of this ventricle will always show relative negativity to a contact placed nearer its right margin, indicating that the general course of the excitation wave is from left to right, so far as this ventricle is concerned. If we examine the same ventricle with the contacts on lines following the long axis of the heart, then the general course of the excitation wave is towards the base over the basal two-thirds* and towards the apex over the apical third of the right ventricle. Measurement of the times at which the intrinsic deflections appear at the different contact points relative to R in our standard lead gave us a general and clear confirmation of these results, and we were satisfied that the earliest region to become excited is at the left border of the right ventricle. But when we attempted to map out the heart in detail by this method of paired contacts, and especially when we came to examine the left ventricle, we were unable to proceed with the certainty which we desired. For it often happened that the deflections from a pair of contacts were extremely numerous, and two or more might have similar amplitudes; consequently, we could not finally decide upon a given deflection as the intrinsic deflection. Curves of this kind, as we have been able to show subsequently, are due to the two contacts resting on points which enter the excitatory state almost simultaneously. This condition is especially frequent over the left ventricle.

For this reason we adopted another method of leading off, using a single contact upon the muscle and maintaining a second contact upon some fixed point on the chest wall. This method has the advantage that all intrinsic deflections have a constant direction (upwards in our curves); as a consequence they can be recognised with greater facility and certainty. Where, as in Plate 11, fig. 26 B (upper curve), the curve obtained has consisted of a minute or small downward deflection followed by a prominent upward spike, and where this spike is the most abrupt (as judged by the thinness of the written line) and has by far the greatest amplitude of any deflection in the curve, we are confident it represents the intrinsic deflection and corresponds to the arrival of the excitation process beneath our solitary muscle contact. Where the curve is of less distinctive form, its form may be altered oftentimes by moving the distal

* ERFMANN, using a different method of leading, comes to a precisely opposite conclusion.

(or chest wall) contact either to another point on the chest wall or on to the heart itself. Moving the distal contact has been employed to confirm our interpretation of the events represented in a curve, where such confirmation has seemed desirable. We find in the ventricle precisely what we found in the auricle, namely, that a change in the position of the distal contact* does not materially influence our measurements; yet such change may modify the shape of the curve profoundly. A number of measurements which bear out this statement are given in Table I. The last observation in this Table is illustrated by Plate 11, fig. 25 A and B. The curve obtained from contacts *a–b* (fig. 4) is shown in fig. 25 A. It consists of a number of small deflections, succeeded by a prominent summit, which has its onset 0·0188 second after *R*. The curve from contacts *a–c* (fig. 4), *c* being upon the left chest wall, is of an entirely different type (fig. 25 B). In this figure the prominent and steep upward movement represents the intrinsic deflection, though it starts well below the base line; it comes at almost exactly the same time interval, namely, 0·0190 second after *R*. Each curve gives a sufficiently accurate reading, but the time at which the excitation wave arrives at *a* is known with greater certainty from the second curve (fig. 25 B), for in the case of this curve

TABLE I.—Changing Distal Contact.
(Extrinsic and Intrinsic Deflections related to *R* in Lead *II*.)

Dog.	Proximal contact.	Distal contact.	Extrinsic.	Intrinsic.
G.F.	1. Central region . .	1 cm. above proximal . . .	−0·0078	0·0055
	2. Unchanged . . .	Right chest wall	−0·0099	0·0037
G.H.	1. Central region . .	1 cm. away from proximal .	−0·0070	0·0042
	2. Unchanged . . .	Right chest wall	−0·0081	0·0035
G.H.	1. On front of L.V. .	Right chest wall	−0·0071	0·0130
	2. Unchanged . . .	Left chest wall	−0·0079	0·0132
	3. Unchanged . . .	Mid abdominal wall . . .	−0·0073	0·0124
G.H.	1. Mid R.V. . . .	1 cm. above proximal . . .	−0·0031	0·0027
	2. Unchanged . . .	Conus	−0·0056	0·0036
G.I.	1. Apex of R.V. . .	Base of R.V.	−0·0089	0·0096
	2. Unchanged . . .	Chest wall	−0·0094	0·0096
G.J.	1. Base of R.V. . .	8 mm. above proximal . .	−0·0112	0·0014
	2. Unchanged . . .	Right chest wall	−0·0121	0·0009
G.J.	1. Mid front L.V. .	Left chest wall	−0·0100	0·0163
	2. Unchanged . . .	Right chest wall	−0·0097	0·0164
G.J.	1. Mid front L.V. .	8 mm. above proximal . .	−0·0066	0·0188
	2. Unchanged . . .	Left chest wall	−0·0100	0·0190

NOTE.—All readings in our Tables are averages for three heart cycles.

* Meaning at the present time by distal contact, that contact which is the last to receive the excitation wave, as opposed to the proximal contact which receives it first.

we know from the arrangement of the contacts that an upward movement signals relative negativity of the single contact upon the heart.

It must be evident that it is a matter of first concern to us, to ensure a correct interpretation of our curves. The procedure just described, namely, movement of the distal contact, is our first method of control. A second and equally satisfactory control is also similar to a method adopted for the auricle. We found in studying this chamber of the heart, that the intrinsic and extrinsic deflections could be separated and identified by taking a series of curves from serially arranged contacts over the edge of the auricular muscle on inferior and superior cava. A similar procedure is possible in the case of the pulmonary artery. A series of readings is taken from contacts lying on the conus and pulmonary artery; an example of such readings is given in fig. 5.

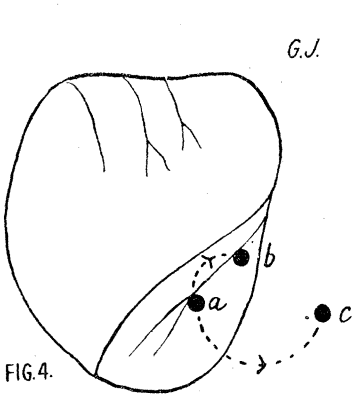


FIG.4.

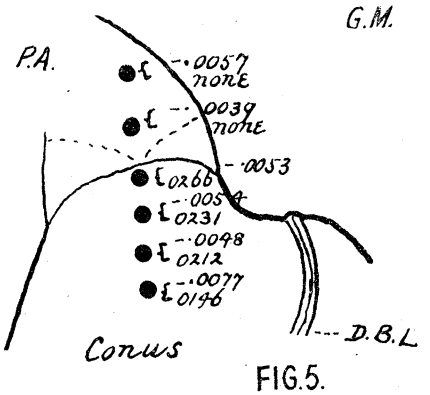


FIG.5.

FIG. 4.—A diagram to illustrate a comparison between two methods of leading from the heart. 1. Two contacts, *a* and *b*, are placed upon the muscle, and the lead is from *a*–*b*. 2. A contact, *c*, is placed on the chest wall, and the lead is from *a*–*c*. The contact from which an arrow points is that which when relatively negative yields an upright spike in the curve. The corresponding curves are shown in Plate 11, fig. 25.

FIG. 5.—An outline (natural size) of the conus and pulmonary artery (P.A.), showing readings of extrinsic deflections (above) and intrinsic inflections (below) from six contacts in a line over conus and artery. D.B.L. = descending branch of left coronary artery.

Opposite each contact site are two figures; the upper one is the time, relative to *R*, at which the first extrinsic deflection appears; the lower one is the time of the corresponding intrinsic deflection. The extrinsic deflections of this series appear at times which are within a few thousandths of a second of each other, and in no definite order. The times of the intrinsic deflections are arranged in an orderly fashion, becoming later as we proceed up the conus; but as soon as the muscle edge is passed, the intrinsic deflection vanishes, showing that, when present, it is dependent upon the activity of muscle immediately beneath the corresponding contact. A similar series of conus leads is shown in fig. 11.

The Form and Amplitudes of Ventricular Curves.

The form of curves taken directly from the ventricle by a single contact is variable. The majority of such curves show an initial dip of variable extent (2–35 millivolts).

This dip is relatively slow and usually unbroken, but it may be notched; it represents the first or extrinsic deflection. There may be a return to the base line before the intrinsic deflection is recorded, but more commonly, as in Plate 11, fig. 25 B, the onset of the intrinsic deflection terminates the dip. The upstroke is always very abrupt, and the recorded image of the fibre is consequently faint. Its amplitude, measured from its point of origin, is almost always considerable (10–80 millivolts). Usually, therefore, the intrinsic deflection is recognised with facility. In exceptional curves and where it is preceded by deep extrinsic effects of long duration, it may not be so easy to identify; in some such curves it consists of a short spike interrupting the downstroke. In other instances, its onset may be confused by the presence of several preliminary extrinsic deflections; if immediately preceded, as it may be, by an upward movement of small amplitude, this upstroke and the intrinsic deflection itself may fuse and an inaccuracy is introduced into the readings. But readings from such curves can almost always be avoided, and the onset of the deflection which represents the curves of the excitation wave can be identified positively in the great majority of the curves.

Distribution of the Excitation Wave over the Surface.

Ventral Surface.

We have studied the distribution of the excitatory process on the surface of the ventricles in more than 30 animals, and, using the methods which have been described, have arrived at results of considerable constancy. It is not possible satisfactorily to examine the whole surface in a single animal, and we first studied the ventral surface, as it is exposed by splitting the sternum. Our observations consisted in ascertaining the times at which the excitation wave appeared, relative to *R* in Lead *II*, at some 20 or more points, freely utilising the surface vessels and other landmarks for accurate charting. In Plates 8 and 10, figs. 20, 21, and 23, three projected outline drawings are published which sufficiently illustrate our results. Speaking of this, the ventral surface of the heart, the first appearance of the excitation wave is almost simultaneous with *R* in Lead *II*. Five points in fig. 20 show readings (0·0037, 0·0051, 0·0055, 0·0067, and 0·0073 second) varying no more than 0·004 second from each other. These points lie in our central region and are surrounded by points showing readings varying from 0·0108 to 0·0131 or 0·0145. All points over the right ventricle are later than those of the central region, and they are later according as they are further removed from it. Thus the excitation wave appears at the base of the right ventricle at times such as 0·0267 and 0·0221 second. If we examine the similar diagrams of figs. 21 and 23, we see a similarity of distribution. The central region (right ventricle) comes first in order, but the earliest region is as a rule not confined but diffuse. The limits and extent of this central region vary a good deal from animal to animal, but the present examples sufficiently illustrate them. In practically all our animals the base of the right ventricle, where it runs into the fat of the groove, comes between 0·0150 and 0·0270 second (the last value is exceptionally high). The conus is later, and near its

exit nearly always surpasses 0·02 second and approaches 0·03 second. The trabeculated region, included between the bulge of the right ventricle and the descending coronary artery, is early (Plate 8, fig. 21, 0·0049, 0·0082, and 0·0100 second) but is later than the central region. Once the descending coronary is crossed, higher values are found; immediately to the left of it they are in the neighbourhood of 0·02 second, further to the left and along the actual left border of the heart they fall again.*

The three diagrams of the ventral surface of the ventricle are accompanied by accurate projections of the underlying structures; the relation of the surface points examined to the superficial muscle bands, the cavities, the papillary muscle, and the free branches of the conducting system are shown.

If those contacts are studied which lie over the long band of muscle fibres, springing from the base of the right ventricle and sweeping to the left ventricle across the interventricular furrow (left marginal figure of fig. 21), such contacts show almost simultaneous reception of the excitation wave. A superficial examination of figs. 20 and 21 immediately shows numerous discrepancies between the sequence of contact points and the line of the fibre; the distribution of the excitation wave is not related to the sweep of the muscle bands.

The same figures, however, give us a distinct hint as to the manner in which the wave is distributed, for there is an unmistakable relation of the earliest points to the spread of the chief branches of the right division of the bundle. The central region is the region of the free wall which is supplied by the earliest outgoing branches, namely, those which spring from the convexity of the right division as it embraces the papillary muscle, and by the free branches which bridge the cavity; the main division and these free branches are clearly depicted in our diagrams. Before we consider this question of the course of the wave more fully, we may complete our account of the surface distribution.

Right Surface.

Having obtained a complete picture of the ventral surface, and observing that the figures for any given region are relatively constant from animal to animal, we may build up the picture and include other heart surfaces by overlapping the fields of observation. This method avoids displacement of the heart in any given animal during the course of an experiment, for only a single surface is examined in a given heart. Three figures (figs. 6, 7, and 8) of the right surface are given as illustrations. The fields of observation include the whole of the right and dorsal portions of right ventricle, and a complete base-apex strip of left ventricle. The whole or part of the central region is also included. In each diagram the central region is the earliest part of the right ventricle to be excited. It is also to be noticed that the apex of the

* According to ERFMANN all points of the superficies are activated simultaneously, a few thousandths of a second only being found between them. We think this conclusion is due to the fact that his observations were almost confined to the base and left apex of the ventral surface.

right ventricle may be included amongst the earlier regions (figs. 6 and 7); on the other hand, it may be relatively late (fig. 8). Following the descending branch of the right coronary artery, the times decrease. The same tendency is seen in moving from the base (dorsally) of the left ventricle to its apex. The figure for the actual apex of the left ventricle, *i.e.* the vortex, is always low (figs. 7, 8, 9, and 10), and often this region rivals the central region on the ventral surface of the right ventricle, in the race for priority.

Left Surface.

The left surface includes a portion of the conus, the whole of the left surface of the left ventricle, and the greater part of its ventral and dorsal surfaces (figs. 9, 10, and 11).

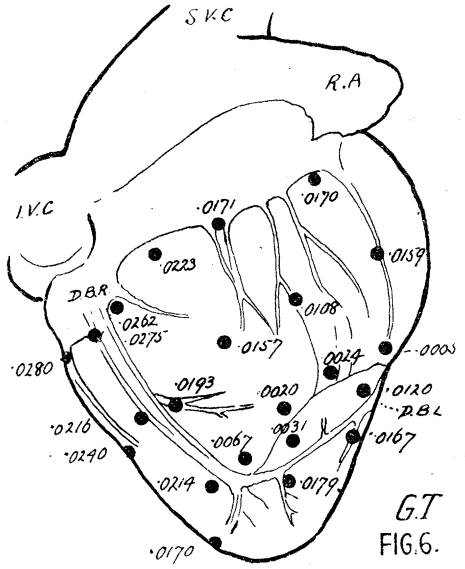


FIG.6.

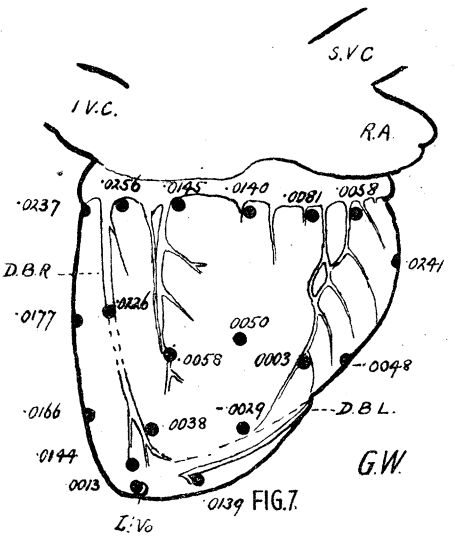


FIG.7.

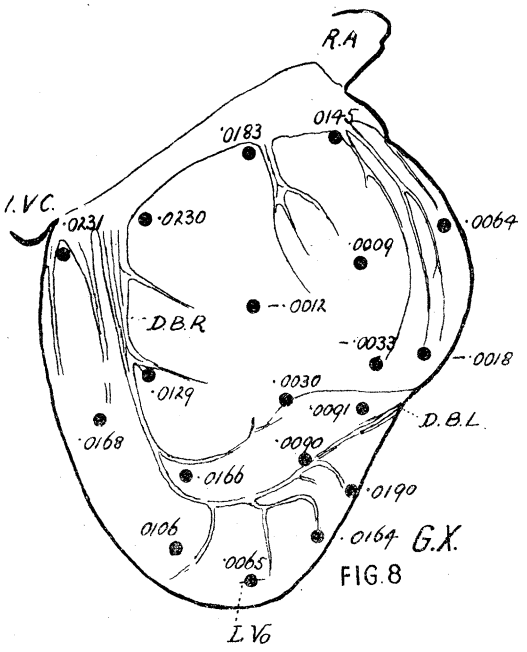


FIG.8

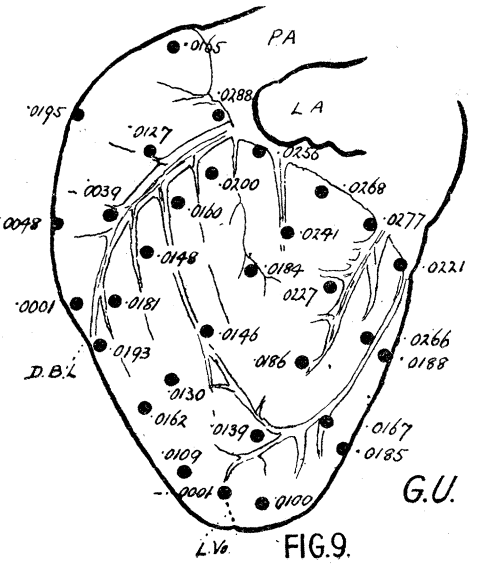


FIG.9.

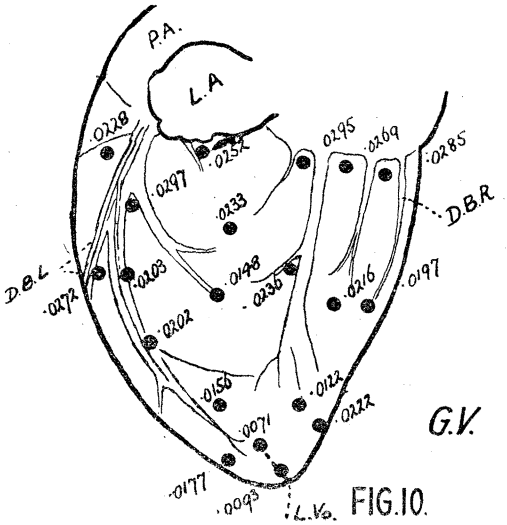


FIG. 10.

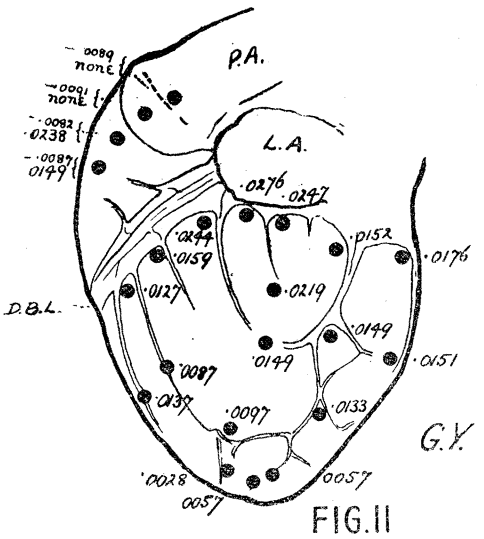


FIG. 11.

FIGS. 6, 7, and 8.—Outlines ($\frac{3}{4}$ natural size) to scale of the right surface of three dogs' hearts (G.T., G.W., and G.X.); giving the time-readings of the intrinsic deflections at a number of points. The view of the heart is not quite the same in the three diagrams. S.V.C. = superior vena cava; I.V.C. = inferior vena cava; R.A. = right appendix; D.B.R. = descending branch of right coronary artery; D.B.L. = descending branch of left artery; L.Vo. = vortex of left ventricle.

FIGS. 9, 10, and 11.—Outlines ($\frac{3}{4}$ natural size) to scale of the left surface of three dogs' hearts (G.U., G.V., and G.Y.); giving time-readings at a number of contacts. L.A. = left appendix; P.A. = pulmonary artery; D.B.R. = descending branch of right coronary artery; D.B.L. = descending branch of left artery; L.Vo. = vortex of left ventricle.

The base of the left ventricle is relatively late, the figures decrease, though not with absolute uniformity, as the apex is approached. A row of contacts near and parallel to the descending branch of the left coronary is always later than the row lying immediately to the left of it. It would be more correct, perhaps, to say that a number of points on the left ventricular surface are excited almost simultaneously, that the base lags a little, as does also the ventral border, while, when the apex is approached, there is a fall in the values; a fall which in many hearts is abrupt and limited to a relatively small area.

The Surface as a Whole.

The distribution of the excitation wave over the surface of the ventricle teaches us much in regard to the distribution through the ventricle as a whole. That the distribution does not follow the superficial muscle paths has been indicated already. That the spread is not a simple spread from muscle element to muscle element is strongly suggested by further considerations; for, both upon the surface of the right ventricle and upon the surface of the left ventricle, there are numbers of points at which the excitation wave arrives simultaneously. Furthermore, the wave appears almost simultaneously over certain parts of the right ventricle, and over certain parts of the left, areas far removed from each other. Thus the central region of the right

ventricle and the vortex of the left ventricle are frequently activated at almost the same moment; again, there is never a very material time interval between the excitation of the right and left bases at opposite margins of the heart. An assumption that the ventricle is first excited in a single region and that the active state is propagated in an orderly succession from this region and over both chambers, or over an entire ventricle, is incompatible with these observations. At the same time, the distribution is ordered upon a definite plan from which there is little departure from heart to heart. It is for this reason that the axial electrocardiogram is relatively constant in form. We are forced to assume, in the case of each heart examined, that propagation to the surface is along a large number of distinct channels. The nature of these channels is especially suggested by the relation of the earliest points of right ventricular activity to the underlying branches of the Purkinje system. The surface distribution over the greater part of the left ventricle is also compatible with the fan-like arrangement of the left Purkinje system. The division of the main stem of the bundle into right and left chief branches explains the earliest appearance of the excitation wave almost simultaneously over right and left ventricle. Compared to the distribution over the surface of the auricle, there are several remarkable contrasts. The surface of the entire ventricle becomes active in two or three hundredths of a second; in the auricle of dogs of the same weight, five hundredths of a second or more elapse before the spread is complete over chambers of much smaller dimensions; yet, as we shall presently show, the rate of propagation in the muscle of the ventricle is actually much slower than in the muscle of the auricle. In brief, propagation through ventricular muscular elements alone cannot account for the observed surface distribution. In the light of these observations it becomes clear that the much discussed questions as to whether right or left ventricle first becomes active, or as to which ventricle first contracts, are of minor importance. Neither chamber can be stated to be the first to develop surface activity. Areas of the right precede areas of the left, areas of the left precede areas of the right. The most important general feature is the rapidity with which all regions are activated.

It will also be clear that the question of priority of base or apex is also of little consequence. It is true that the apex of the right ventricle is usually active before any basal point on the surface of the same chamber, but it may pass simultaneously with certain regions of the base into activity, or may on occasion actually follow them. In any case, the interval is but a hundredth of a second, or a little more or less. Moreover, neither base nor apex is first; the first point is always situate in the central region.

In the case of the left ventricle, the vortex always leads, and if this point may be taken as the apex, then the apex of this ventricle is always active before the rest of the ventricle; but the surface no more than a centimetre away may be a hundredth part of a second later, or more, and may be as late as other points far removed from it.

The Direct Proof of a Purkinje as Opposed to a Muscle Distribution.

Before our report is complete we shall advance reasons for the view that the surface distribution is not governed by the Purkinje arrangement alone; another factor plays an important part. But at the present it may be well to describe certain experiments which clearly establish the importance of the Purkinje system.

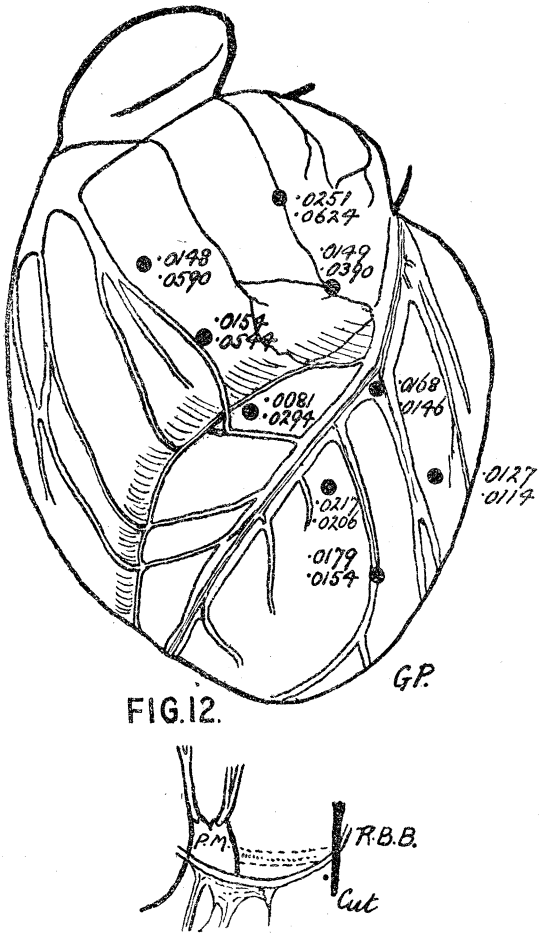
Section of the Right Bundle Division.

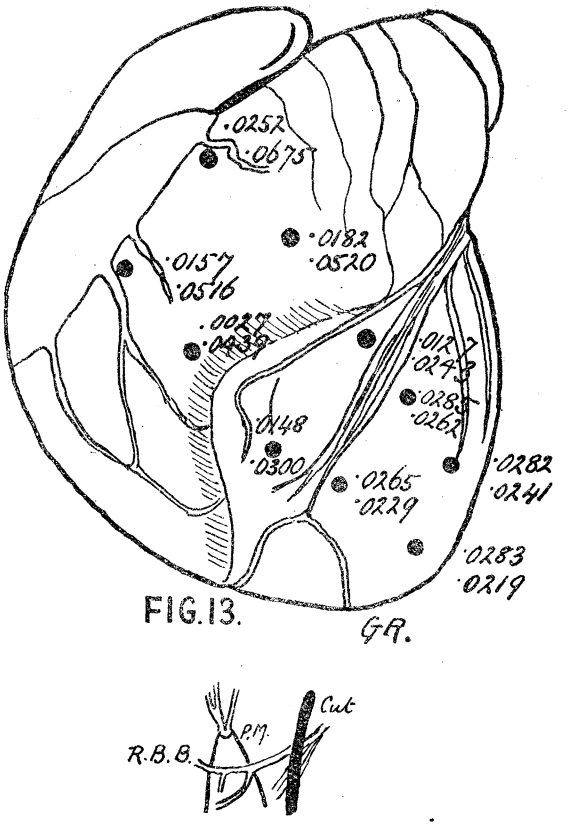
The change in an axial electrocardiogram which follows section of the main right branch of the bundle has been described by Eppinger and Rothberger.* It is very profound. The change in the curve might, in itself, be taken as proof of change in the sequence of muscular activity. We have thought it desirable to obtain clear evidence that this is so. We cut the right branch by introducing a specially contrived and guarded knife into the ventricle through the right auricular appendix. The instrument ends in a blunt point which is passed through the tricuspid orifice and thence to the septal side of the large papillary muscle; it is felt through the thin wall of the right ventricle where this joins the septum and immediately above the dimple in the trabeculated area, which forms the root of the papillary muscle. Using these landmarks, the right branch may be divided with practical certainty and cleanly, just before it curves around the papillary muscle. That the division has been complete is ascertained subsequently, the heart being specially prepared and stained for this purpose. Two examples are shown in figs. 12 and 13; the corresponding scale drawings of the incisions are placed beneath the main figures. In the heart of fig. 12 two rows of points were chosen, having special regard to their relation to vessels for the subsequent replacement of contacts. The times at which the excitation wave appeared at the nine contacts are written on the diagram; the top figure of each pair representing the reading before, the bottom figure of each pair the reading after, the section. We may first examine the effect upon the left ventricle. Four contacts lie on the left ventricle, and the section of the right bundle division is without effect. The order in which these points become active is precisely what it was. The reading before section is in each instance later than the reading after section by 1–2 thousandths of a second. This change is attributable to a change in the standard of measurement; for, after the section, R our original standard is lost and is replaced by the opening phases of an electrocardiogram of different but characteristic type (see Plate 11, fig. 26 C and D). Of the remaining contacts, four lie on the free wall and the fifth over the trabeculated region of the right ventricle. It is these five contacts which show conspicuous change. At all of them the excitation wave is markedly delayed and along each line it progresses from left to right. Whereas the contact over the edge of the trabeculated region was the first to

* 'Zeitschr. f. Klin. Med.,' vol. 70, p. 1 (1910).

show activity originally, after section, it became later than any contact over the left ventricle.

Similar changes are to be observed in the diagram of the second experiment (fig. 13); four contacts on the left ventricle show practically no change other than what may be accounted for by the alteration of standard, errors of measurement, and replacement of contacts; a conspicuous delay is seen at the two contacts on





FIGS. 12 and 13.—Outlines (natural size) to scale of the ventral surfaces of two dogs' hearts (G.P. and G.R.); showing readings at series of contacts before (top figure) and after (bottom figure) transection of the right bundle division. The relation of the cut to the bundle division and the papillary muscle in the corresponding heart is shown below in scale drawings. P.M. = papillary muscle; R.B.B. = right branch of bundle.

the trabeculated region and at the four contacts on the free wall of the right ventricle. Originally, the first contact to receive the excitation wave lay on the free wall opposite the centre of the trabeculated region (0·0027); after section the order changes and, as in fig. 12, there is a uniform sequence from left to right over the right ventricle. After the section the wave of the right ventricle was evidently transmitted from the left ventricle to the right in both experiments. Plate 11, figs. 26 and 27, are examples of the simultaneous curves from which the

readings on our diagrams have been obtained. In fig. 26 A and B, direct curves from two of the contacts are shown simultaneously with the normal standard curve from Lead *II*; in fig. 26 C and D, direct curves from the same two points are shown simultaneously with the altered electrocardiogram, the result of section. Plate 11, fig. 27, is similarly arranged. The measured interval *R* or *R'* to corresponding intrinsic deflection (*In*) is written on each curve.

Endocardial Damage.

If a contact is placed upon the conus arteriosus and the time at which the excitation wave appears is estimated, and is re-estimated after a knife edge has been drawn across the base of the conus internally, so that the endocardium is cut or scratched, a change is noted. There is a delay in the appearance of the excitation wave after section. Four experiments of this kind are tabulated in Table II. Here the time

TABLE II.—Natural Conus Intrinsic Deflection before and after Endocardial Section. (Readings related to intrinsic from point below section.)

Dog.	Length of incision.	Depth.	Thickness of wall.	Intrinsic.	
				Before.	After.
H.D.	30 mm.	$1\frac{1}{2}$–4 mm.	4–5 mm.	−0·0015	0·0028
H.E.	27 ,,	0–3 ,,	5–6 ,,	0·0100	0·0330
H.F.	Endocardium scratched in places over a line of 15 mm.		2–5 ,,	0·0120	0·0334
H.G.	Endocardium scratched over 20 mm.		5 ,,	0·0031	0·0238

difference between two contacts, one below and the other above the section, is given before and after the section. (A diagram of the corresponding pericardial cut is given in fig. 14.)

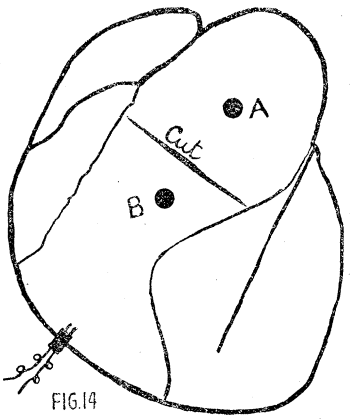


FIG. 14.—A diagram to illustrate the method of showing that the conduction interval between two contacts is uninfluenced by cutting the superficial muscle fibre lying between them. The result is the same whether the natural or excited beat is studied. In the latter case the muscle contacts are placed in line with the stimulating electrode. Each muscle contact is paired with its own chest wall contact.

This experiment affords confirmatory evidence of the distribution through Purkinje tissue.

Section of Smaller Free Branches.—On several occasions we have successfully cut one or more of the free branches traversing the cavity of the right ventricle, using a knife edge concealed in an instrument formed like a shepherd's crook. This procedure alters the axial electrocardiogram, producing a diminution of R and an increase of S.

Muscle Section.

If a contact (fig. 14, A) is placed upon the conus and a cut is made below it and across the base of the conus, penetrating the muscle fibres for a few millimetres, the time at which the excitation wave appears relative to its appearance at a contact below the cut (fig. 14, B) is uninfluenced (see Table III).

TABLE III.—Natural Conus Intrinsic Deflection (from point A) before and after Pericardial Section.

(Readings related to intrinsic from point (B) below section.)

Dog.	Length of incision.	Depth.	Thickness of wall.	Intrinsic.	
				Before.	After.
H.D.	31 mm.	1–1½ mm.	3½–6 mm.	0·0092	0·0091
H.E.	22 ,,	2–3 ,,	3½–7 ,,	0·0210	0·0192
H.G.	26 ,,	2–3 ,,	4–5 ,,	0·0099	0·0105

Further, if we choose an area upon the left ventricle, where there are no conspicuous vessels, and four deep cuts are made joining each other to form a complete square around a central contact, the appearance of the excitation wave is found to be uninfluenced by these cuts (Table IV). In performing this experiment we have to be careful to obtain the second curves as rapidly as possible after the sections are complete, and before change in the muscle can occur from disturbance of the vascular supply.

TABLE IV.

Dog.	Before.	After.	Depths of cuts.	Thickness of muscle.
H.E.	0·0251	0·0240	5–7 mm.	13 mm.
H.F.	0·0223	0·0226	4–8 ,,	8–13 ,,
H.G.*	0·0159	0·0133	4–6 ,,	7–8 ,,

* In the same animal a cut parallel to the pericardial surface in another region and between the muscle layers gave delay. The readings before and after the cut were 0·0216 and 0·0285 respectively.

This experiment shows conclusively that the excitation wave spreads from within outwards, and is propagated in the ventricle without regard to the arrangement of the muscle bands.

Rates of Propagation.

What the rate of propagation in the mammalian ventricle may be is a problem not easy to solve. It has been attempted by Gotch; he used a base-apex lead and measured the interval between the onset and culmination of the chief deflection, considering this time to be the base-apex transmission interval. Now a measurement of this kind is justifiable only if it be known beforehand that the natural excitation wave has a simple spread along the muscle fibres and exactly in the line of the contacts. Both premisses were assumed by Gotch; but, as we can clearly show, neither is warranted by the facts. It is impossible to calculate and express the rate of propagation even approximately, from the times at which the natural excitation wave appears at given points on the ventricular surface, even if we have accurate estimates of these readings. The course of the excitation wave, relative to a pair of surface points, is not the same for different regions of the heart. If we use our own estimates of the surface readings in this fashion, the calculated rate varies from several hundred millimetres per second to infinity. Evidently the wave may start between two contacts. Still more important, it may not spread to any extent from one muscle element to another, but may be separately initiated in a large number of small areas through a special system of distribution; such is indeed our thesis.

It is necessary at the outset to study the simplest forms of propagation, forms of propagation which are, in a measure, subject to control. For this purpose we promote artificial waves of excitation in the ventricle by stimulating it with rhythmic and threshold induction shocks at a rate in excess of the natural heart rhythm. Two contacts are placed at a measured distance apart upon the ventricle, and contractions are forced from a point in line with them (see fig. 14). The interval between the time-readings at the two contacts, each paired to an indifferent contact on the chest wall, gives us a measure of conduction rate over the muscle strip examined.

Influence of Heart Rate.—We made a number of preliminary observations upon the influence of heart rate. This we considered to be important, seeing that in all experiments upon forced rhythms the heart rate is enhanced. Our results are shown in Table V. This Table includes the intervals for natural and excited rhythm, measured alternately. We also give the distance of contacts from each other, and the distance of the proximal contact (that which first receives the excited wave) from the point of stimulation. We find that the rate of the excited rhythm may vary widely without producing appreciable change in the rate of propagation. There may be a little lowering of conduction rate as the heart rate enhances, but it is trifling in experiments in which rates of 170 or 200 to 300 are tested. It will be noticed in this and succeeding Tables, that the interval for the natural beat is always much shorter

than the interval for the excited beat (Plate 12, fig. 28); an observation which has also been made by ERFMANN.

Influence of Fibre Direction.—We studied this question over the right ventricle (Table VI) and found higher rates in the direction of the superficial fibres than across them, for the most part; but our results did not bring us satisfaction for the reason that regions of the ventricle studied for straight or cross conduction necessarily varied in respect of underlying structures. Over the left ventricle, where muscle regions can be chosen in which this factor is less disturbing, we could find little or no difference in rate (Table VII). Our results are sufficient we think to show that the direction of the superficial fibres has no material influence. But these studies brought out a fact of very considerable importance.

TABLE V.—Transmission Rates.

(Influence of Heart Rate.)

Dog.	Contacts.	Contacts apart in mm.	Natural beat.		Excited beat.			
			Heart rate.	Interval in secs.	Heart rate.	Interval in secs.	Stimulus to proximal contact in mm.	Transmission rate in mm. per sec.
G.E.	Apex to base of R.V. . .	22	206	0·0022	238	0·0145	35	1507
			184	0·0028	348	0·0182		1208
G.F.	Mid R.V. to conus . . .	27	167	0·0144	170	0·0227	21	1189
			168	0·0137	194	0·0223		1211
			164	0·0136	223	0·0239		1129
G.H.	Mid R.V. to conus . . .	17	139	0·0051	158	0·0119	17	1429
			150	0·0053	171	0·0109		1559
			147	0·0059	194	0·0111		1531
			145	0·0056	204	0·0119		1429
			146	0·0056	244	0·0109		1559
			146	0·0051	258	0·0113		1504
			143	0·0054	294	0·0109		1559
G.I.	Central region to conus .	13	214	0·0048	218	0·0078	Constant	1667
			216	0·0054	249	0·0085		1528
			214	0·0053	274	0·0088		1477
G.J.	Central region to base R.V.	28	177	0·0107	179	0·0166	Constant	1686
			172	0·0121	194	0·0163		1718
			174	0·0110	224	0·0165		1696
			170	0·0109	249	0·0169		1656
			169	0·0120	274	0·0171		1644
			152	0·0102	342	0·0173		1618
G.G.	Apex R.V. to conus . .	22	140	0·0182	185	0·0219	22	1004
			89*	0·0184	221	0·0221		996

* Heart dilating.

TABLE VI.—Transmission Rates in Right Ventricle.

Dog.	Contacts.	Contacts apart in mm.	Natural beat.		Excited beat.			
			Heart rate.	Interval in secs.	Heart rate.	Interval in secs.	Stimulus to proximal contact in mm.	Transmission rate.
			1. Leading in Direction of Fibres.					
G.F.	Mid R.V. to conus .	27	168	0·0137	194	0·0223	21	1211
G.E.	Apex to base of R.V.	22	206	0·0022	238	0·0145	35	1507
G.G.	Apex R.V. to conus .	22	140	0·0182	185	0·0219	22	1004
G.H.	Mid R.V. to conus .	17	147	0·0059	194	0·0111	17	1531
G.I.	Central region to conus	13	216	0·0054	249	0·0085	—	1528
G.I.	Conus to conus . .	17	212	0·0101	282	0·0114	—	1491
G.J.	Central region to base R.V.	28	170	0·0109	249	0·0169	—	1656
G.O.	Mid R.V. to base R.V.	22	100	0·0076	184	0·0103	23	2136
G.P.	Mid R.V. to base R.V.	22	159	0·0086	182	−0·0012	15	Infinity
			2. Leading Across Fibres.					
G.O.	Central region to right margin	19	109	0·0143	189	0·0335	15	570
G.O.	Central region to right margin	19	112	0·0160	185	0·0223	34	842
G.N.	Central region to right margin	13	215	0·0081	262	0·0119	21·5	1092
G.N.	Central region to right margin	14	227	0·0074	258	0·0131	20	1068
G.M.	Mid R.V. across . .	13	121	0·0039	172	0·0142	15	916
G.L.	Central region to right margin	14	160	0·0102	189	0·0184	16	761
G.K.	Conus to conus . . .	16	134	−0·0018	184	0·0119	35	1344
G.J.	Conus to conus . . .	19	183	0·0042	206	0·0390	—	487
G.J.	Conus to conus . . .	19	182	0·0035	212	0·0405	—	469
G.P.	Mid R.V. across . .	16	163	0·0001	185	0·0097	9	1679
G.R.	Across R.V.	14	169	0·0070	186	0·0157	43	891

TABLE VII.—Transmission Rates in Left Ventricle.

Dog.	Contacts apart in mm.	Natural beat.		Excited beat.			
		Heart rate.	Interval in secs.	Heart rate.	Interval in secs.	Stimulus to proximal contact in mm.	Transmission rate in mm. per sec.
		1. Leading in Direction of Fibres.					
G.N.	13	217	−0·0009	255	0·0200	13	650
G.O.	19	119	0·0017	193	0·0356	8	534
G.P.	14	158	0·0000	194	0·0241	11	581
		2. Leading Across Fibres.					
G.N.	13	204	−0·0001	265	0·0306	14	424
	13	213	−0·0051	258	0·0311	15	418
G.O.	17	113	−0·0028	198	0·0371	9	459
G.P.	22	156	−0·0050	191	0·0294	10	749

TABLE VIII.—Transmission Rates across Interventricular Groove.

Dog.	Contacts.	Contacts apart in mm.	Natural beat.		Excited beat.			
			Heart rate.	Interval in secs.	Heart rate.	Interval in secs.	Stimulus to proximal contact in mm.	Transmission rate.
G.F.	Aortic base to conus .	25	166	0·0241	181	0·0578	10	432
G.I.	Aortic base to conus .	23	202	−0·0018 0·0082	264	0·0379	10	607
G.I.	L.V. to mid R.V. . .	21	208	−0·0099	262	0·0235	—	894
G.I.	L.V. to apex R.V. . .	19	189	−0·0097	258	0·0239	—	795
G.J.	L.V. to central region R.V.	19	178	−0·0189	198	0·0283	—	671
G.J.	L.V. to central region R.V.	37	179	0·0091	202	0·0771	—	480
G.K.	Trabeculated area to mid conus	22	128	0·0080	180	0·0623	15	333
G.K.	Mid L.V. to mid R.V.	22	124	−0·0174	177	0·0570	20	386
G.L.	Trabeculated area to R.V.	23	158	−0·0050	186	0·0509 0·0587	10	452 392
G.M.	Trabeculated area to R.V.	22	115	−0·0072	174	0·0529	18	416
G.M.	L.V. to mid R.V. . .	24	112	−0·0082	182	0·0567	9	423
G.N.	L.V. to mid R.V. . .	17	225	−0·0133	242	0·0416	8	409
G.N.	L.V. to trabeculated area	14	211	−0·0112	248	0·0186	15	754
G.O.	L.V. to mid R.V. . .	17	112	−0·0192	188	0·0271	8	627
G.O.	L.V. to trabeculated area	10	112	−0·0118	189	0·0200	8	500

Influence of Muscle Thickness.—Excited waves are propagated much more slowly over the surface of the left ventricle than over the surface of the right, and conduction is also invariably slow across the interventricular groove (Table VIII). The calculated rates for the right ventricle vary usually between 1000 and 1500 mm. per second (Plate 12, fig. 28). Those for the left ventricle vary usually between 400–600 mm. per second; the rates over the interventricular groove (Plate 12, fig. 29) and between points on and off the trabeculated region are of similar magnitude. There is a distinct and constant variation of rate over certain areas of the heart which we can attribute only to the constitution of the deeper structures. Our view is that the conduction rate in muscle is slow, and in the Purkinje system fast; and that when the surface of the heart is stimulated, the excitation wave penetrates the muscle and enters the Purkinje network. When the wall examined is thin, conduction will be more through the Purkinje substance and less through muscle; when the wall is thick, the reverse will be the case; so in the one instance conduction will be more rapid and in the other instance less rapid. Such at all events was the hypothesis which we determined further to examine. If the conditions are as we imagine them to be, and two contacts are placed on the right

ventricle a considerable distance from the point of stimulation, the one contact on the outer surface and the other upon the inner or endocardial surface, we should expect to find that the internal contact is the first to receive the excitation. Such proves to be the case. Now, when the heart beats naturally, activity is always signalled by an internal contact before it is signalled by an external contact placed immediately over it. When the heart is stimulated at a surface point some distance from such contacts, not only does the endocardial surface receive the excitation wave before the epicardial surface, *but it does so by precisely the same time interval as during the natural heart beat* (Table IX, Plate 12, fig. 30, italicised figures). This statement is only true, however, when the point of stimulation is sufficiently distant. The muscle surface is stimulated and the excitation wave spreads by two routes. It spreads over the superficial muscle towards the external contacts (path *aa* in fig. 15), it penetrates the muscle, enters the Purkinje substance and reaches the internal contact, to penetrate the muscle once more and reach the external contact (path *bb* in fig. 15). But the distance of the stimulating electrode from the contacts is of prime importance. If it is but a few millimetres, the path *aa* will be as quick as the other or quicker. By measuring the thickness of muscle and the distance between stimulating electrodes and contacts, we may arrive at certain conclusions in respect of the relative rates of conduction. Take, for example, the last experiment of Table IX. The muscle is 6 mm. in thickness and the distance 15 mm. The path through 6 mm. muscle + 15 mm. Purkinje + 6 mm. muscle is preferred to the path through 15 mm. muscle; or, briefly, 15 mm. of Purkinje are preferred to 3 mm. of muscle. We have clear evidence from this and other examples that the rate of travel through Purkinje substance is much faster than through muscle. We calculate

TABLE IX.—Transmission from Internal to External Contacts.

Dog.	Contacts.	Contacts apart in mm.	Natural beat.		Excited beat.			Natural transmission rate (mm. per sec.).
			Heart rate.	Interval (internal to external).	Heart rate.	Interval (internal to external).	Stimulus to external contact in mm.	
G.L.	Base of R.V. internal to external	4	115	*0·0095*	215	*0·0098*	*32*	421
			115	0·0092	185	0·0071	10	
G.N.	Base of R.V. internal to external	4	113	*0·0113*	172	*0·0109*	*18*	354
			113	0·0113	176	0·0026	10	
G.O.	Base of R.V. internal to external	6	112	*0·0205*	192	*0·0226*	15*	293
			113	*0·0207*	188	*0·0227*	15†	
			109	0·0204	193	0·0170	8†	

* Stronger stimulus.

† Threshold stimulus.

that it is at least three to five times as fast, regarding the Purkinje tissue not as a network, but as a straight band of fibres.

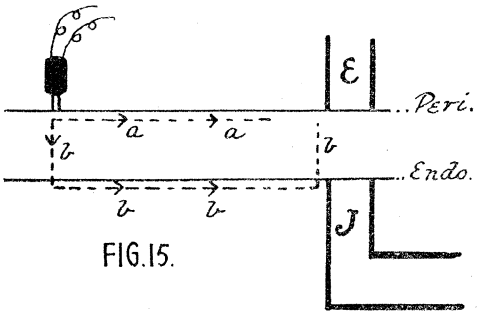


FIG. 15.

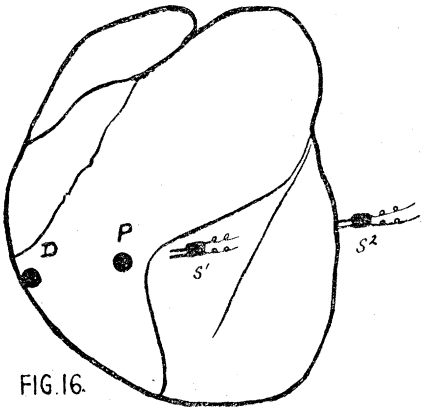


FIG. 16.

FIG. 15.—A diagram showing the ventricular wall in section and the paths travelled by the excitation wave, from the point of stimulation to the external and internal contacts.

FIG. 16.—A diagram illustrating an arrangement of contacts, with the stimulating electrodes placed near to, and far from, the proximal contact. When the ventricle is excited at S^2 the interval between the appearance of the excitation wave at P and D is shortened.

The distance of the point of stimulation from the contacts shows its influence in an experiment of a somewhat different type. Two contacts (P and D) are placed across the right ventricle (fig. 16), and a point of stimulation is chosen a little to the left (lying usually over the trabeculated region or near its border) and in line with them (S^1). The time interval between the contacts is ascertained for natural and excited beats; the latter, as usual, exceeds the former. The stimulating electrodes are now moved along the same line to the left border of the left ventricle (S^2) or towards it, thus increasing their distance from the proximal contact (P). The time interval between the two contacts is invariably reduced by this procedure; and the reduction, more often than not, is such that the interval for excited and natural beats becomes the same (Table X, examples marked with asterisks). Our explanation is that the excitation wave, instead of taking the shorter but slower path through the muscle of the septum, prefers the longer but faster path through the left division of the bundle over the septum and down the right division to the muscle underlying the contacts. The race along the two paths is often a close one, and consequently the reduction is not always so extensive; but in these circumstances the new value for the excited beat lies between its former value (when the stimulating electrodes are close up) and its value for the natural beat. That the endocardial surface, and presumably the Purkinje network, is followed when an excitation wave spreads from a point of stimulation can be shown quite clearly. We place two contacts on the right ventricle and stimulate at a point some distance away but in line with them (fig. 14). The time interval between the appearance of the excitation wave at the two contacts is established, and a long incision is then made between them and at right angles to them. The incision penetrating the superficial muscle fibres for one-third or one-half their thickness does not

TABLE X.—Transmission Rates.

(Influence of distance of stimulation point.)

Dog.	Contacts.	Contacts apart in mm.	Natural beat.		Excited beat.			
			Heart rate.	Interval in secs.	Heart rate.	Interval in secs.	Stimulus to proximal contact in mm.	Transmission rate in mm. per sec.
G.K.	Central region to margin R.V.	17	128	0·0060*	182	*0·0066**	*41*	*2758*
G.L.	Central region to margin R.V.	14	164	0·0087*	199	*0·0104**	*38*	*1346*
			160	0·0102*	189	0·0184	16	761
G.M.	R.V. across	15	137	0·0044*	169	*0·0042**	*42*	*3571*
		13	121	0·0039	172	0·0142	15	916
G.N.	,, ...	14	227	0·0070*	242	*0·0092**	*42*	*1522*
			227	0·0074	258	0·0131	20	1068
G.O.	Mid R.V. to base R.V. .	19	112	0·0160	185	*0·0223*	*34*	*857*
			109	0·0143	189	0·0335	15	570
G.P.	,, ,, .	16	167	−0·0008	190	*0·0036*	40	*4444*
			163	0·0001	185	0·0097	16	1650
G.R.	,, ,, .	14	169	0·0070	186	*0·0157*	*43*	*892*
			173	0·0045	184	0·0266	13	527
G.R.	,, ,, .	11	173	0·0091*	184	*0·0087**	*40*	*1264*
			169	0·0072	179	0·0216	11	509
G.Z.	R.V. across	17	115	0·0066*	195	*0·0065**	*45*	*2461*
			115	0·0061	198	0·0302	17	530

* Note the approximation of these readings in the columns referring to natural and excited beats.

influence conduction in the least (Table XI). The wave is conveyed across the incision just as readily as it was conveyed across the same region in its undamaged state.

TABLE XI.—Excited Conus Intrinsic Deflection before and after Pericardial Section.

(Readings related to intrinsic from conus point below section.)

Dog.	Length of incision.	Depth.	Thickness of wall.	Intrinsic.	
				Before.	After.
H.D.	31 mm.	1–1½ mm.	3½–6 mm.	0·0208	0·0192
H.E.	22 ,,	2–3 ,,	3½–7 ,,	0·0143	0·0143
H.G.	26 ,,	2–3 ,,	4–5 ,,	0·0177	0·0160

Clearly this can occur only if the original course was a deep one. So it happens that if a similar cut is made on the endocardial surface the excited wave is interfered with, the time interval increasing conspicuously (Table XII).

TABLE XII.—Excited Conus Intrinsic Deflection before and after Endocardial Section (Readings related to intrinsic from conus point below section.)

Dog.	Length of incision.	Depth.	Thickness of wall.	Intrinsic.	
				Before.	After.
H.D.	31 mm.	1½–4 mm.	4–5 mm.	0·0026	0·0126
H.E.	27 „	0–3 „	5–6 „	0·0112	0·0234
H.F.	Endocardium scratched in places over 15 mm.		2–5 „	0·0104	0·0247
H.G.	Endocardium scratched over 20 mm.		5 „	0·0057	0·0274

Actual Rates of Propagation.

To speak of the rate of propagation in the ventricular wall would bring us no nearer the essential problem. We have, however, to discuss the actual rates of propagation in its two elements, ventricular muscle and Purkinje substance. The high rates of conduction along the thin wall of the right ventricle are due according to our view to escape into the Purkinje tissue, the low rates along the thicker muscle of the left side to the relatively lower powers of conduction in the muscle itself. Seeing that there is no reason to believe that the rate of conduction in the muscle is variable in different regions, or that there is appreciable variation according to the direction of the superficial fibres, we ascribe all the chief variations in rate to early or late escape to the Purkinje network. Holding this view we regard the lowest conduction rates obtained, *i.e.*, those approaching 300 or 500 mm. per second, as representing the real ventricular rate most closely; while we regard the highest rates, *i.e.*, 1500 or 2000 mm. per second, as low estimates* of the network rate. These estimates would suggest that network conduction is approximately five times as fast as muscle conduction; an estimate in remarkably close accord to a previous one.

But network conduction does not fully represent Purkinje conduction, for the fibres of the network are not straight, but are, for the most part, very wavy. Conduction in such strands as contain straight fibres, *i.e.*, the chief divisions of the bundle, should be 50 or 100 per cent. faster.

Our estimate for Purkinje substance as such is from 3000 to 5000 mm. per second and we favour the last figure rather than the first.†

* Low because these calculations are for Purkinje substance with two short stretches of *muscle* added.

† It seems clear from our experiments that if an excitation is artificially propagated from the ventricular surface this wave rapidly finds its way into the Purkinje system. It is also evident

Our estimate of conduction rates in the three tissues of the heart which are concerned may be stated as follows :—

Purkinje tissue	3000–5000 mm. per sec.
Auricular muscle	1000 mm. per sec.
Ventricular muscle	300–500 mm. per sec.

In harmony with these estimates is the fact that by far the highest glycogen content of the heart is to be found in Purkinje tissue (NAGAYO) while auricular muscle is stated by BERBLINGER* to be more heavily laden than ventricular. The functional significance of these differences in the power of conduction will be alluded to a little later.

THE ENDOCARDIAL LINING.

Observations upon the endocardial surface of the ventricle present many natural difficulties. We have employed many different forms of electrode; the most satisfactory results are obtained with single and rigid contacts of glass tubing curved to suit the point or points examined. The glass tube has a broad outlet filled by a kaolin plug; the shaft of the tube contains copper sulphate and a copper wire, the opening and an inch or more of the shaft being sealed with hard paraffin wax. The contact is introduced through an auricular appendix and paired with a second contact upon the chest wall. In one or two experiments closely set double contacts were used internally, but these proved less satisfactory. The introduction of an internal electrode disturbs the position of the heart to some extent and in unfortunate experiments may lead to serious disturbance of mechanism.

We reject all observations in which the normal form of axial electrocardiogram is not maintained. All internal leads are undertaken as the final observations of a given experiment. Most of our readings have been taken from the lining of the free wall of the right ventricle, and these are the only observations which we regard with entire confidence. In studying this region with a rigid curved electrode the thin muscular wall may be lifted a little on the contact, so that it bulges externally and so that the contact may be easily felt through the wall and its exact position ascertained and maintained. In the circumstances there is no uncertainty, the contact is known to be satisfactory. But when we attempt to obtain readings from the septum on the

that the rate of propagation in the system is very rapid. When an idio-ventricular rhythm dominates the movements of the ventricle and premature contractions are forced from the ventricle, the length of the returning cycle (see first article) is little if at all longer than an initial or natural cycle. This fact has never received adequate explanation. It now seems that the time lost by the excitation wave in travelling from the point stimulated to the rhythm centre in the auriculo-ventricular bundle would not be appreciable. If the natural ventricular cycle has a duration of a second or more, an addition of two or three hundredths of a second would pass unnoticed.

* 'Beiträge z. pathol. Anat. u. allg. Pathol.,' vol. 53, p. 207 (1912).

right side, and especially when we introduce the electrode into the left chamber, accurate location is almost impossible.*

The power of the left ventricle is so great and the wall so rigid that the electrode may be unmanageable and the fling very considerable. Consequently we lay most stress upon readings from the thin and free wall of the right chamber.

Internal and External Readings.

If we place one electrode upon the inner and the other upon the outer surface immediately over it, so that the two contacts grip the muscle lightly between them; then the internal contact invariably receives the excitation current first. This fact is clearly shown by Table XIII. The interval between the two readings (internal and

TABLE XIII.—Comparison of Internal and External Leads.
(Appearance of extrinsic and intrinsic deflections relative to *R* in Lead *II.*)

Dog.	Extrinsic.		Intrinsic.		Contacts.*
	Internal.	External.	Internal.	External.	
G.A.	None	−0·0033	−0·0015	0·0131	On anterior papillary muscle and over same.
G.F.	None	−0·0108	−0·0082	0·0088	On anterior papillary muscle and over same.
G.J.	−0·0101	−0·0104	−0·0023	0·0082	Base of R.V. and over same.
	−0·0098	−0·0110	−0·0013	0·0020	Apex of R.V. and over same.
G.K.	−0·0068	−0·0056	−0·0032	0·0172	Base of R.V. and over same.
	−0·0059	−0·0067	−0·0046	0·0069	Central region and over same.
G.L.	−0·0077	−0·0048	−0·0005	0·0147	Base of R.V. and over same.
	−0·0120	—	−0·0010	0·0049	Central region and over same.
G.M.	−0·0110	−0·0087	−0·0022	0·0102	Mid R.V. and over same.
	−0·0068	−0·0069	−0·0022	0·0100	Base of R.V. and over same.
H.C.	—	—	−0·0001	0·0213	Base of R.V. and over same.
	−0·0118	−0·0073	−0·0029	0·0094	Apex of R.V. and over same.
	−0·0109	−0·0073	−0·0020	0·0035	Central region and over same.

* With the exception of the first two experiments, in which double contacts were used, each contact named was paired with a chest wall contact.

external) is not the same for different regions of the wall. It is greatest over the base near the auriculo-ventricular groove, where the muscle is thick, and least in the central region where the muscle is thinnest. If we measure this muscle thickness and calculate the rate of propagation on the basis of the corresponding internal-external interval (Table IX) we find it to be about 300–400 mm. per second, values which harmonise

* We are helped in the location of such contacts by running a long pin through the heart near the contact while the electrode is *in situ*.

with our former calculation of the rate in ventricular muscle. We consider therefore that the time of appearance of the excitation wave at the external contact is governed largely by the thickness of muscle underlying it. Eventually this subject will be discussed more fully.

In Table XIV we have summarised all our endocardial readings, and may briefly consider them from the regional standpoint.

Central Region.—There are 6 readings (0·0046, −0·0010, −0·0030, −0·0002, −0·0026 and −0·0020) from this region.

The endocardial surface becomes excited as a general rule a little before R has its onset. The figures cannot strictly be compared with each other; it is necessary to take into account the external surface distribution in each heart; for this reason we

TABLE XIV.—Deflections from Internal Contacts.

Dog.	Contact.	Intrinsic.	Extrinsic.	Q.	Earliest external intrinsic.	Earliest external extrinsic.
G.F.	R.V. anterior papillary muscle	−0·0082	Absent*	−0·0058	−0·0031	−0·0111
	Same a little higher	−0·0072	Absent*			
G.J.	R.V. base	−0·0023	−0·0101	−0·0058	−0·0029	−0·0121
	R.V. apex	−0·0013	−0·0098			
G.K.	R.V. base	0·0032	−0·0068	−0·0053	0·0037	−0·0077
	R.V. central region	0·0046	−0·0059			
G.L.	R.V. base	−0·0005	−0·0077	−0·0096	0·0021	−0·0161
	R.V. central region	−0·0010	−0·0120			
	Same (a little higher)	0·0002	−0·0166			
G.M.	R.V. mid	−0·0022	−0·0110	−0·0048	0·0000	−0·0077
	R.V. anterior papillary muscle	−0·0008	−0·0203?			
	R.V. mid-septum	−0·0033	−0·0075			
	R.V. base	−0·0022	−0·0068			
G.C.	R.V. high septal	0·0243	—*	−0·0063		
G.T.	R.V. mid	−0·0025	−0·0070	−0·0081	−0·0005	−0·0080
G.U.	L.V. apex	0·0110	−0·0077	−0·0105	−0·0001	−0·0132
	L.V. mid-septal	−0·0036	−0·0139			
G.V.	L.V. apex	−0·0019	−0·0100	—	—	−0·0096
	L.V. mid-septal	−0·0085	—			
G.W.	R.V. apex	−0·0041	−0·0172	0·0107	−0·0048	−0·0160
	R.V. central region	−0·0030	−0·0087			
	R.V. base	−0·0016	−0·0134			
	R.V. high septal	0·0144	−0·0075			
	R.V. conus	0·0038	−0·0150			
	L.V. high septal	−0·0068	−0·0192			

* Double contacts.

TABLE XIV—*continued.*

Dog.	Contact.	Intrinsic.	Extrinsic.	Q.	Earliest external intrinsic.	Earliest external extrinsic.
G.Z.	R.V. apex	0·0003	−0·0078	−0·0048 (approx.)	0·0024	−0·0086
	R.V. base	0·0009	−0·0086			
	R.V. conus (high) .	0·0025	−0·0070			
	R.V. central region .	−0·0002	−0·0078			
G.X.	R.V. apex	−0·0003	−0·0090	−0·0082	−0·0033	−0·0138
	R.V. conus	0·0202	−0·0089			
	R.V. base	0·0138	−0·0100			
G.Y.	L.V. apex	−0·0027	−0·0142	−0·0066	0·0028	−0·0156
	L.V. mid-septum . .	0·0066	—			
	L.V. high septum .	0·0022	—			
H.B.	R.V. apex	0·0031	−0·0088	−0·0041	−0·0010	−0·0099
	R.V. base	0·0018	−0·0084			
	R.V. central region .	−0·0026	−0·0094			
	R.V. papillary muscle	−0·0039	−0·0115			
	R.V. mid-septum . .	0·0060	−0·0098			
	R.V. conus	0·0092	−0·0057			
	R.V. conus (higher) .	0·0127	−0·0035			
H.C.	R.V. apex	0·0029	−0·0118	None	−0·0038	−0·0083
	R.V. base	−0·0001	—			
	R.V. central region .	−0·0020	−0·0109			
H.D.	L.V. apex	0·0031	—	—	—	—

include a column in our Table showing the earliest external reading obtained over the central region. In the case of the relatively high figure + 0·0046 which stands amongst internal readings, it is to be noted that the corresponding external readings were also higher than usual.

Apex of Right Ventricle.—From this region there are six readings (−0·0013, −0·0041, 0·0003, −0·0003, 0·0031 and 0·0029). The readings are of similar order to those of the central region. Despite this fact, the epicardial surface of the apical region reads higher than that of the central region; this is to be accounted for by the greater thickness of muscle beneath the former.

The Base of the Right Ventricle.—There are nine readings from this region (−0·0023, 0·0032, −0·0005, −0·0022, −0·0016, 0·0009, 0·0138, 0·0018, −0·0001) and two from an area between this and the central region (−0·0022 and −0·0025). The base readings are higher than those of the central region, but not so high as might have been anticipated from the distribution of the Purkinje system to this region. One figure 0·0138 is exceptionally high; we do not regard it as representative, and view it with some suspicion; it is possible that the corresponding contact was placed too near the base.

The Conus Region.—There are five readings (0·0038, 0·0025, 0·0202, 0·0092 and 0·0127). We have one exceptionally high reading in this series too; the remainder being tolerably uniform. Two of the readings (0·0092 and 0·0127) were taken from the same animal, the last (0·0127) being obtained from a contact immediately beneath the pulmonary valves. The Purkinje path to the conus is the longest of any in the right ventricle; relatively high figures were therefore to be anticipated. The muscle of the conus is as a rule no thicker than that at the base of the right ventricle; the high external readings over the conus, as compared to the base, are consequently to be attributed to the relations of the conducting system to these two portions of the wall.

We pass to less satisfactory observations upon other internal surfaces.

The Papillary Muscle.—We have attempted to obtain readings from the large muscle in the right ventricle, marking our contact point by transfixing the heart. Three contacts seemed to us to have been successfully placed and the readings are relatively low (−0·0082, −0·0008 and −0·0039). The papillary muscle seems to be one of the earliest points to become active in the right ventricle; the intrinsic deflection distinctly preceding the onset of *R*. *R* has been attributed by NICOLAI to activity of this muscle; our results do not support this view.

Right Side of Septum.—Our readings are variable, −0·0033 and 0·0060 for the mid-septal region, and 0·0243 and 0·0144 for the high septal region. These last two readings are very high, but not incompatible with the anatomical conditions. It will be remembered that Purkinje fibres in the neighbourhood of the septal cusp of the tricuspid valve are apparently sparse or absent, and this is the region upon which the contacts lay.

Apex of Left Ventricle.—Of four readings (0·0110, −0·0019, −0·0027, 0·0031) one is exceptionally high and we are at a loss to explain it. It should be remembered that readings are obtained after the experiment has terminated and that we do not become aware of such exceptions until it is too late to repeat the observations. We must accept them therefore for what they are worth, though we cannot but suspect that from time to time some unnoticed factor has crept in. Single high readings have also been noted from the base and conus of the right ventricle. We have attempted to ascertain the significance of these occasional high readings without success. When an electrode is introduced into a heart cavity, it is in contact with the whole endocardial lining through the blood; but that this introduces no appreciable error is clear from a specially devised experiment. We place a contact upon the outer surface of the heart, and estimate the time for this point before and after flooding the pericardium, slung as a hammock, with blood. The two readings are very similar (Table XV).

We thought that possibly contacts on the endocardial lining might have been completed only at the commencement of systolic contraction in certain instances.

TABLE XV.—Deflections before and after Flooding Pericardium.

Dog.	Region tested.	Extrinsic.	Intrinsic.
H.B.	L.V. near apex	−0·0070	0·0192 Before.
		−0·0067	0·0168 After.
H.C.	R.V. near apex	−0·0076	0·0137 Before.
		−0·0056	0·0177 After.

To test the effect of a contact jumping in this fashion, we arranged an electrode on the external surface, so that contact was made in systole only, the contact being covered with free blood in the pericardium. Subsequently we took a curve from the nearest ventricular point with the contact well maintained. The result threw no light upon our exceptional values (Table XVI).

TABLE XVI.

Dog.	Contact.	Extrinsic.	Intrinsic.
H.B.	Jumping	−0·0048	0·0102
	Constant	−0·0067	0·0091
H.C.	Jumping	−0·0091	0·0022
	Constant	−0·0073	0·0009

As a matter of fact a simple blood-contact in the centre of the right ventricular cavity gives a low value (Dog G.Z., 0·0011 ; Dog H.B., −0·0027).*

Left Lining of the Septum.—These contacts are the most precarious of any. Of three readings from the mid-septal region (−0·0036, −0·0085, 0·0066), two were low values, the third relatively high. Small contacts introduced through the aorta to test the region immediately beneath the aortic valves gave, in two animals, readings of −0·0168 and 0·0022 respectively. The former is the lowest reading we have obtained from any part of the heart. This is of interest, since from the distribution of the Purkinje substance this is the region where we should expect earliest activity. Directly below the valves the left division of the bundle appears (Plate 9, fig. 22), and the rebranching begins almost immediately.

But there are small areas close at hand which appear to have little or no supply. Very low values and considerably higher values would be anticipated in this neighbourhood.

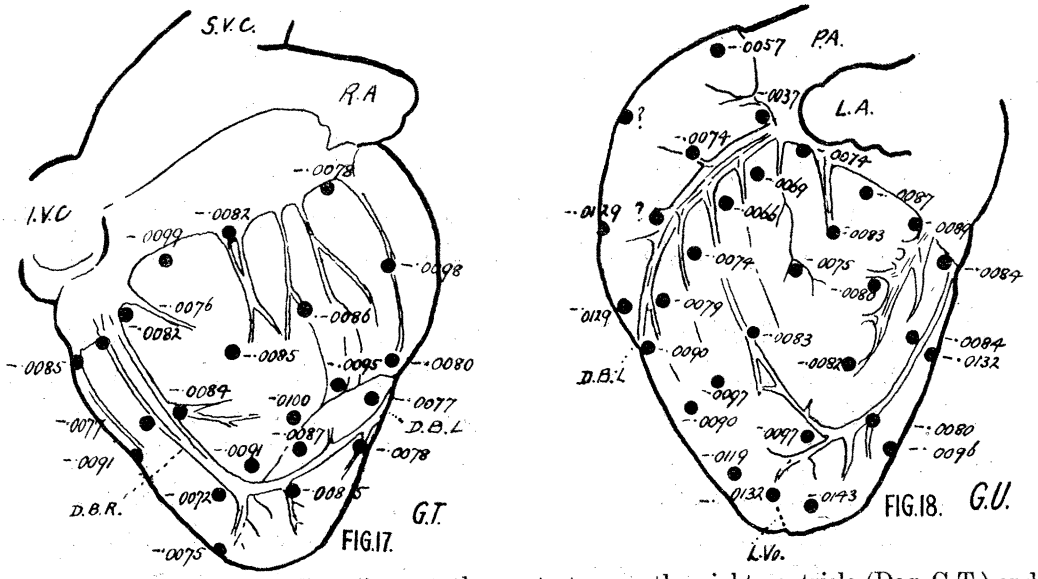
* It has since occurred to us that the exceptional high reading may be brought about by an electrode rubbing and damaging an area of the network surrounding the contact. In such a case the reading would be too high because conduction to the contact would be through muscle for some distance. The Purkinje substance is very susceptible to pressure, witness the effect of lightly clamping the *A–V* bundle.

The Endocardial Lining as a Whole.

Two facts of chief importance seem to be established. (1) The lining of the ventricular wall is always activated before the pericardial surface, and our results show that the interval between the internal and external activity is controlled by the thickness of muscle at the corresponding point. A simple muscular spread of the excitation wave from within outwards, with uninterrupted penetration of the several muscle layers accounts for the phenomena observed. (2) The internal surface is supplied at a very early period of the ventricular cycle, and at a number of points activity appears almost simultaneously. The earliest region to be activated, of which we can speak positively from direct observation, is the area supplied by the early outgoing branches of the right bundle division; other regions, as will presently be understood, are even earlier, and it is suspected, both from anatomical studies and from solitary direct leads, that the lining of the left side of the septum is in reality the first to be involved in the ventricle as a whole. The conus is a region in which we can speak definitely of a late arrival of the excitation wave.

The Earliest Appearance of the Excitation Wave.

The earliest sign of the ventricular excitation wave is not to be found in R in axial leads. This is preceded in the majority of electro-cardiograms by a small depression Q, which is unquestionably ventricular in origin. The reading for Q in respect of R is, as a rule, between $-0{\cdot}0050$ and $-0{\cdot}0100$ second. But the earliest sign is to be found in extrinsic deflections. In figs. 6 and 9, a number of observations upon intrinsic deflections are charted. The earliest extrinsic deflections, over the same contact points are charted in figs. 17 and 18, and the extrinsic deflections corresponding to other figures are tabulated (Tables XVII and XVIII).



FIGS. 17 and 18.—The extrinsic deflections at the contacts over the right ventricle (Dog G.T.) and over the left ventricle (G.U.). The observations are from the same hearts as those shown in figs. 6 and 9.

TABLE XVII.—Dog G.L.

Contact.		Extrinsic.	Intrinsic.
1		−0·0025	0·0151
2		−0·0046	0·0241
3		−0·0045	0·0261
4		−0·0061	0·0231
5		−0·0050	0·0198
6		−0·0059	0·0169
7		−0·0038	0·0150
8		−0·0076	0·0049
9	R.V.	−0·0078	0·0082
10	R.V.	−0·0059	0·0033
11		−0·0053	0·0167
12		−0·0066	0·0100
13		−0·0063	0·0021
14		−0·0028	0·0073
15		−0·0057	0·0178
16		−0·0069	0·0014
17		−0·0080	0·0054
18		−0·0053	0·0073
19		−0·0063	0·0195
20		−0·0042	0·0213
21		−0·0052	0·0203
22	L.V.	−0·0020	0·0146
23		−0·0013	0·0187
24		−0·0000	0·0196
25		−0·0044	0·0141

Q = −0·0096.

TABLE XVII.—Dog G.M.

Plate and contact.	Extrinsic.	Intrinsic.
1	−0·0077	0·0146
2	−0·0048	0·0212
3	−0·0054	0·0231
4	−0·0053	0·0266
5	−0·0039	absent
6	−0·0057	absent
7	−0·0052	0·0204
8	−0·0033	0·0222
9	−0·0040	0·0211
10	−0·0069	0·0160
11	−0·0070	0·0129
12	−0·0059	0·0187
13	−0·0069	0·0000
14	−0·0061	0·0027
15	−0·0036	0·0185
16	−0·0042	0·0075
17	−0·0069	0·0029
18	−0·0044	0·0183
19	−0·0051	0·0084
20	−0·0053	0·0133
21	−0·0035	0·0235
22	−0·0049	0·0083
23	−0·0043	0·0049
24	−0·0044	0·0165
25	−0·0053	0·0155
26	−0·0059	0·0157
27	−0·0063	0·0149
28	−0·0039	0·0188
29	−0·0058	0·0190

Q = −0·0048.

TABLE XVIII.—Dog G.W.

Plates and contact.		Extrinsic.	Intrinsic.
1		−0·0110	0·0058
2		−0·0087	0·0081
3		−0·0069	0·0140
4		−0·0093	0·0145
5		−0·0084	0·0256
6		−0·0082	0·0237
7		−0·0152	0·0241
8		−0·0149	0·0003
9		−0·0133	0·0050
10		−0·0114	0·0058
11		−0·0080	0·0226
12		−0·0102	0·0177
13		−0·0133	0·0038
14		−0·0113	−0·0029
15		−0·0125	−0·0048
16		−0·0135	−0·0013
17		−0·0149	0·0166
18		−0·0160	0·0139
19		−0·0158	0·0144
20	Internal contacts.	−0·0172	−0·0041
21	Internal contacts.	−0·0087	−0·0030
22	Internal contacts.	−0·0134	−0·0016
23	Internal contacts.	−0·0075	0·0144
24	Internal contacts.	−0·0150	0·0038
25	Internal contacts.	−0·0192	−0·0168

Q = −0·0107.

When we lead from a pair of contacts one of which lies on the heart and the other on the chest wall, the times at which extrinsic deflections appear are, with a few exceptions, wonderfully uniform. In fig. 17 (G.T.) the values range from −0·0072 to −0·0100; in fig. 18 (G.U.) the majority of the readings range from −0·0037 to −0·0119, though a few readings are lower. The average of readings usually approximates to the value for Q but there are always some extrinsic deflections which precede Q.

That the extrinsic deflection results from the excitation of some deep lying mass of muscle cannot be doubted; that most extrinsic deflections from a single heart represent the activation of the same region of that heart seems clear from the general uniformity of the readings. It is certain that the earliest extrinsic deflection from a given contact is not an expression of activity in the deeper layers of the muscle wall at the point investigated, for, if that were so, the values should rise over the right ventricle when the right branch division is transected. In point of fact they do not rise (Table XIX). It seems clear, therefore, that extrinsic deflections recorded from a contact over the

right ventricle may represent the passage of an excitation wave through portions of the left ventricle.

TABLE XIX.—Deflections before and after Section of Bundle Branch.

Before section.			After section.		
Plate.	Extrinsic.	Intrinsic.	Plate.	Extrinsic.	Intrinsic.
		Dog G.P. (see fig. 12).			
9 (L.V.)	−0·0071	0·0179	18	−0·0102	0·0154
10 (L.V.)	−0·0085	0·0217	19	−0·0083	0·0206
11 (T.A.)	−0·0098	0·0081	20	−0·0116	0·0294
12 (R.V.)	−0·0098	0·0154	21	−0·0093	0·0544
13 (R.V.)	−0·0085	0·0148	22	−0·0090	0·0590
14 (L.V.)	−0·0060	0·0127	23	−0·0122	0·0114
15 (L.V.)	−0·0081	0·0168	24	−0·0103	0·0146
16 (R.V.)	−0·0074	0·0149	25	−0·0112	0·0390
17 (R.V.)	−0·0086	0·0251	26	−0·0088	0·0624
		Dog G.R. (see fig. 13).			
6 (R.V.)	−0·0050	0·0157	16	−0·0069	0·0516
7 (R.V.)	−0·0058	0·0027	17	−0·0089	0·0439
8 (T.A.)	−0·0089	0·0148	18	−0·0083	0·0300
9 (L.V.)	−0·0048	0·0265	19	−0·0048	0·0229
10 (L.V.)	−0·0050	0·0283	20	−0·0054	0·0219
11 (L.V.)	−0·0083	0·0282	21	−0·0064	0·0241
12 (L.V.)	−0·0031	0·0285	22	−0·0016	0·0262
13 (T.A.)	−0·0056	0·0127	23	−0·0058	0·0243
14 (R.V.)	−0·0023	0·0182	24	−0·0044	0·0520
15 (R.V.)	−0·0050	0·0252	25	—	0·0675

The earliest sign of activity in the ventricle as a whole, as estimated from the values of extrinsic deflections, comes between 0·0070 and 0·0192 second before the upstroke of *R*. When we were studying the auricle we found that the value of the earliest extrinsic deflection practically coincided with the value of the earliest intrinsic deflection in this chamber as a whole. In the auricle this fact can be easily established because the excitation wave spreads from a single centre, whose position is favourable to observation.

In the ventricle our earliest extrinsic deflections clearly indicate that some region is activated as a rule 0·0100 or 0·0150 second before *R* appears (and 0·0050 second or more before *Q* is written). We had hoped to find intrinsic deflections of like value, but in this we have been disappointed. Sometimes our earliest intrinsic would approach to within 3, 4 or 6 thousandths of a second of the earliest extrinsic deflection; in other instances a hiatus of a hundredth of a second or a little more would be left (see Table XIV). That there is generally a hiatus is perhaps not surprising, seeing that many readings cannot be secured from the lining of the heart; our view that the excitation wave appears first on the left side is strongly supported by those

experiments in which a number of points on the right endocardial surface have been investigated. In none of these has that area been discovered which is responsible for the earliest extrinsic deflection. Were it in the right heart we believe it could hardly escape detection.

Actual and Calculated Readings.

Our hypothesis is that the time at which the excitation wave appears at any point on the surface of the ventricle is controlled by two chief factors; firstly, by the length of the Purkinje tract from its starting point to the network immediately beneath the point in question; and, secondly, by the thickness of the ventricular wall. In two experiments we have attempted to make these measurements. In measuring the length of the Purkinje tract we place a piece of cotton upon the inner surface of the ventricle, so that it overlies what we judge to be the shortest Purkinje path, from the division of the main bundle to that part of the endocardium which is nearest to our marked point on the epicardium. The cotton is subsequently measured in millimetres. In estimating the thickness of muscle we do not always measure vertically to the surface, for frequently an oblique path to the nearest endocardial depression is the shorter one. We measure the heart after hardening in the distended position with formalin; and calculate how long the excitation wave will take to travel along so many millimetres of Purkinje substance, on the basis of a rate of 5000 mm. per second; we use 500 mm. per second as a basis for the rate of propagation in the muscle. In this way we arrive at the total time for the appearance of the surface excitation wave. To express this calculated value in relation to R we must subtract the interval between the commencing distribution in the bundle branches and the appearance of R; our closest estimate of this interval is the time which elapses between the earliest extrinsic deflection and R.

Thus if the Purkinje measurement is 51 mm., we allow 0·0102 second; if the muscle thickness is 4 mm., we allow an additional 0·080 second = 0·0182 second. If the earliest extrinsic comes 0·0100 second before R, our final estimate stands at + 0·0082.

We publish our two experiments, with the corresponding calculated values, in Tables XX and XXI. In one instance the left ventricle, in the other the right ventricle, has been utilised. Considering the many possible sources of error in measurement, and remembering that here and there at infrequent intervals twigs of the Purkinje network penetrate the muscle for a few millimetres, the calculated values approximate to the actual values in a most satisfactory manner. Especially is this the case in the left ventricular Table. The maximal divergence in both Tables is less than a hundredth of a second; the average divergence is but a few thousandths of a second.

But for our argument we are content to point out that the actual and calculated *orders* of the points bear far more than a coincidental relation to each other; and that

by calculation alone we may obtain a large series of surface values which gives a very accurate general survey of the actual distribution of the excitation wave.

TABLE XX.—Actual and Calculated Intrinsic Deflections.
(Right Ventricle, Dog G.X.)

Muscle thickness in mm.	Purkinje pathway in mm.	Calculated intrinsic.	Actual intrinsic.	Actual extrinsic.	Order of contact.
1·0	45	−0·0028	−0·0033	−0·0073	12
1·75	50	−0·0003	−0·0018	−0·0111	11
2·0	65	0·0032	0·0030	−0·0094	10
2·5	67	0·0046	0·0090	−0·0066	14
3·0	75	0·0072	0·0166	−0·0102	9
3·0	56	0·0034	0·0009	−0·0100	6
3·0	73	0·0068	−0·0012	−0·0117	7
3·5	45	0·0022	0·0091	−0·0083	13
4·0	60	0·0062	0·0064	−0·0088	5
4·5	67	0·0086	0·0129	−0·0138	8
5·0	73	0·0108	0·0183	−0·0079	2
5·5	71	0·0114	0·0145	−0·0092	1
6·0	78	0·0138	0·0230	−0·0089	3

NOTE.—Q = −0·0082. Earliest intrinsic = −0·0138.

TABLE XXI.—Actual and Calculated Intrinsic Deflections.
(Left Ventricle, Dog G.V.)

Muscle thickness in mm.	Purkinje pathway in mm.	Calculated intrinsic.	Actual intrinsic.	Actual extrinsic.	Order of contact.
4·0	51	0·0082	0·0071	−0·0055	5
4·0	53	0·0086	0·0093	−0·0096	6
7·0	43	0·0126	0·0156	−0·0048	4
7·0	45	0·0130	0·0177	−0·0077	21
8·0	47	0·0154	0·0122	−0·0095	7
9·0	44	0·0168	0·0148	−0·0067	2
9·5	33	0·0156	0·0203	−0·0039	15
10·0	53	0·0206	0·0236	−0·0049	8
10·0	31	0·0162	0·0197	−0·0075	20
10·0	36	0·0172	0·0216	−0·0053	13
10·0	54	0·0208	0·0269	−0·0057	11
10·5	61	0·0232	0·0295	−0·0050	9
11·0	31	0·0182	0·0202	−0·0056	3
11·0	45	0·0210	0·0192	−0·0094	19
11·3	27	0·0180	0·0272	−0·0010	17
11·5	41	0·0212	0·0233	−0·0030	14
12·0	42	0·0224	0·0252	−0·0040	10
12·0	43	0·0226	0·0285	−0·0052	12
13·0	32	0·0224	0·0297	−0·0056	1

NOTE.—Q not measurable. Earliest intrinsic (internal) = −0·0100.

The Excitation Wave in the Whole Heart.

We are now in a position to summarise our observations upon the excitation wave in the heart as a whole.

That activity first reveals itself in the region of the sino-auricular node and in the neighbourhood of the head of that structure has been shown, we consider, beyond question. It spreads from this node in every direction, progressing to all the margins of the musculature; the propagation through the auricle may be likened to the spread of the fluid poured upon an almost flat surface. Distribution of the excitation wave throughout the auricular tissue as a whole is expedited by the central position at which it originates, by the convergence of the chief muscle band to this region and by a high rate of conduction in the auricular tissue.* Flowing through the auricle, the wave reaches the auriculo-ventricular node where it is delayed,† it spreads from this through the bundle and passes to the ventricle. In the muscle of the ventricle conduction is slowest, for its function of distribution is a minor one; rapidity of distribution is provided by the architecture of the special tissues. The main bundle splits into two branches, right and left, and then subdivides into a wide arborisation and communicates with a network which pervades almost the whole sub-endocardial space. This special system of fibres is endowed with the highest order of conducting power. The final spread through the muscle is by penetration of the whole thickness at innumerable points.

In both chambers of the heart a quick and rapid movement of the whole of the contractile tissue seems desirable. In the auricle, the smaller chamber, the plan is primitive, depending as it does upon the quality and arrangement of the contractile tissue itself; in the ventricle, the large and chief driving chamber, such a plan appears to be insufficient and so a system of distribution is specially provided, a system which has reached a high grade of development.

The system of distribution in the ventricle appears to us to possess a second virtue; it protects the tissues. If the contraction wave followed the separate muscle bands, these would contract separately and at different times. It is difficult to see how in such circumstances dissecting ruptures of the walls or laceration of the arterioles, which penetrate all layers at right angles, could be avoided.

It has been assumed tacitly in this report that conduction is through muscular and not nervous channels; this, the generally accepted view, is strongly supported by what appears to be a clear relation between conductive power and the constitution of the striated elements. Where glycogen is held most richly by the striated cells, conduction is fastest; where it is stored less richly, conduction is slower. There is a great delay in the region of the A-V node; the glycogen content ends abruptly as the bundle is traced into this node (Nagayo, *loc. cit.*). Similarly, conduction is fastest

* 'Phil. Trans.,' vol. 205, p. 375 (1914).

† Lewis, White and Meakins, 'Heart,' vol. 5, p. 289 (1913–14).

where the striated cells are largest. Following the tissue elements from auricle to ventricle, a great decrease in size is observed at the A-V node, a sudden increase to the largest type of cell in the bundle and network, and a decrease at the junction of network and ventricular muscles. The variations of velocity are disposed in corresponding fashion.

TABLE XXII.—A Table giving the Times and Values of the Initial Deflections of the Axial Electrocardiogram (Lead *II*) in those Animals from which Surface Readings are published in Illustrations.

Dog.	Fig.	Average heart rate.	*Q*.	*R*.	Summit of *R*.	Point of *S*.
G.K.	20	152	−0·0053 (0·5 mm.*)	0·0000	0·0155 and 0·0226 (11 and 16 mm.)	0·0423 (7·5 mm.)
G.L.	21	143	−0·0096	0·0000	0·0187 (12 mm.)	0·0424 (3 mm.)
G.M.	23	101	−0·0048	0·0000	0·0185 (23 mm.)	0·0376 (2 mm.)
G.T.	6	146	−0·0081 (1·0 mm.)	0·0000	0·0091 and 0·0269 (2 and 8 mm.)	0·0401 (3·5 mm.)
G.W.	7	106	−0·0107 (1·0 mm.)	0·0000	0·0259 (23 mm.)	None.
G.X.	8	152	−0·0082 (1·5 mm.)	0·0000	0·0228 (11 mm.)	0·0345 (1 mm.)
G.U.	9	161	−0·0105 (0·5 mm.)	0·0000	0·0221 (7 mm.)	None.
G.V.	10	176	None.	0·0000	0·0159 (6·5 mm.)	0·0407 (4 mm.)
G.Y.	11	195	−0·0066 (2·5 mm.)	0·0000	0·0193 (20 mm.)	None.

* 10 mm. = 1 millivolt. This table was added in proof stage (April 21, 1915).

EXPLANATION OF PLATE FIGURES.

Plate 8.

Fig. 19.—A projected drawing (twice natural size) of the interior of the right ventricle of a dog. The outer wall has been freed along the posterior interventricular groove and between the tricuspid and pulmonary valves and thrown downward. The ensheathed branches of the right division of the bundle are seen. The tracing is a drawing of the right branch of the bundle, the Purkinje arborisation and network, displayed by special carmine staining.

Fig. 20.—A projected drawing ($1\frac{2}{5}$ natural size) of the cavity of the right ventricle (Dog G.K.) showing the relations of the large papillary muscle and free arborisation to the trabeculated region and surface. The overlying tracings are projections of the superficial muscle fibres and of the surface of the heart with the contacts and readings of the experiment.

Fig. 21.—A similar projection ($1\frac{2}{5}$ natural size), with similar overlying tracings of the heart of Dog G.L.

Plate 9.

Fig. 22.—A projection (twice natural size) of the cavity of the left ventricle of a dog, opened between the two papillary muscles, and through the mitral valve. The tracing shows the left branch of the bundle, its arborisation and network. Prepared in similar fashion to fig. 19.

Plate 10.

Fig. 23.—A projection ($1\frac{2}{5}$ natural size) of the heart of Dog G.M., similar to that of fig. 20. The overlying tracings are projections of the cavity of the left ventricle, showing the relations of the papillary muscles to the surface, and of the surface of the heart with the contacts and readings of the experiment.

Plate 11.

Fig. 24.—A photograph ($\frac{3}{7}$ natural size) of the interior of the right heart of an ox, in which the sheaths of the Purkinje network have been injected. *p.v.* = pulmonary valves ; *t.v.* = tricuspid valve segments ; *c.b.* = moderator band. To show the extent of the network.

Fig. 25.—Dog G.J. Two curves ($\frac{2}{3}$ natural size), each simultaneous with the

standard electrocardiogram, showing the effects of changing a distal contact. The leads are illustrated in fig. 4 in the text.

Fig. 25 A.—The upper curve is from two contacts (a–b) on the left ventricle.

Fig. 25 B.—The upper curve is from point a on the left ventricle to the left chest wall (c). The form of curve in changing the distal contact from b to c alters considerably. The relation of the first extrinsic deflection to R ($-0{\cdot}0066$ and $-0{\cdot}0100$) varies; the relation of the intrinsic deflection to R ($0{\cdot}0188$ and $0{\cdot}0190$) remains unchanged. *Ex.* = extrinsic; *In.* = intrinsic deflection.

Ordinates (upper curves), 5 scale divisions = 8·4 millivolts.

Abscissæ = 0·2 second.

Fig. 26.—Dog G.P. Four figures ($\frac{2}{3}$ natural size) showing the effect of cutting the chief branch of the right division of the bundle.

Figs. 26 A and 26 C are simultaneous records from a point on the right ventricle (upper curve in each) and from Lead *II* (lower curve in each). Figs. 26 B and 26 D are similar records from a point on the left ventricle Figs. A and B were taken before and figs. C and D after section of the branch of the bundle. The corresponding contacts may be found by consulting fig. 12.

Ordinates (upper curves), 5 scale divisions = 10 millivolts.

Abscissæ = 0·2 second.

Fig. 27.—Dog G.R. Four records ($\frac{2}{3}$ natural size) arranged in precisely the same fashion as those of fig. 26; and exemplifying a similar experiment. The corresponding contacts may be found by consulting fig. 13.

Ordinates (upper curves), 5 scale divisions = 10 millivolts.

Abscissæ = 0·2 second.

Plate 12.

Fig. 28.—Dog G.K. Two simultaneous curves (natural size) (1) from a conus to chest wall lead, and (2) from a point lower and more to the left on the conus.

As shown in the two cycles to the right of the figure, while the heart beat naturally, the excitation wave appeared almost simultaneously at the two conus points. While the heart responded to excitation at a point on the left ventricle in line with these contacts, the contact proximal to the point stimulated received the excitation wave 0·0119 second before the other (the distance of the contacts apart was 16 mm.).

Ordinates (both curves), 5 millimetres = 10 millivolts.

Abscissæ = 0·2 second.

Fig. 29.—Dog G.K. Similar curves from the same animal showing the slower conduction across the interventricular groove. (1) Curve taken from a lead from right ventricle to right chest wall. (2) Curve from a lead from left ventricle to left chest wall. The first three cycles were excited from the left border of the heart, at a point in line with the ventricular contacts; the latter lay 22 mm. apart, and the excitation wave took 0·0570 second to pass between them.

Ordinates (both curves), 5 millimetres = 10 millivolts.

Abscissæ = 0·2 second.

Fig. 30.—Dog G.O. Simultaneous curves (1) leading from an internal point near the base of the right ventricle to the right chest wall, (2) leading from an external point opposite the internal point to the left chest wall (see fig. 15).

While the heart beat naturally (last two cycles) the excitation wave at the internal point preceded that at the external point by an estimated time of 0·0205 second. While the heart beat in response to excitation at a point 15 mm. from the external contact, the excitation wave at the internal point preceded that at the external point by an estimated time of 0·0226 second.

Ordinates (both curves), 5 millimetres = 10 millivolts.

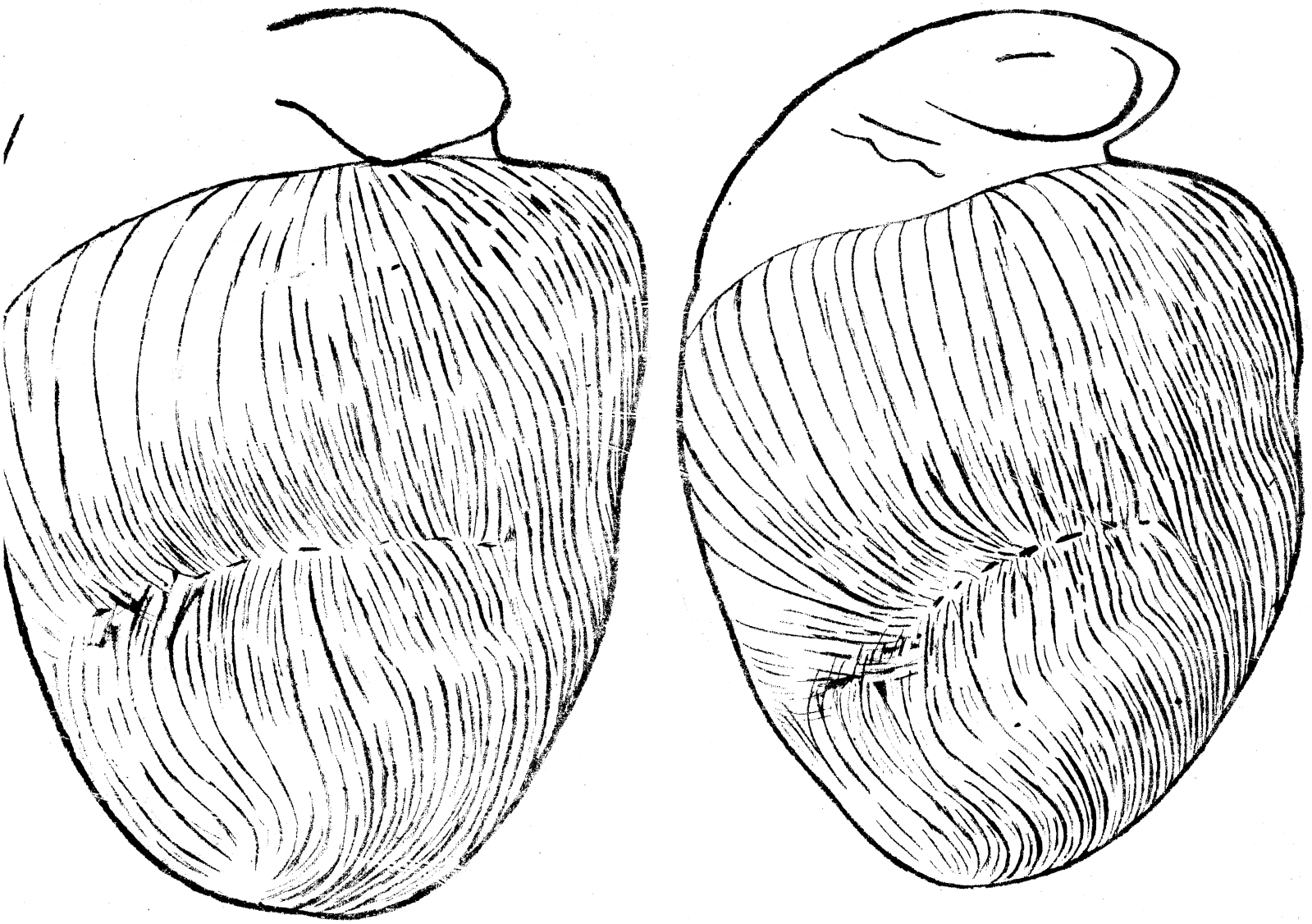
Abscissæ = 0·2 second.

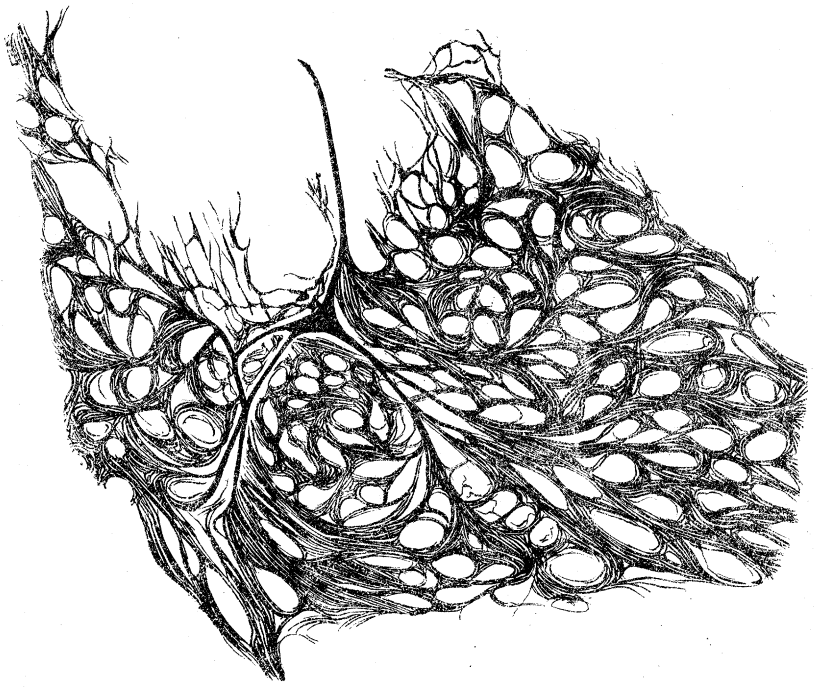
Fig. 31.—Dog G.O. An exactly similar experiment to that illustrated in fig. 30, with the exception that the point of stimulation lay only 6 mm. from the external point. The excitation wave at the internal point now precedes that at the external point by a lessened interval, namely, 0·0170 second, because the external point receives the excitation wave directly and not through the Purkinje system.

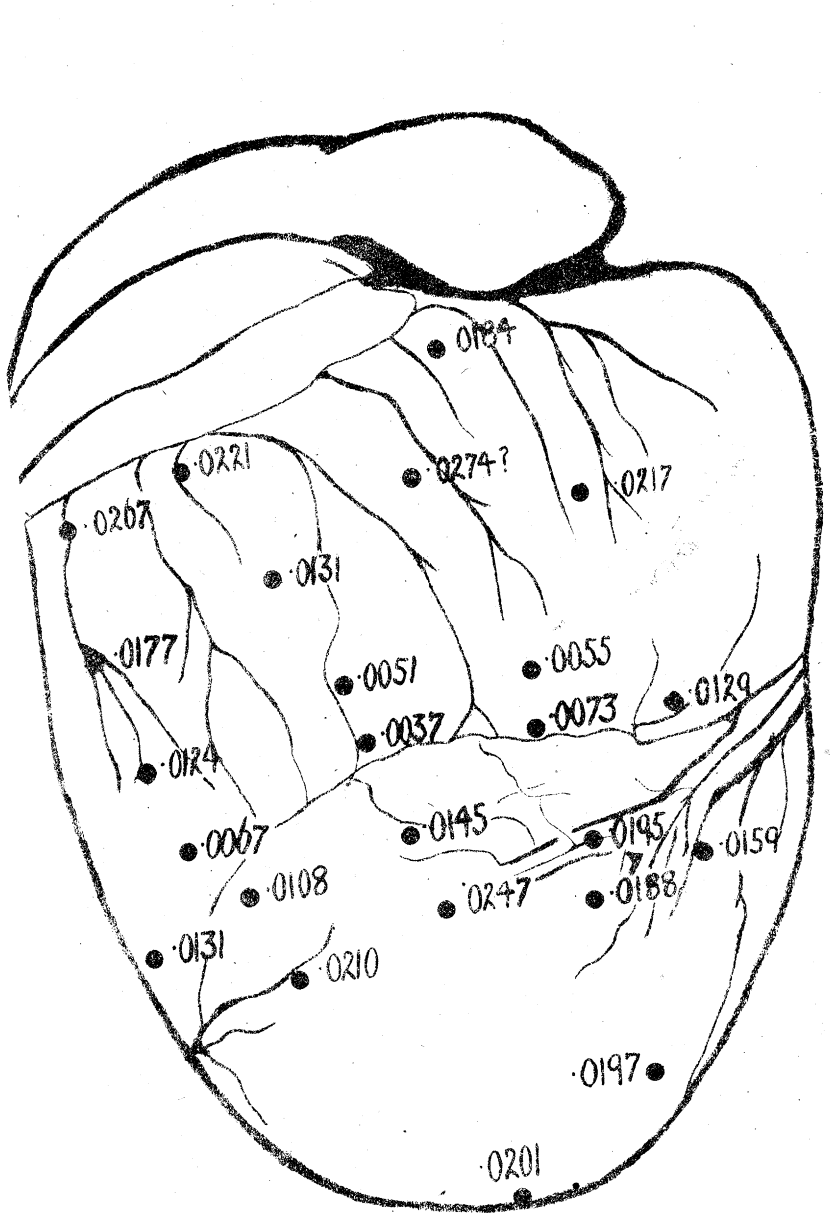
Ordinates (both curves), 5 millimetres = 10 millivolts.

Abscissæ = 0·2 second.

NOTE.—The ordinates for all standard curves (Lead *II*) are on the scale of 3 centimetres (30 scale divisions in reduced figures) = 3 millivolts.



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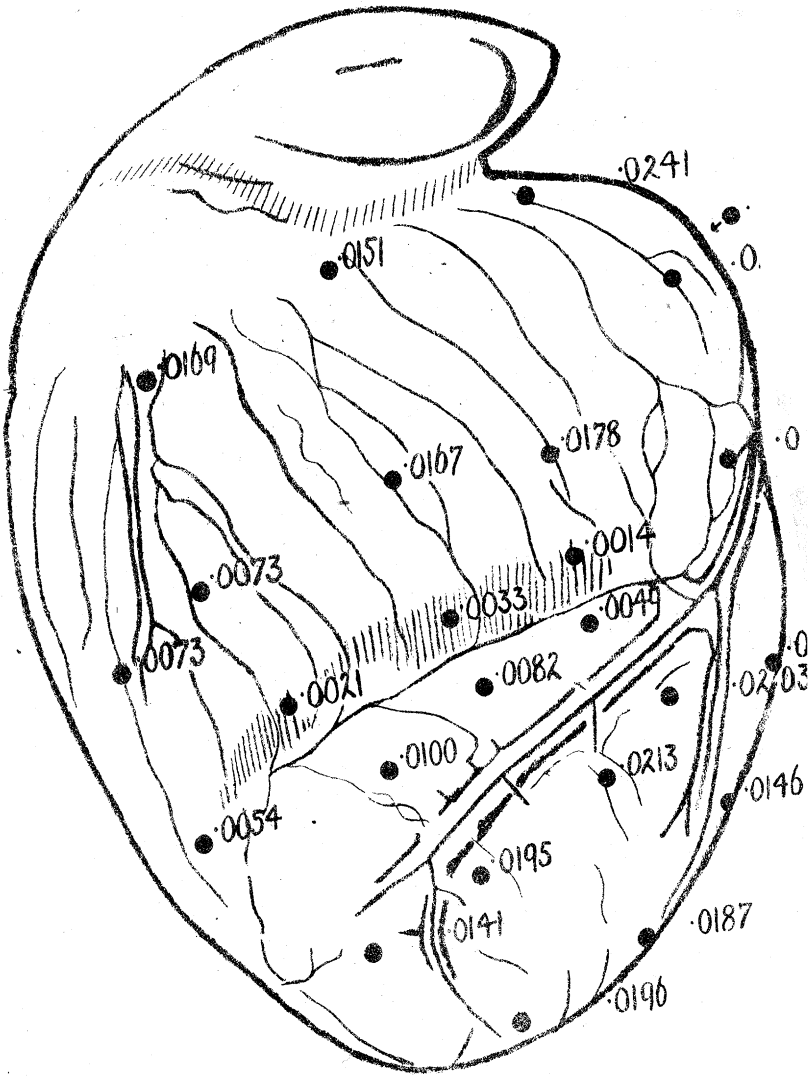
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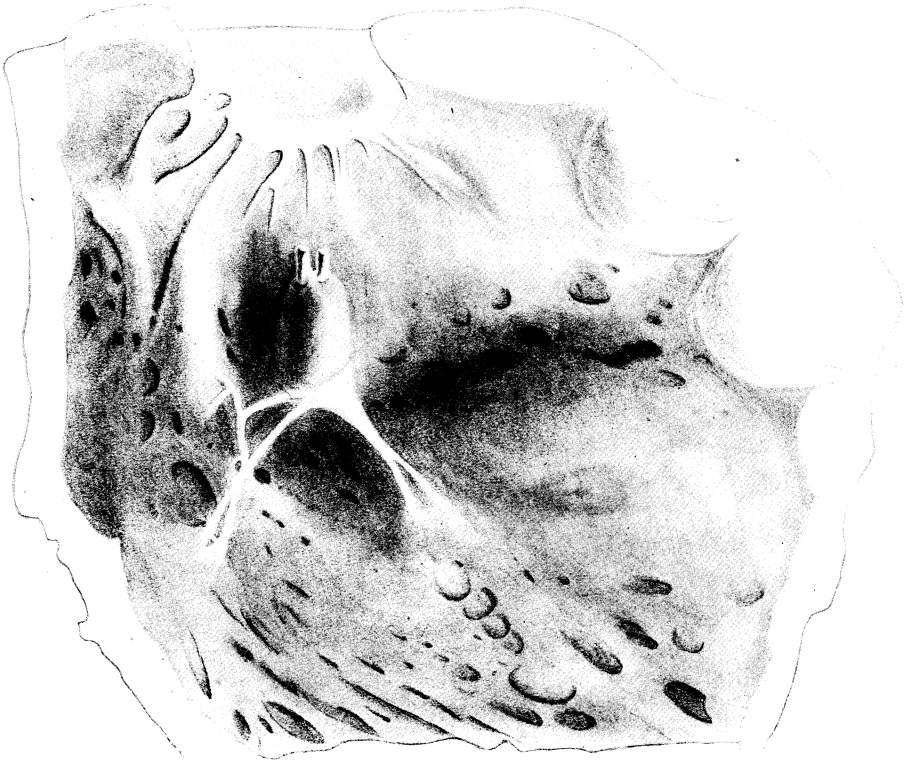

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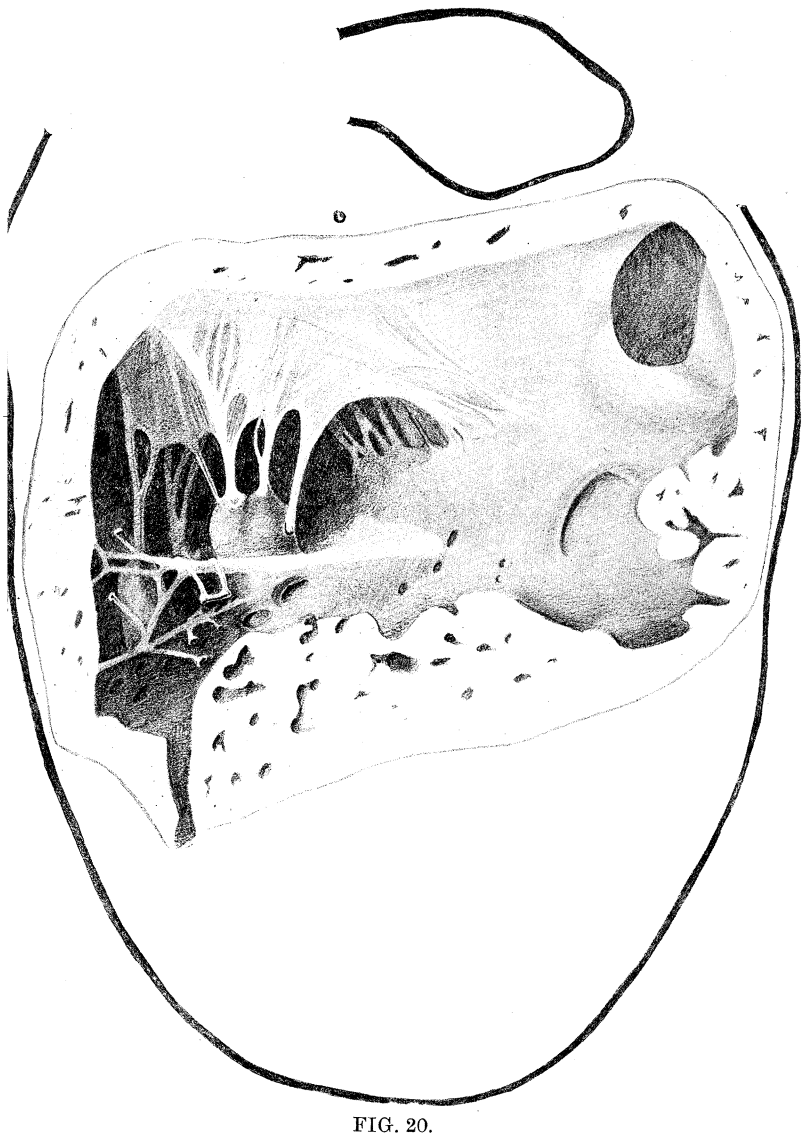

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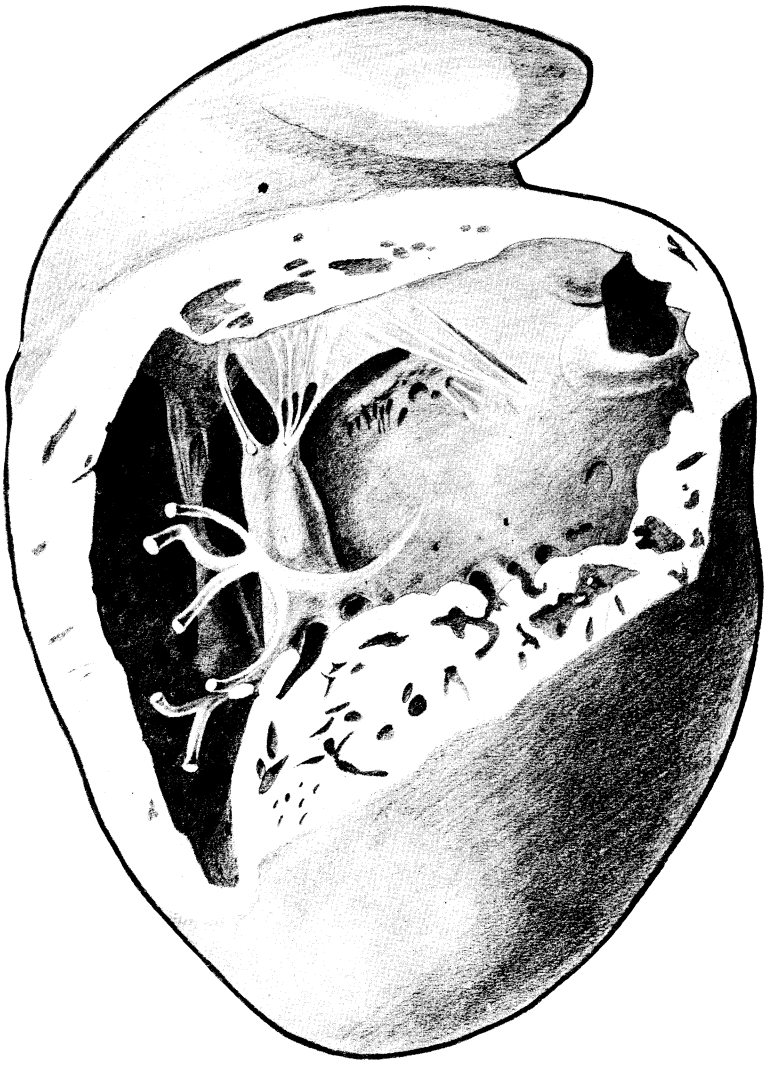

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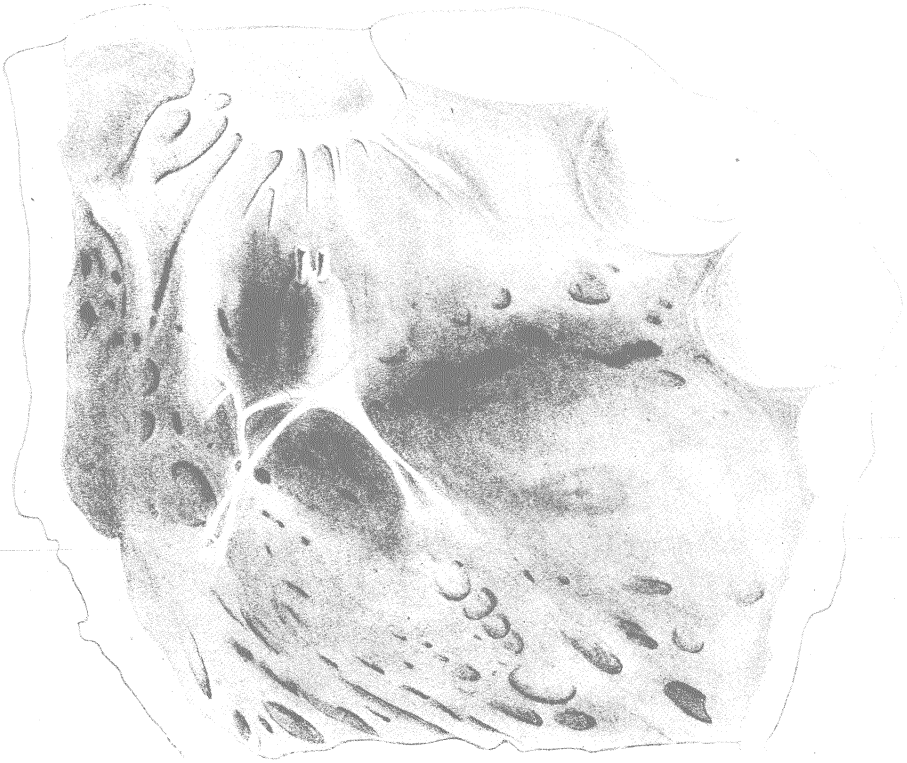

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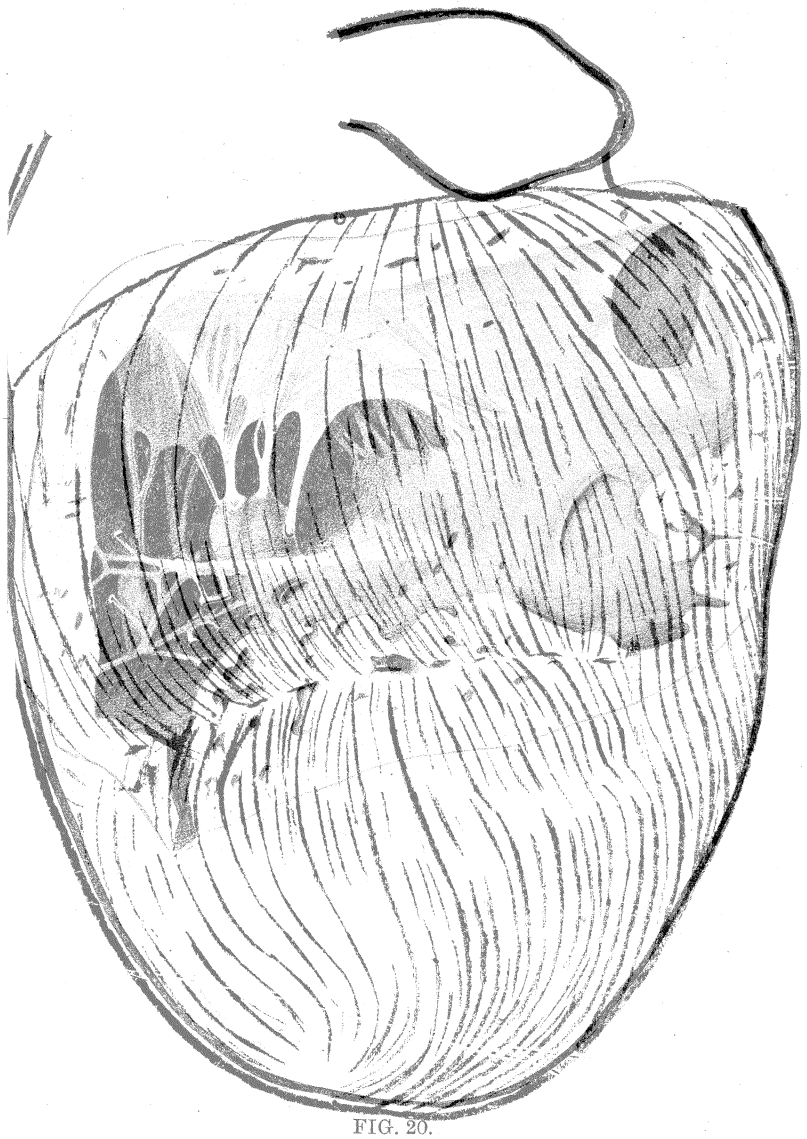

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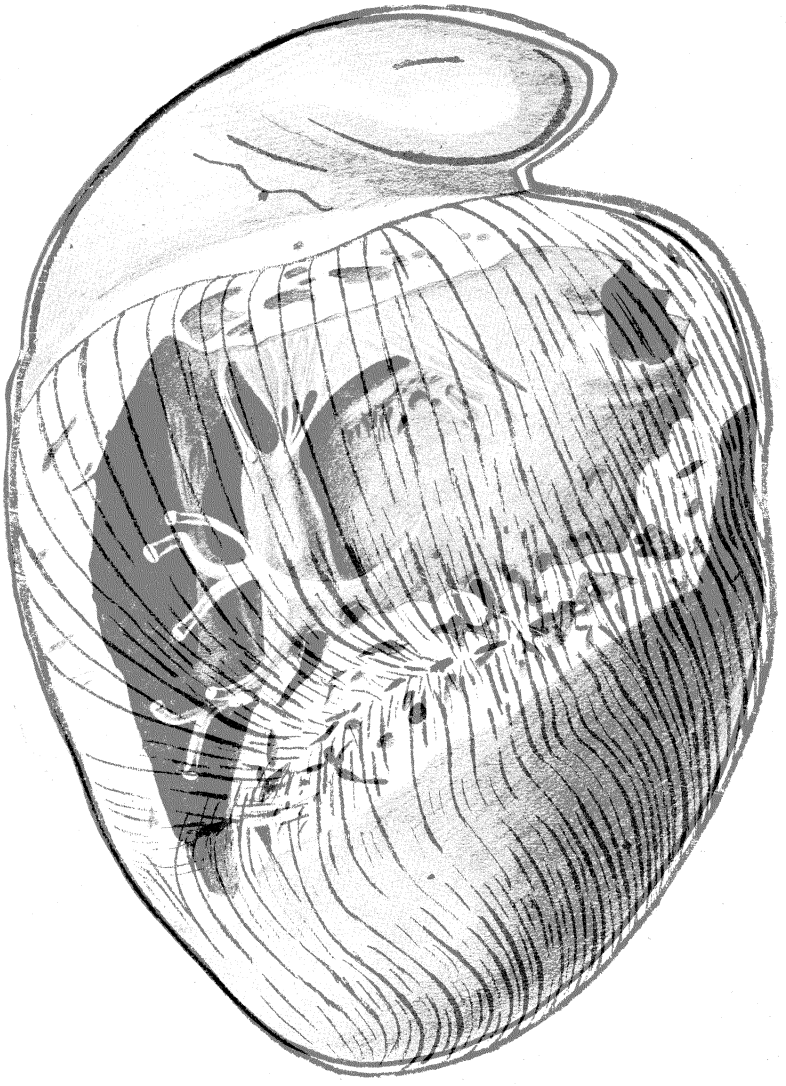

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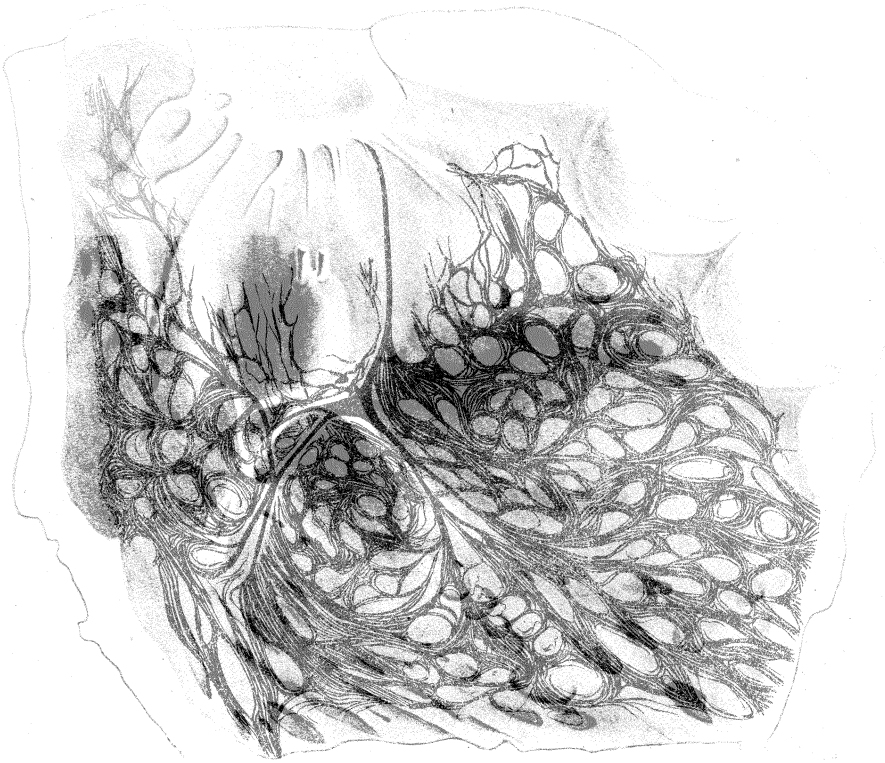

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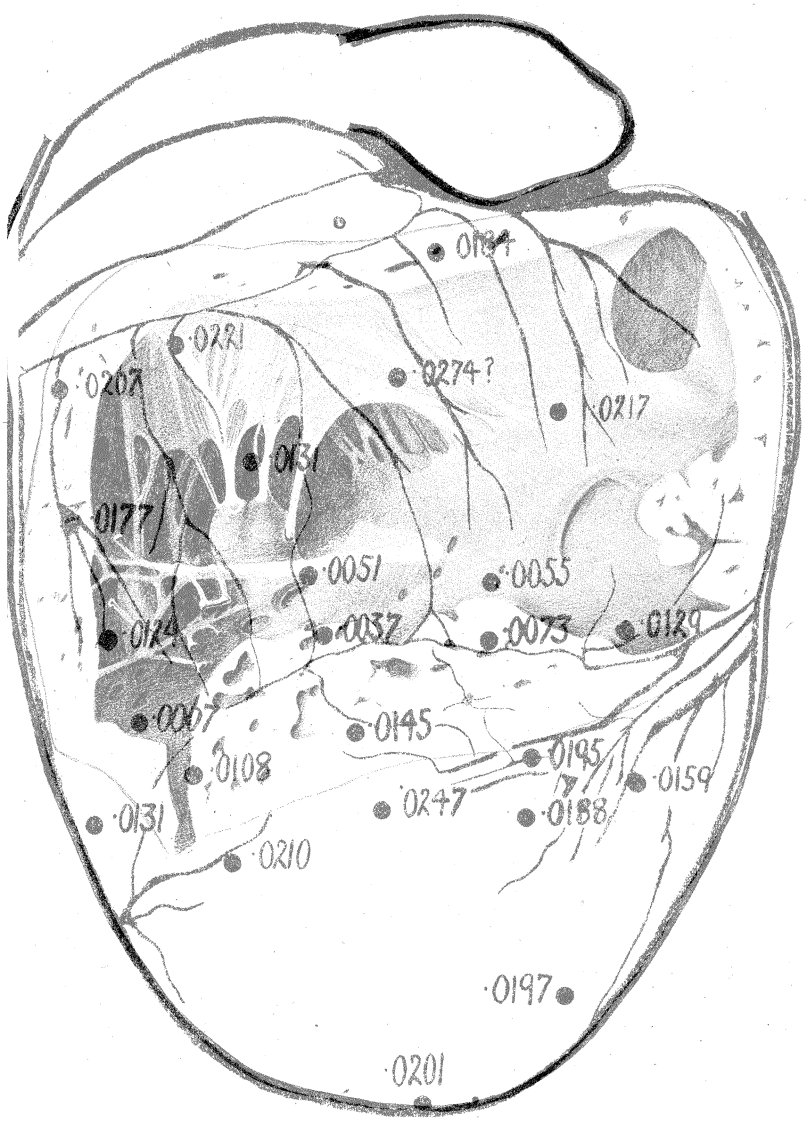

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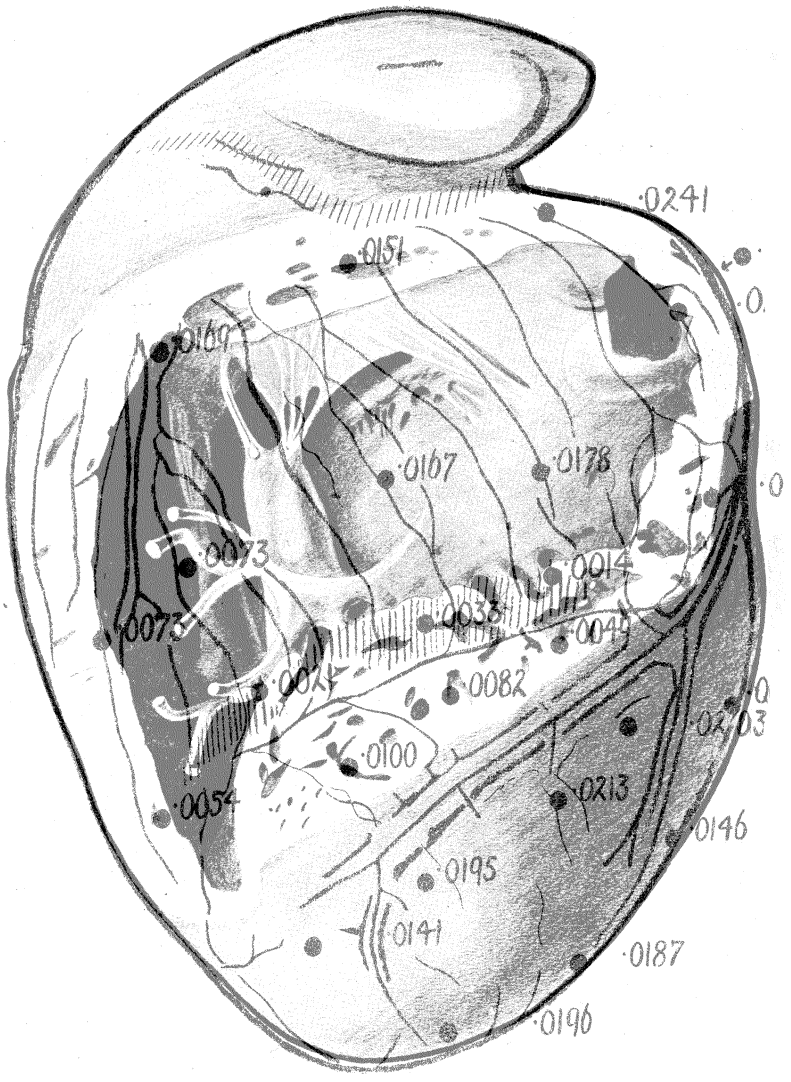

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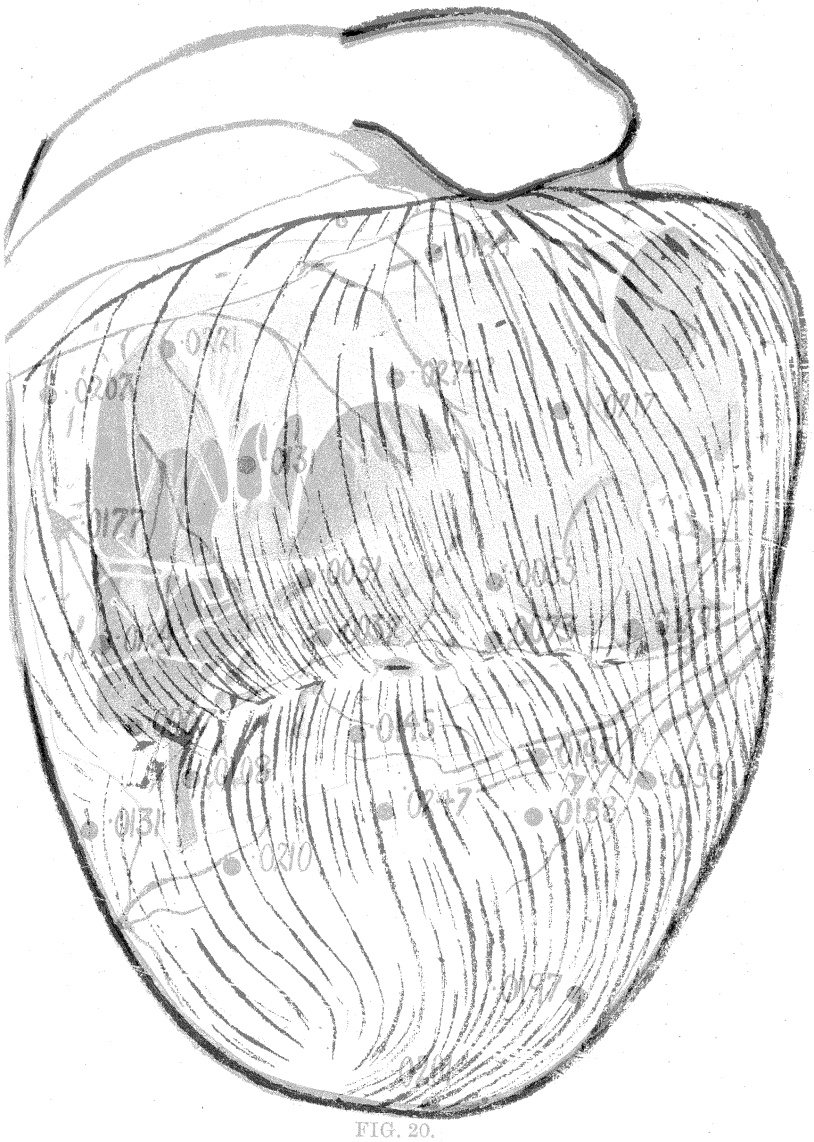

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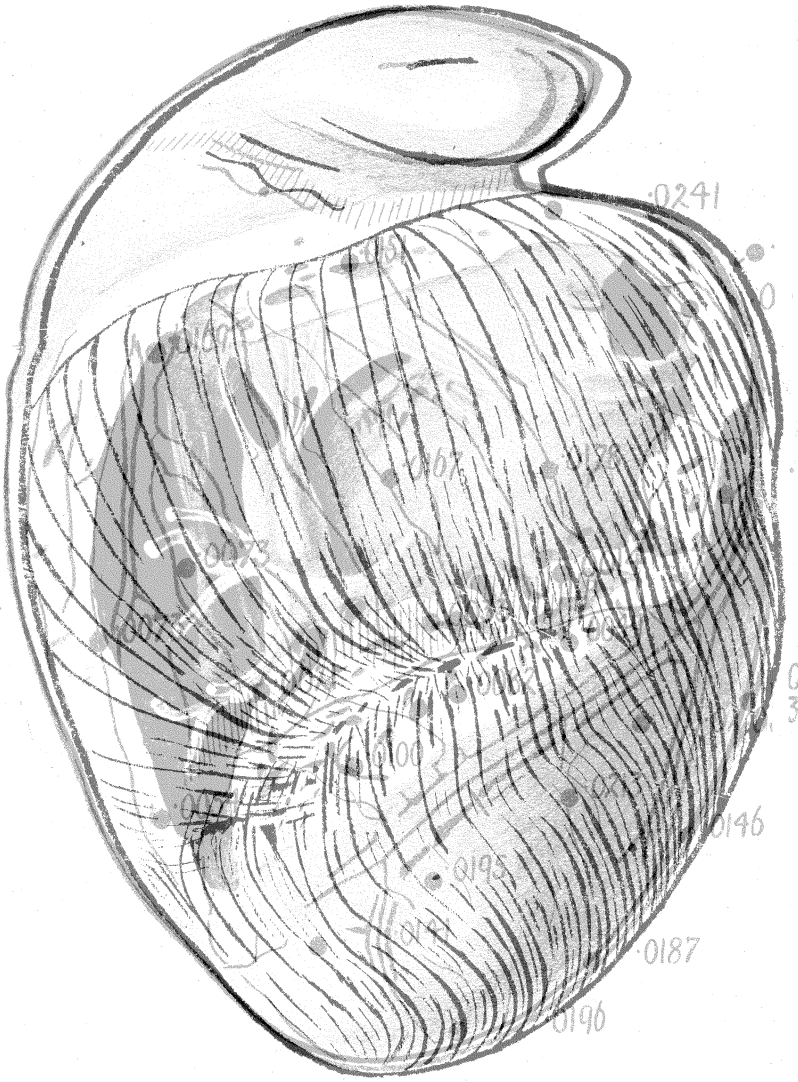

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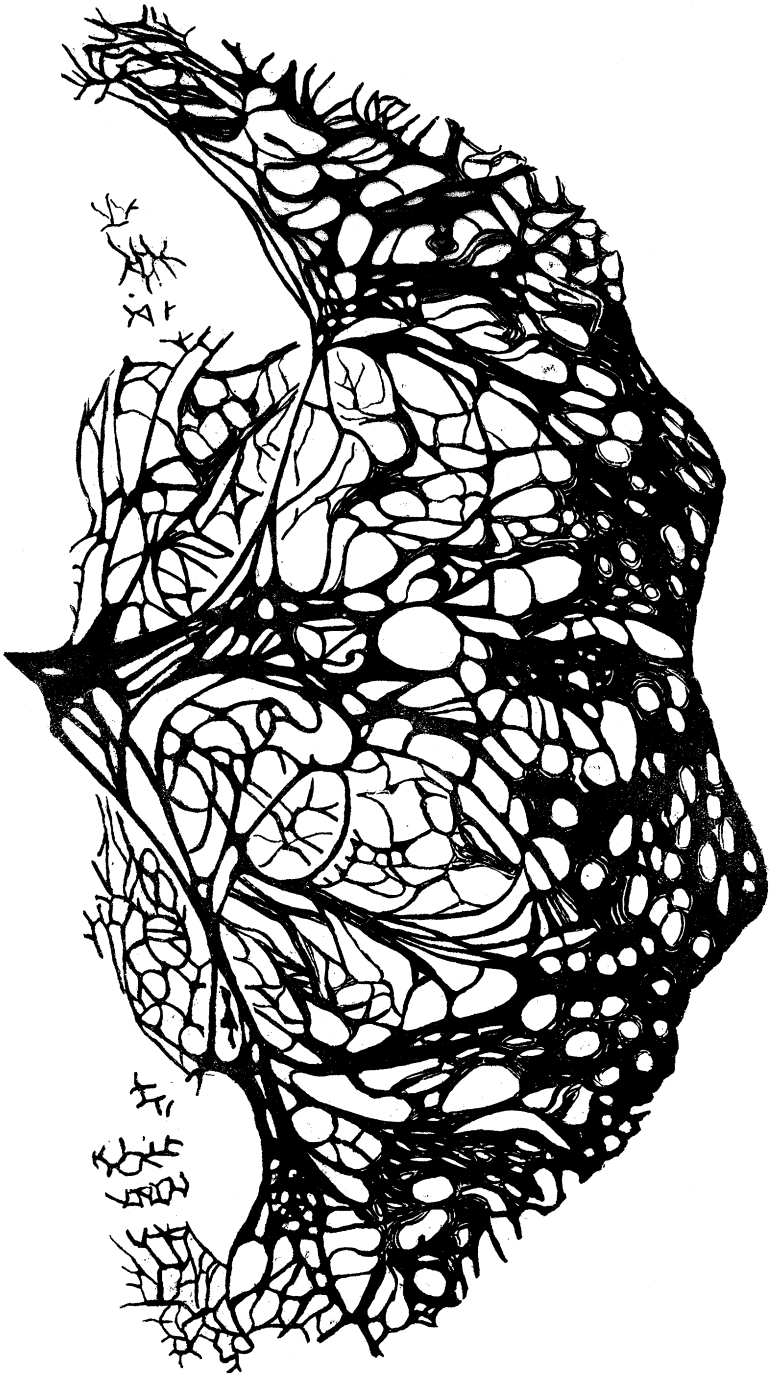

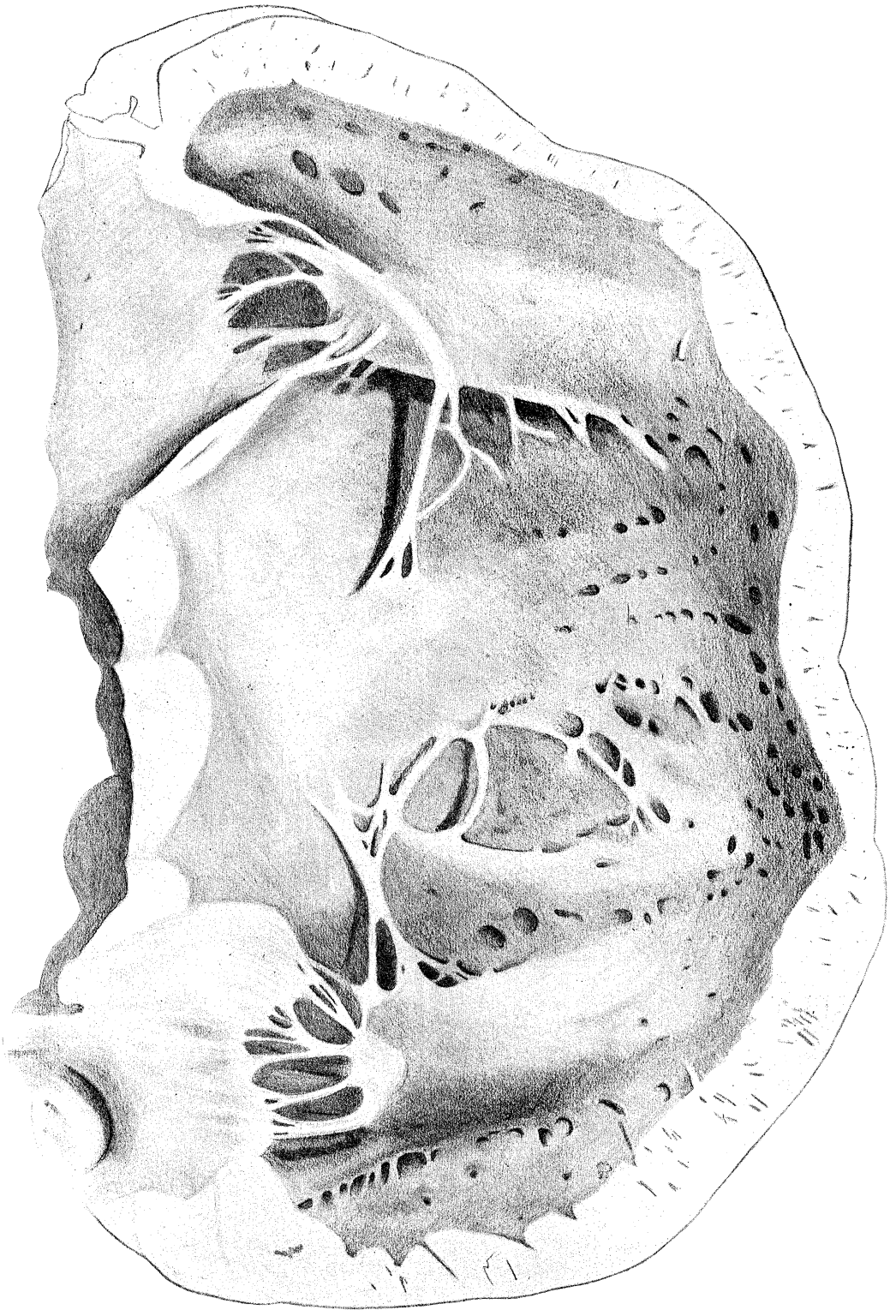

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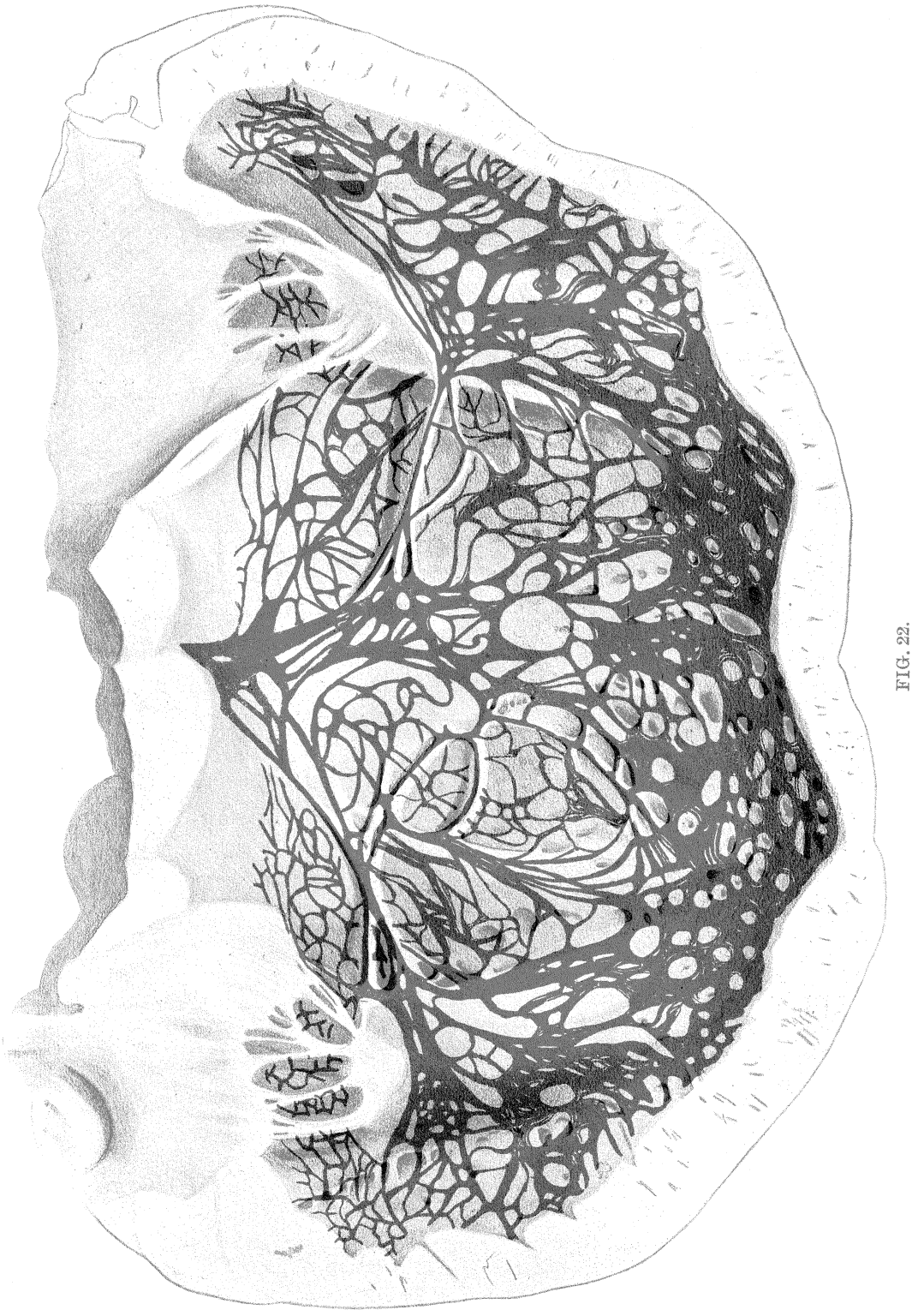

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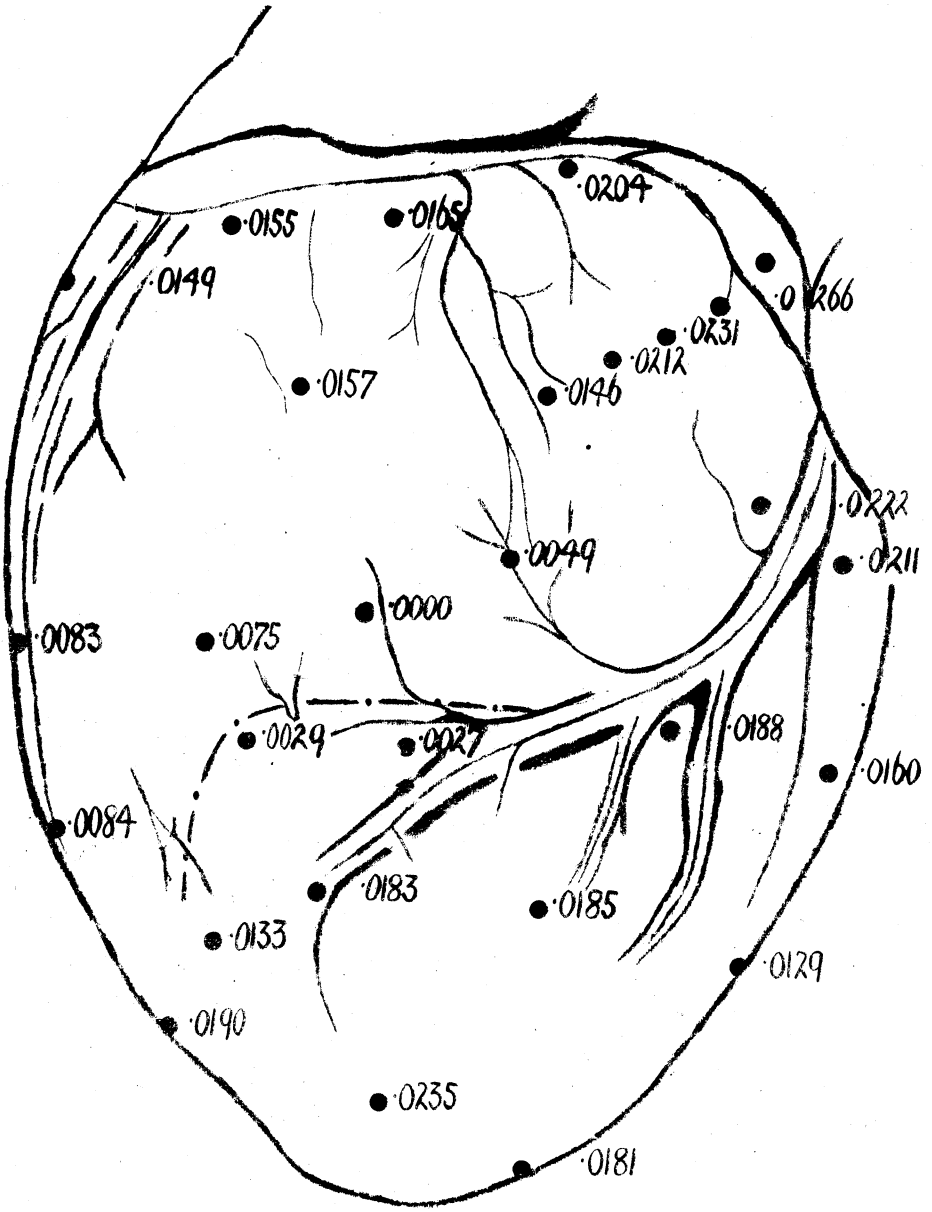
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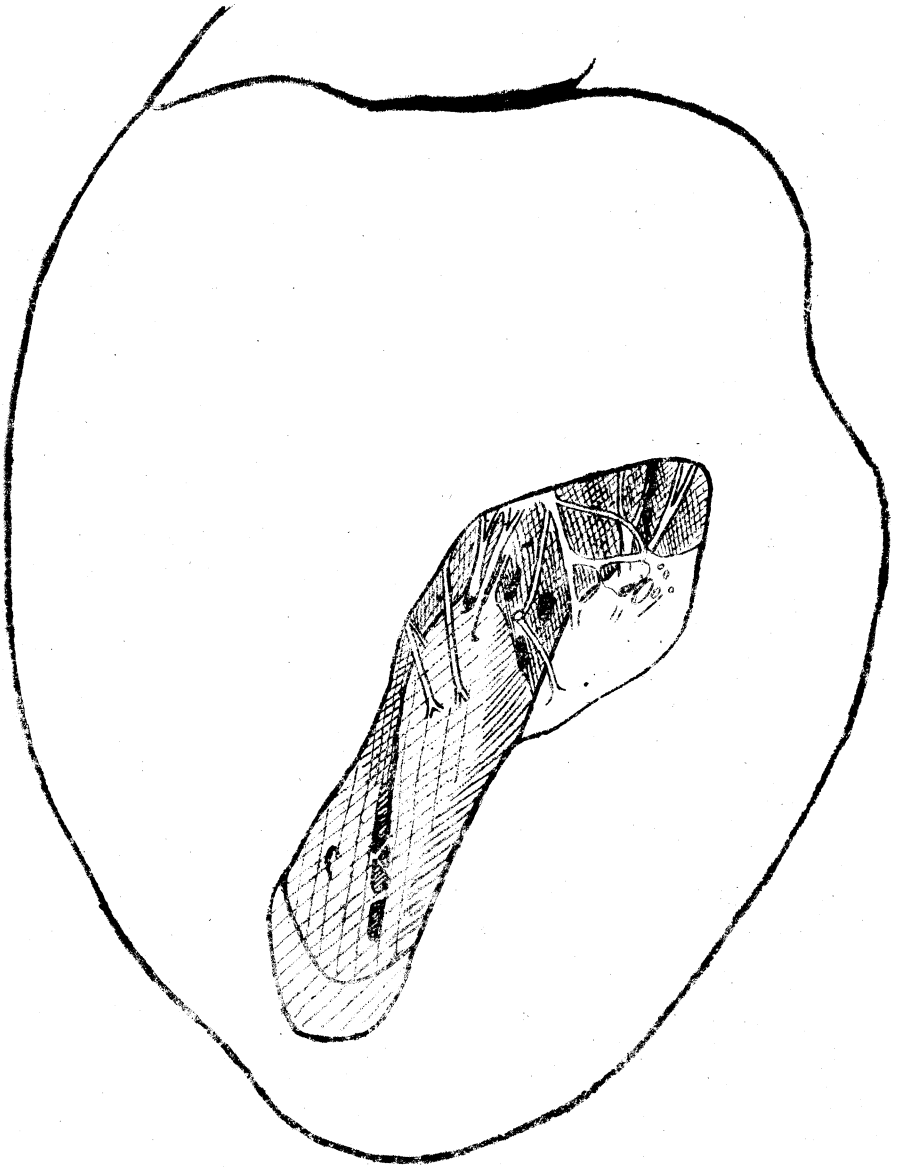

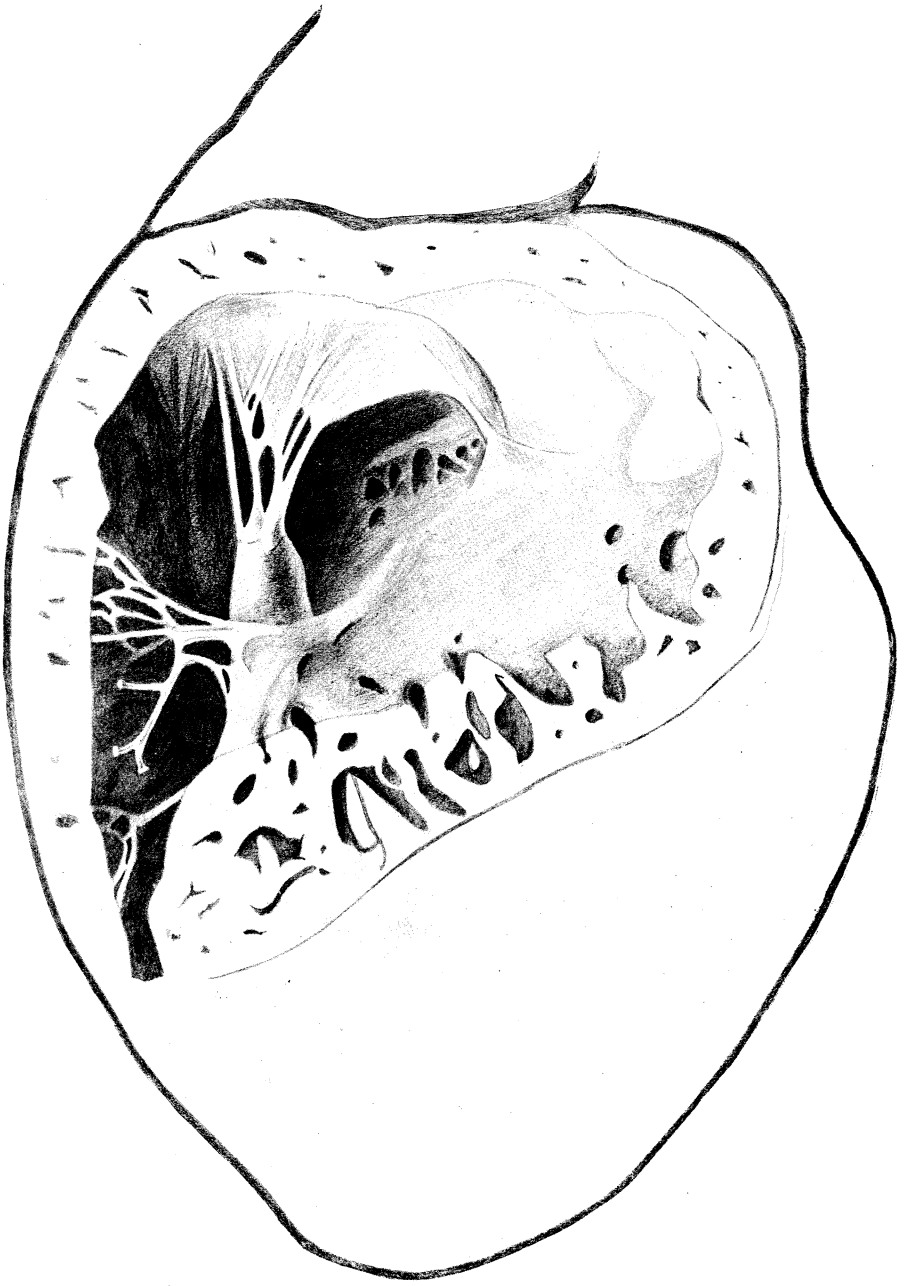

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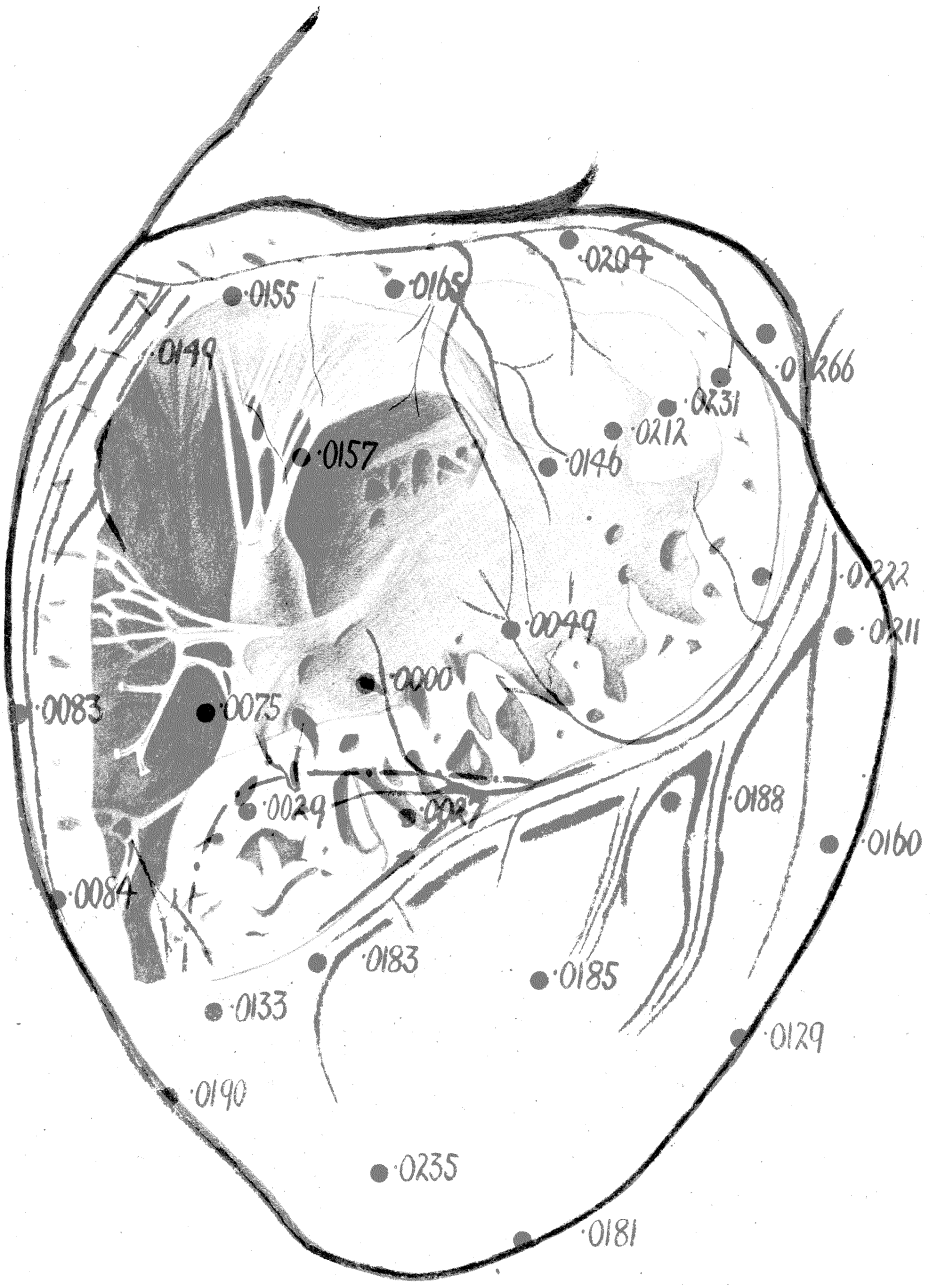
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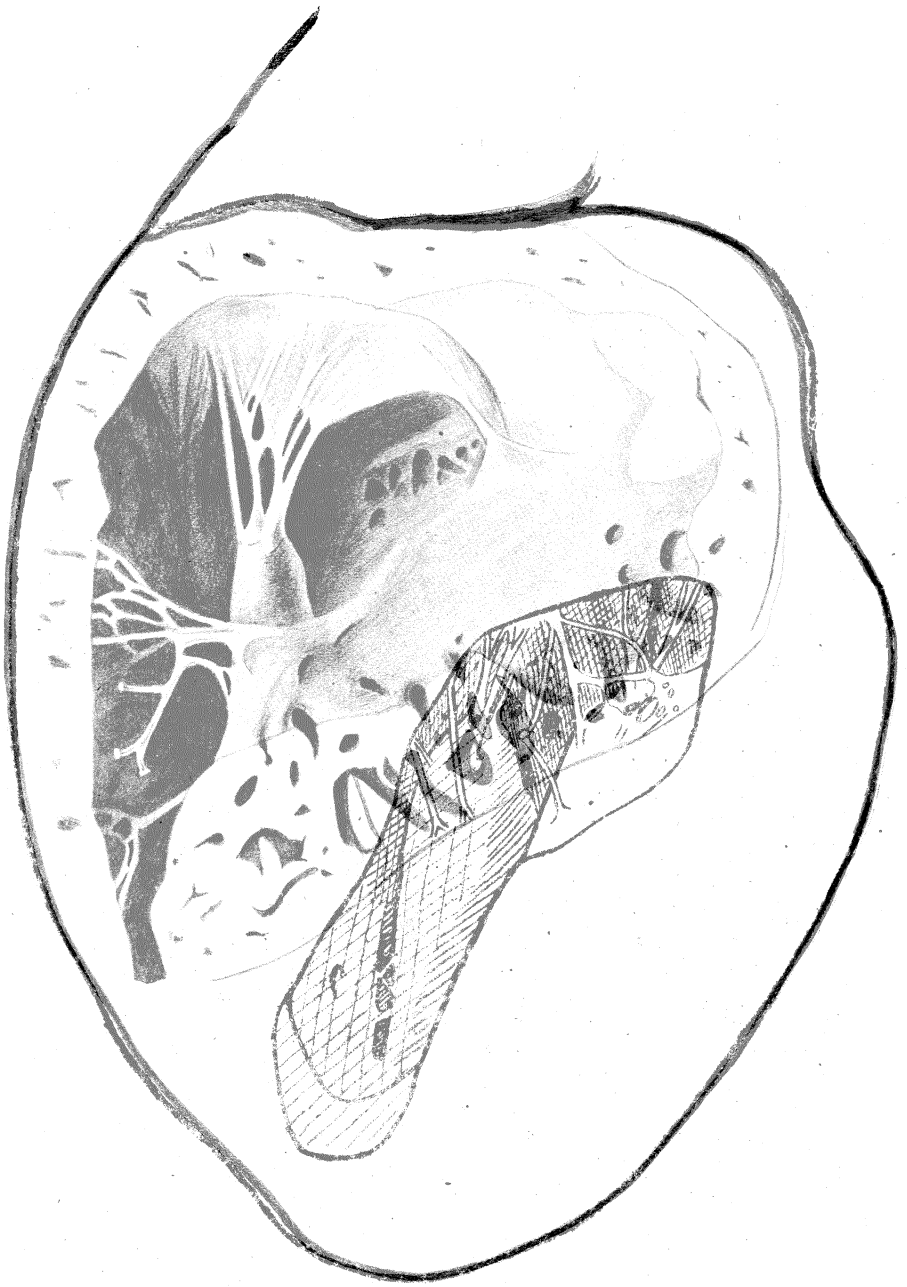

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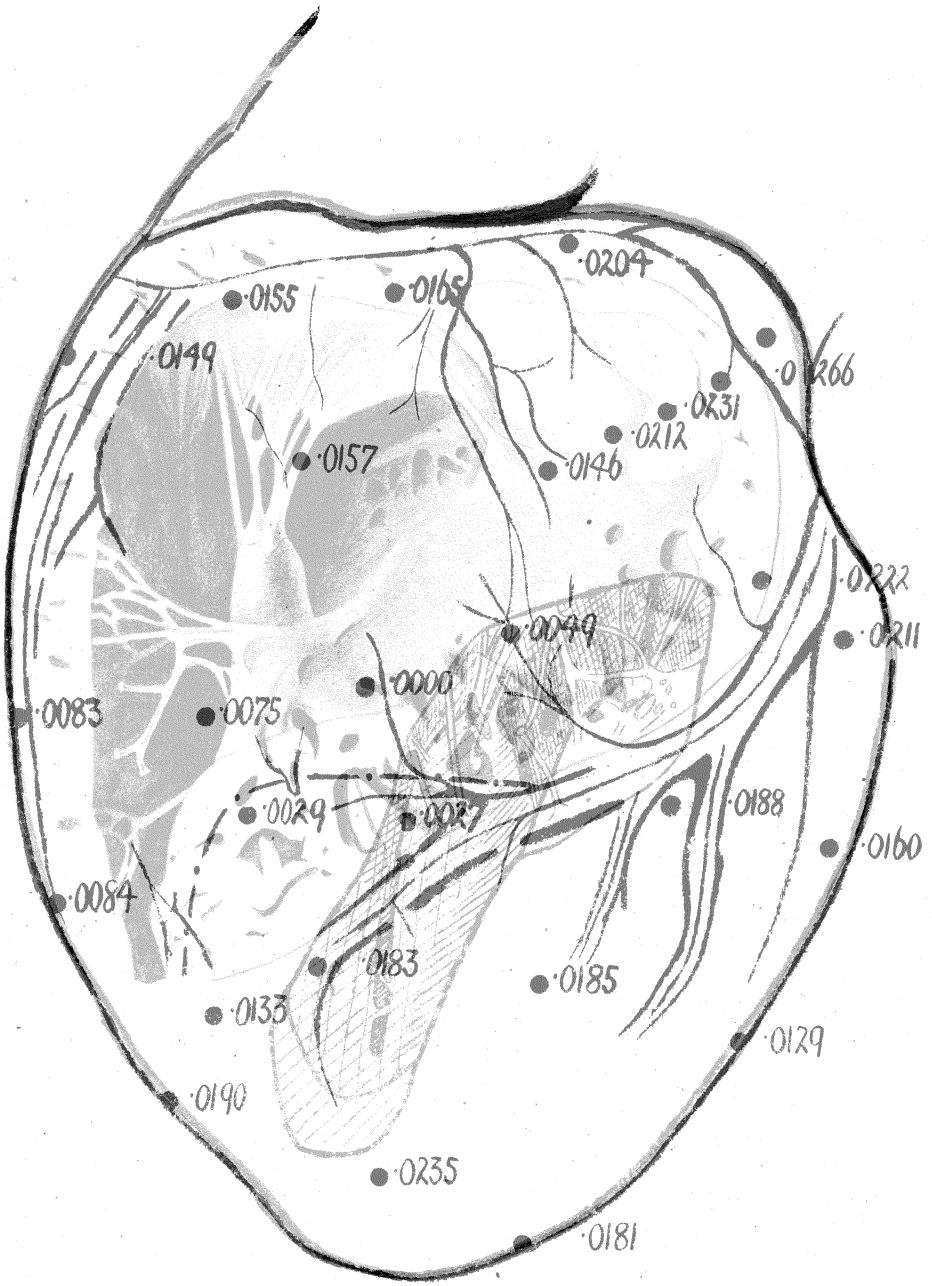
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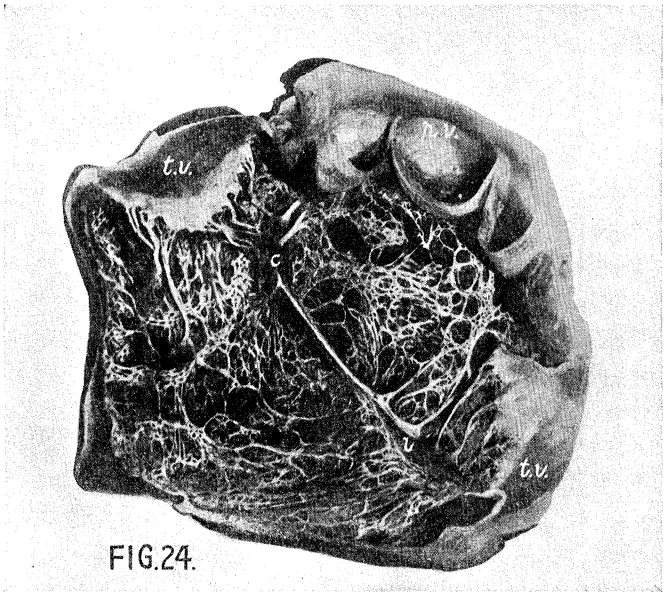
FIG. 23.



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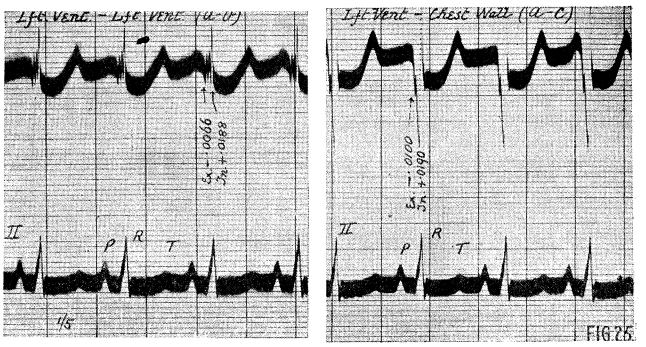
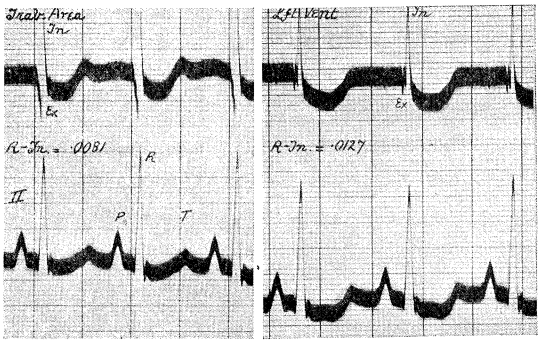
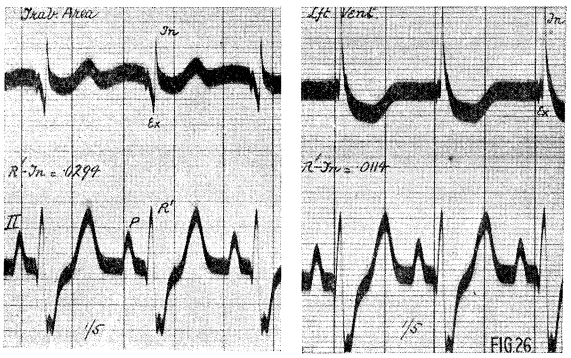


A. B.

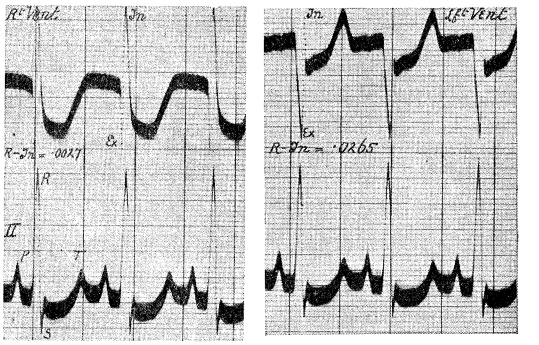
FIG. 25.

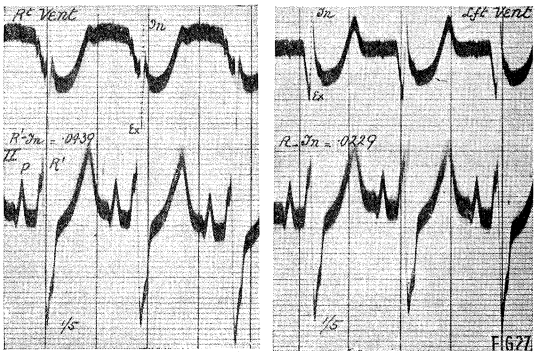




C. D.

FIG. 26.

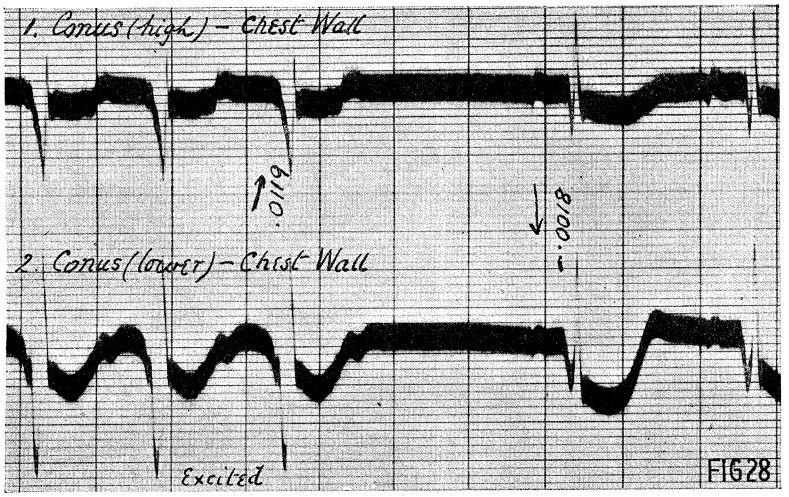




A. B.

C. D.

FIG. 27.



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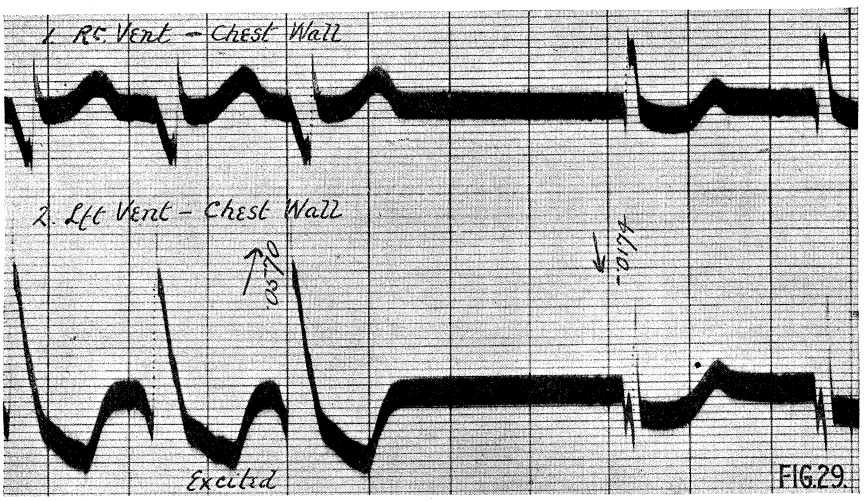

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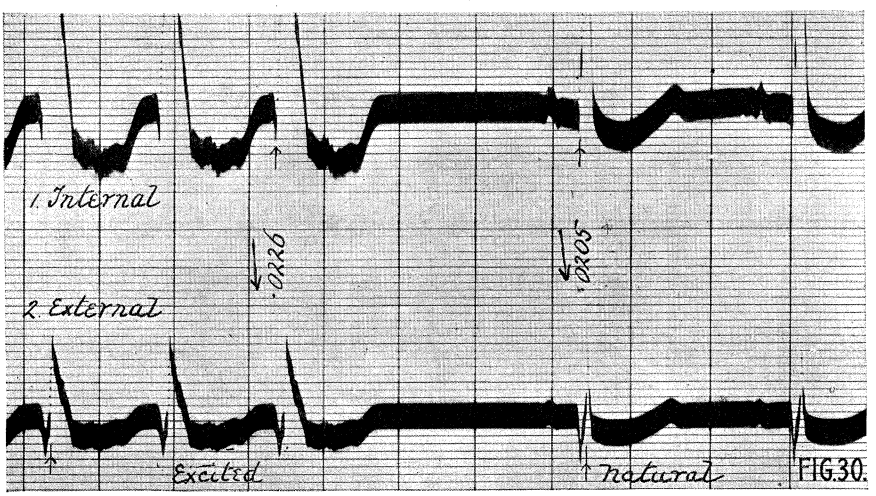

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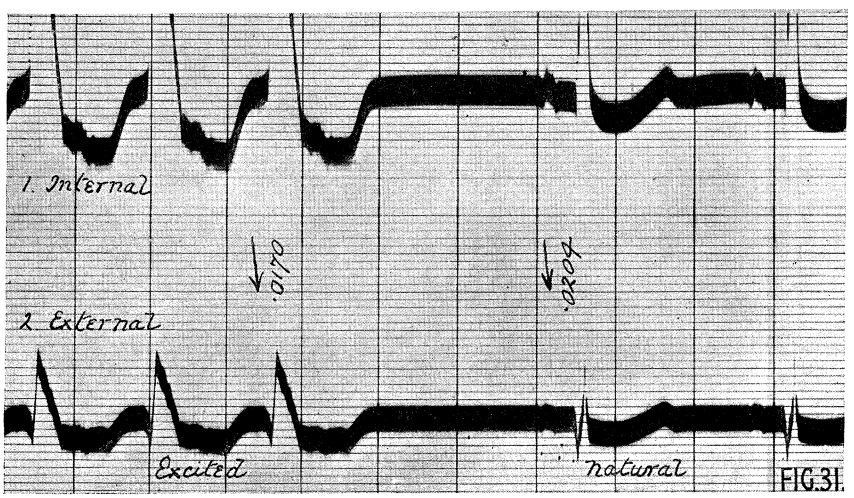

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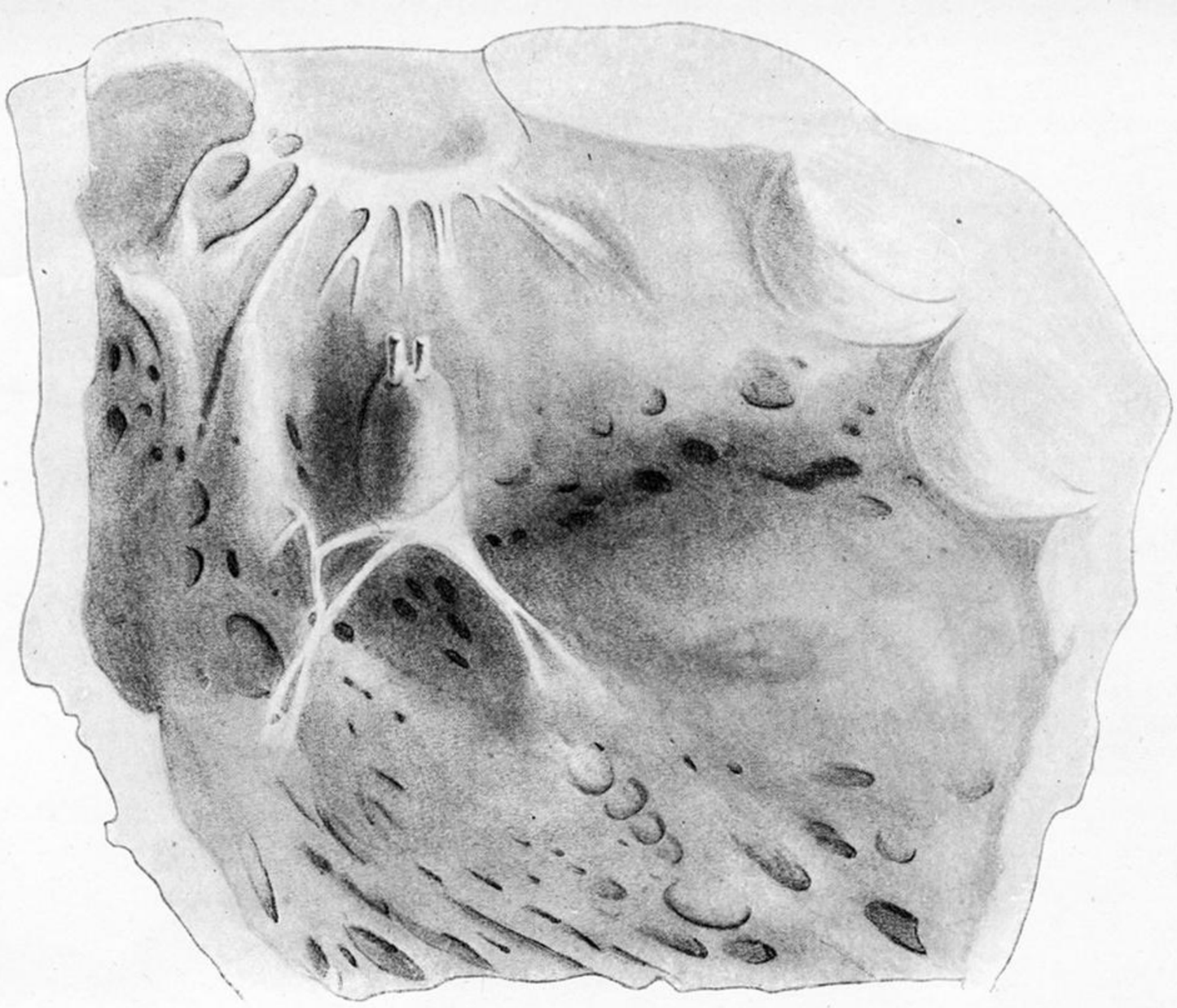

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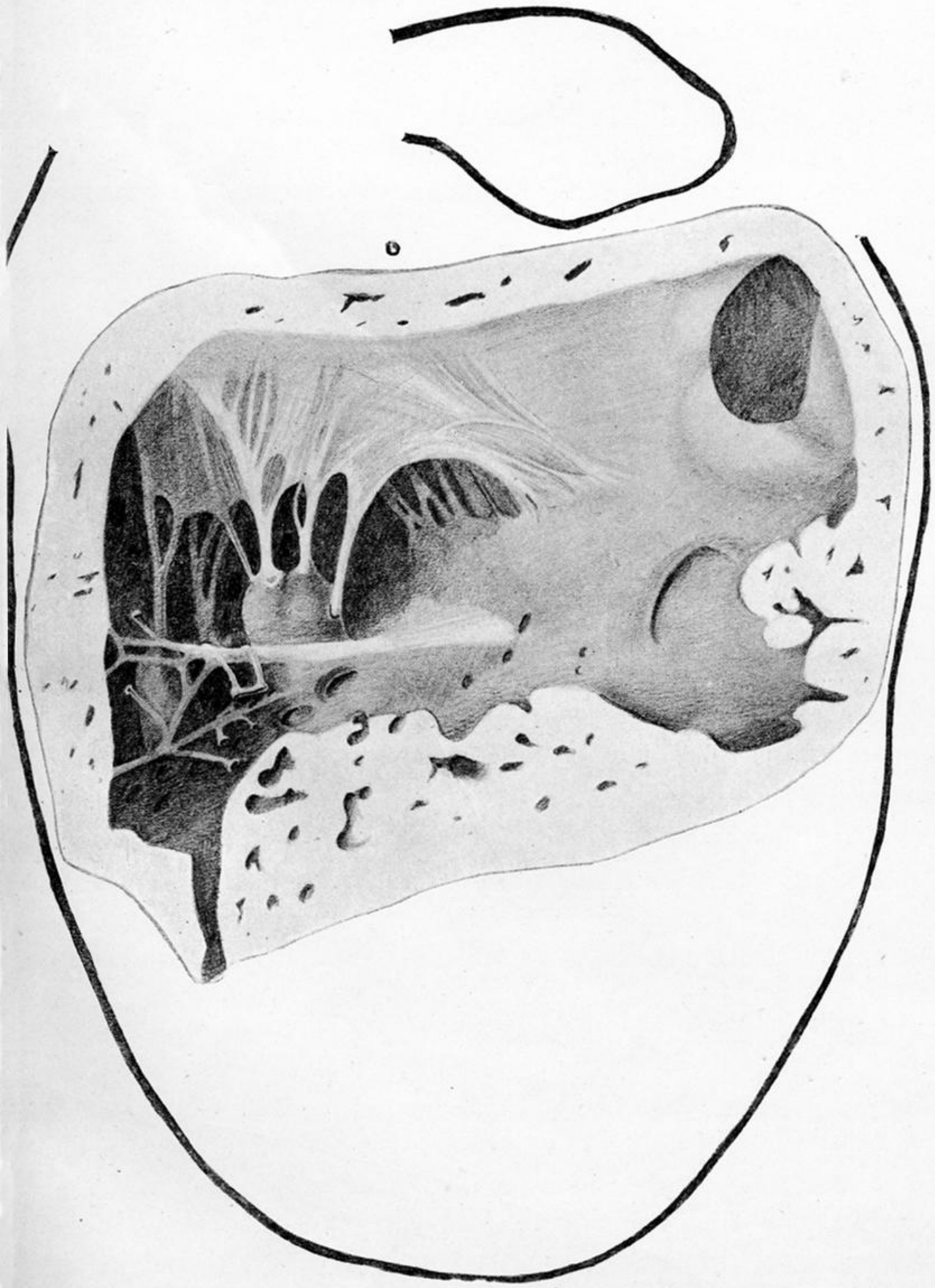

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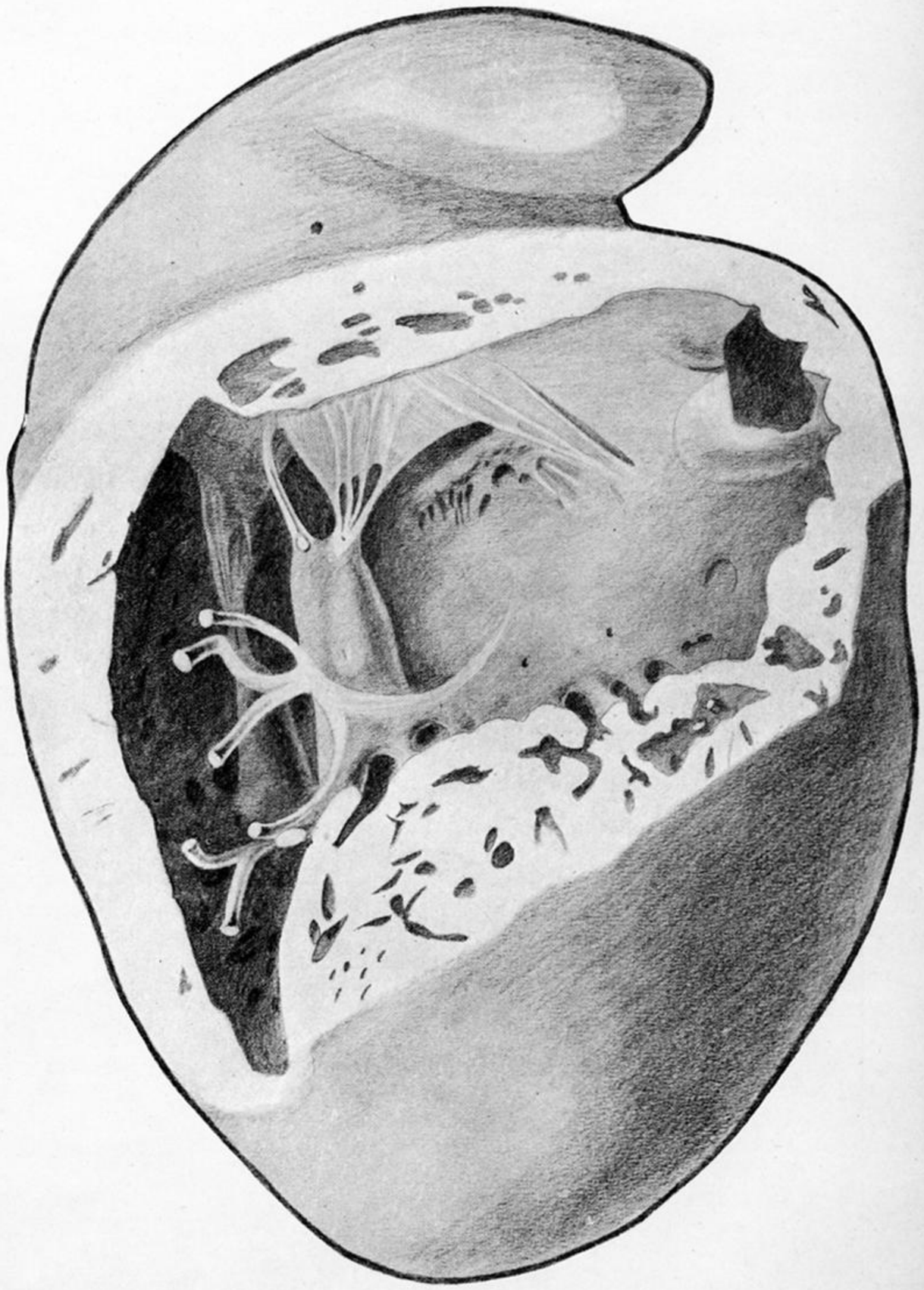

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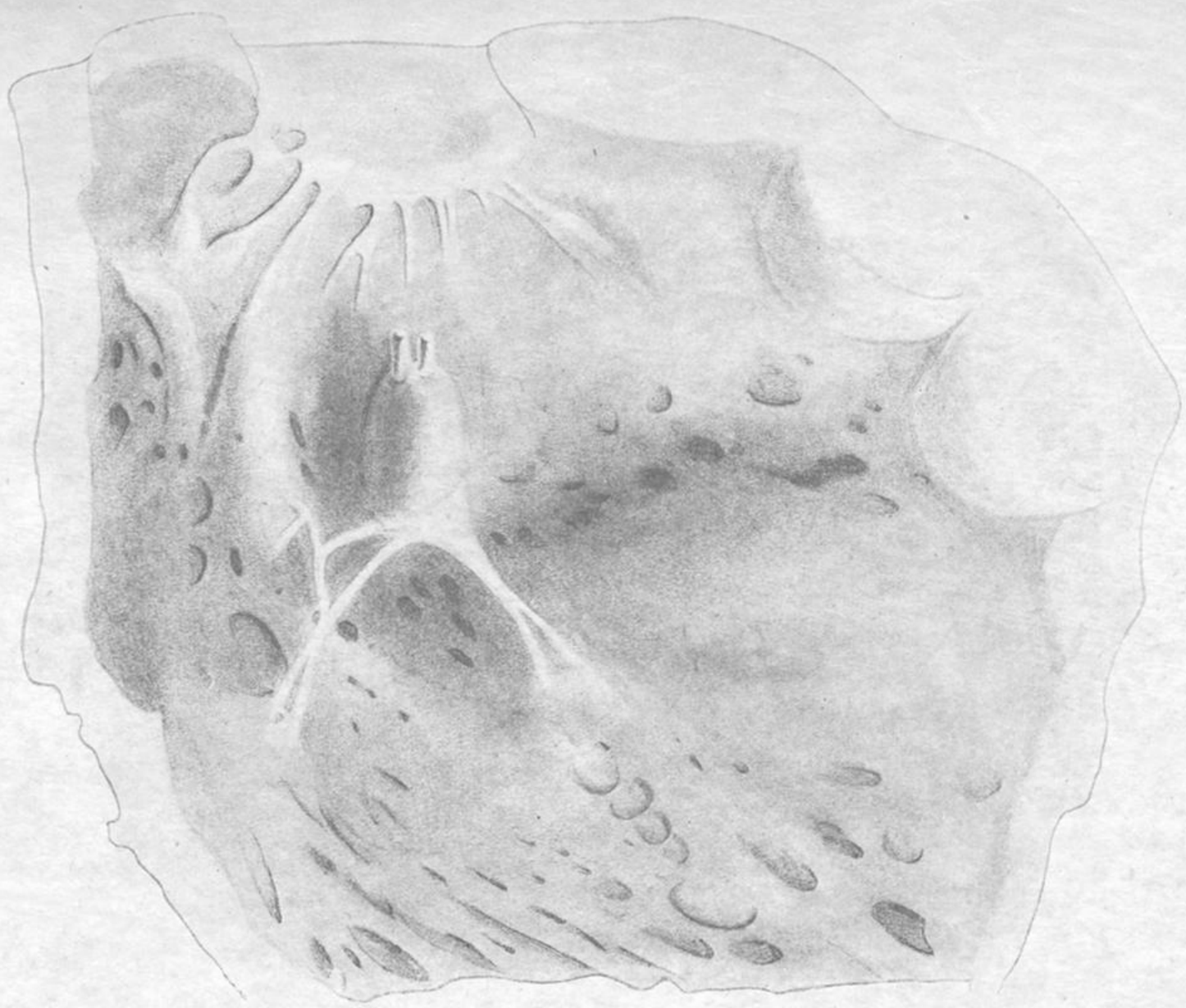

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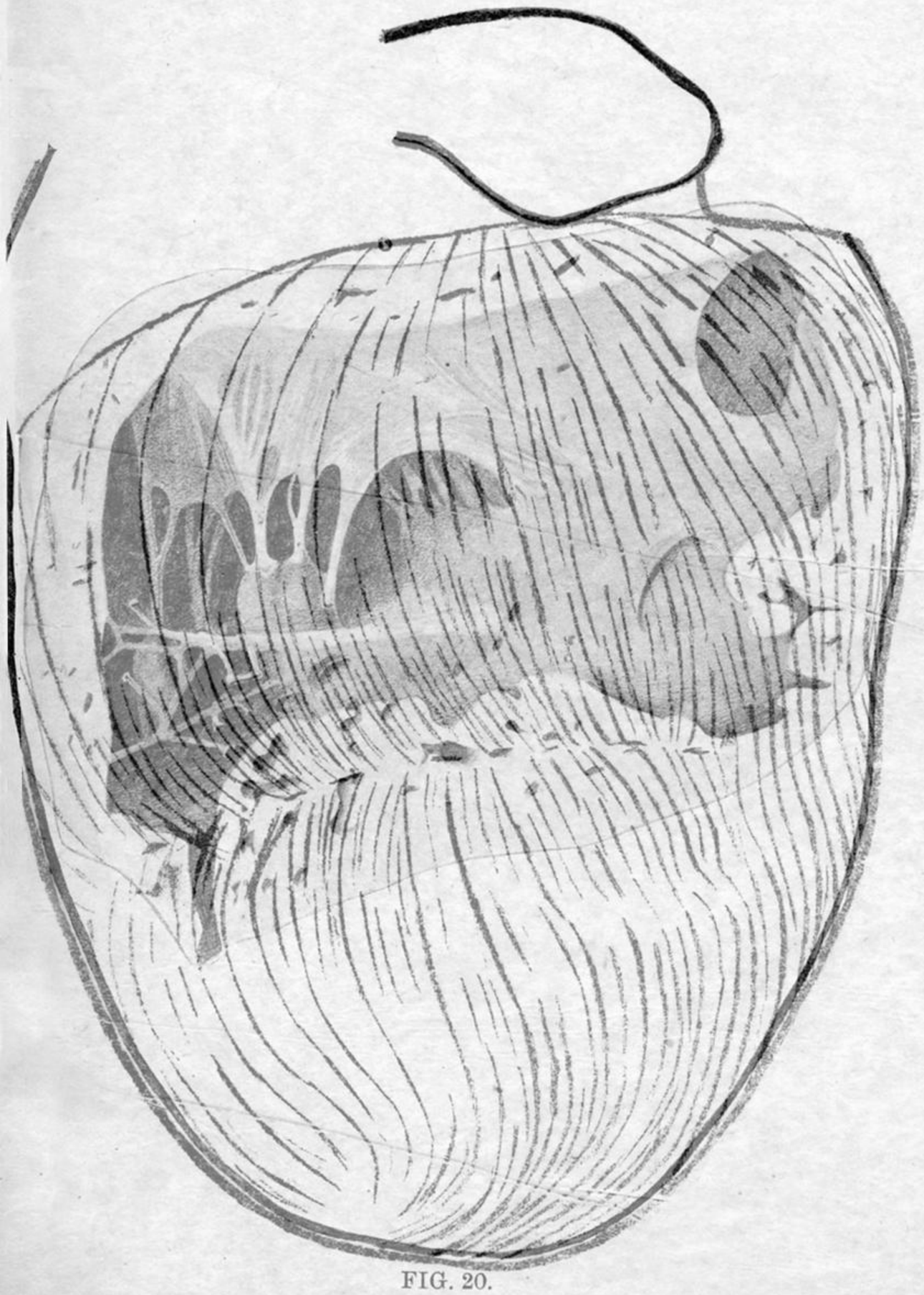

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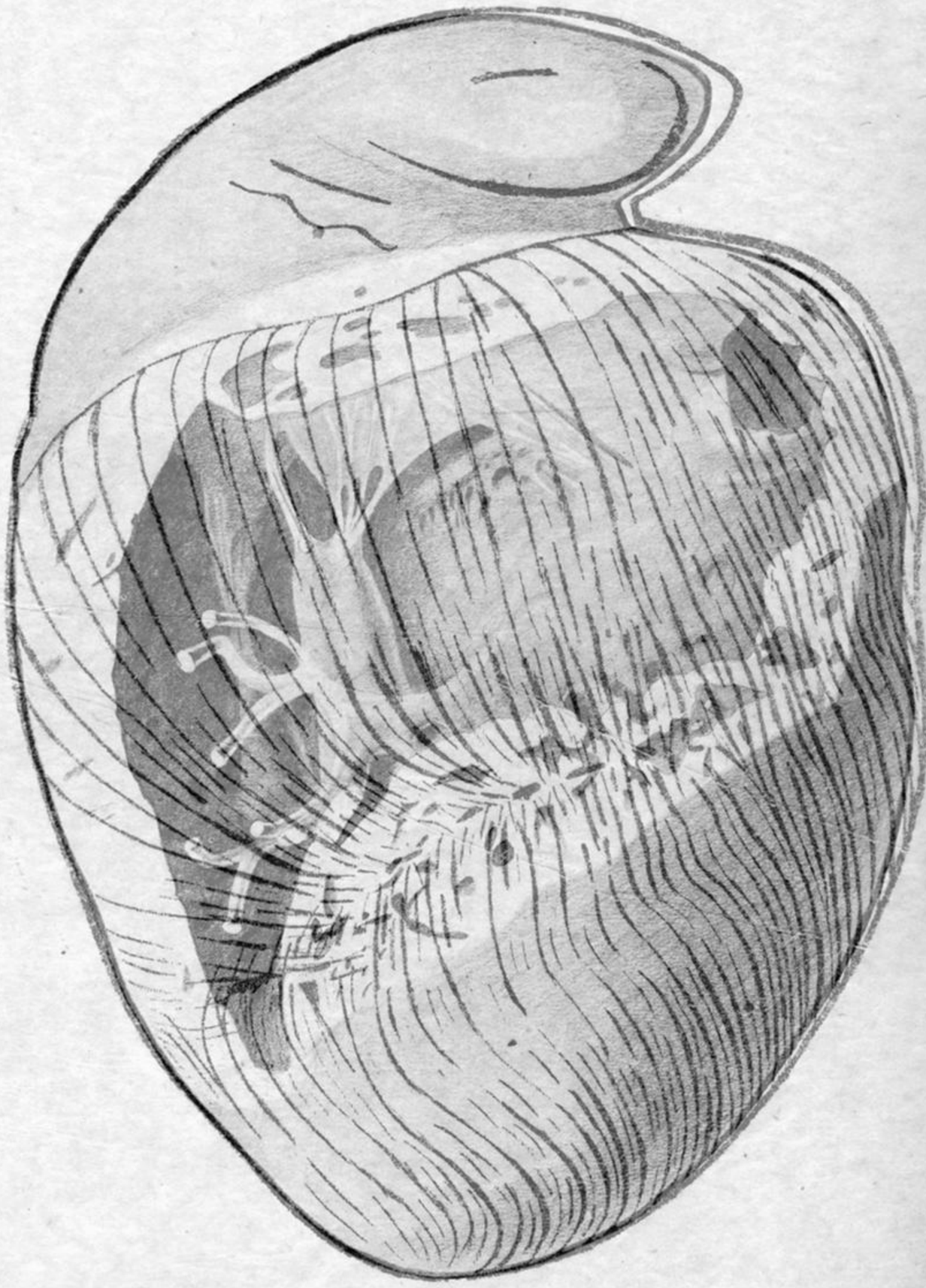

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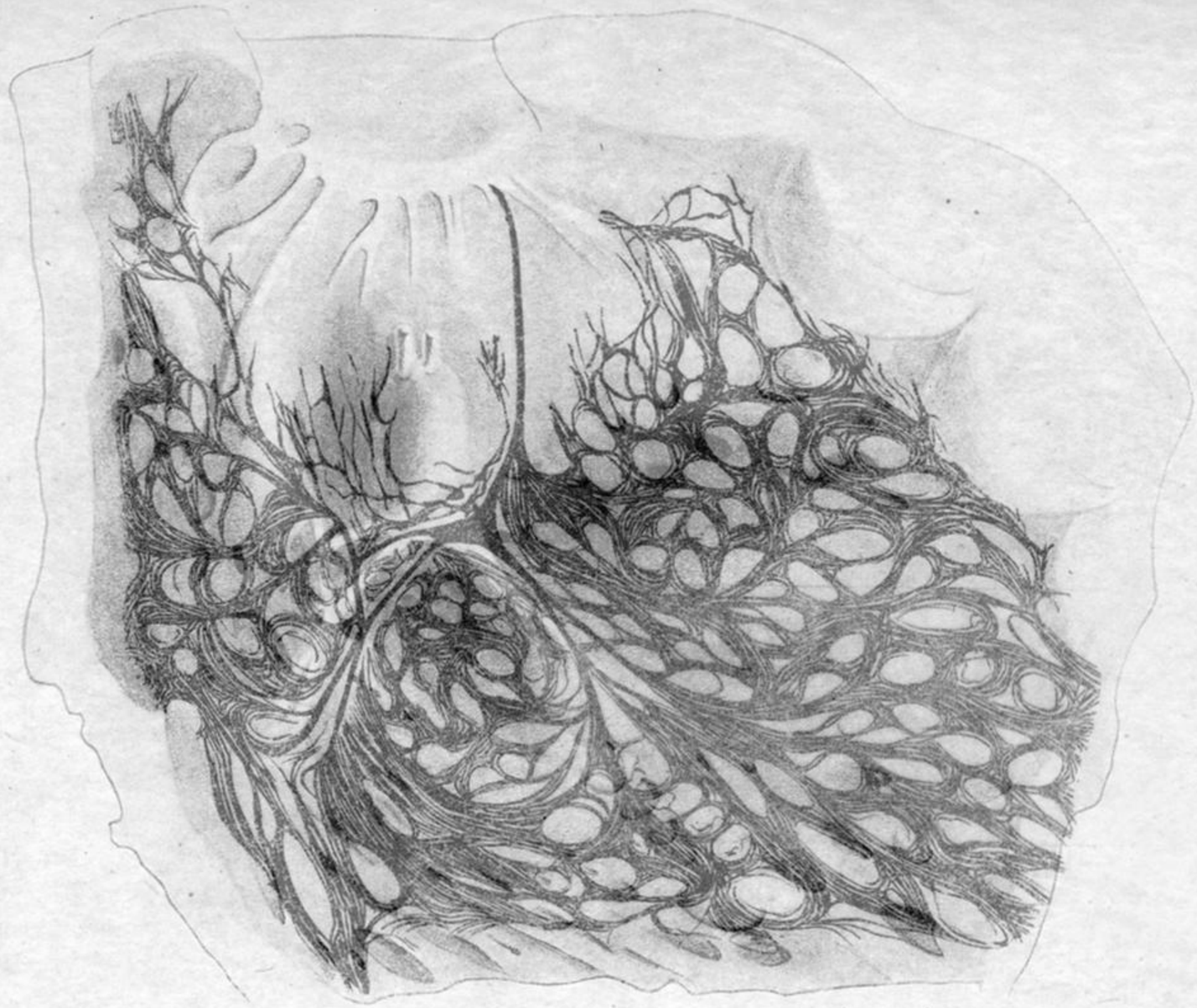

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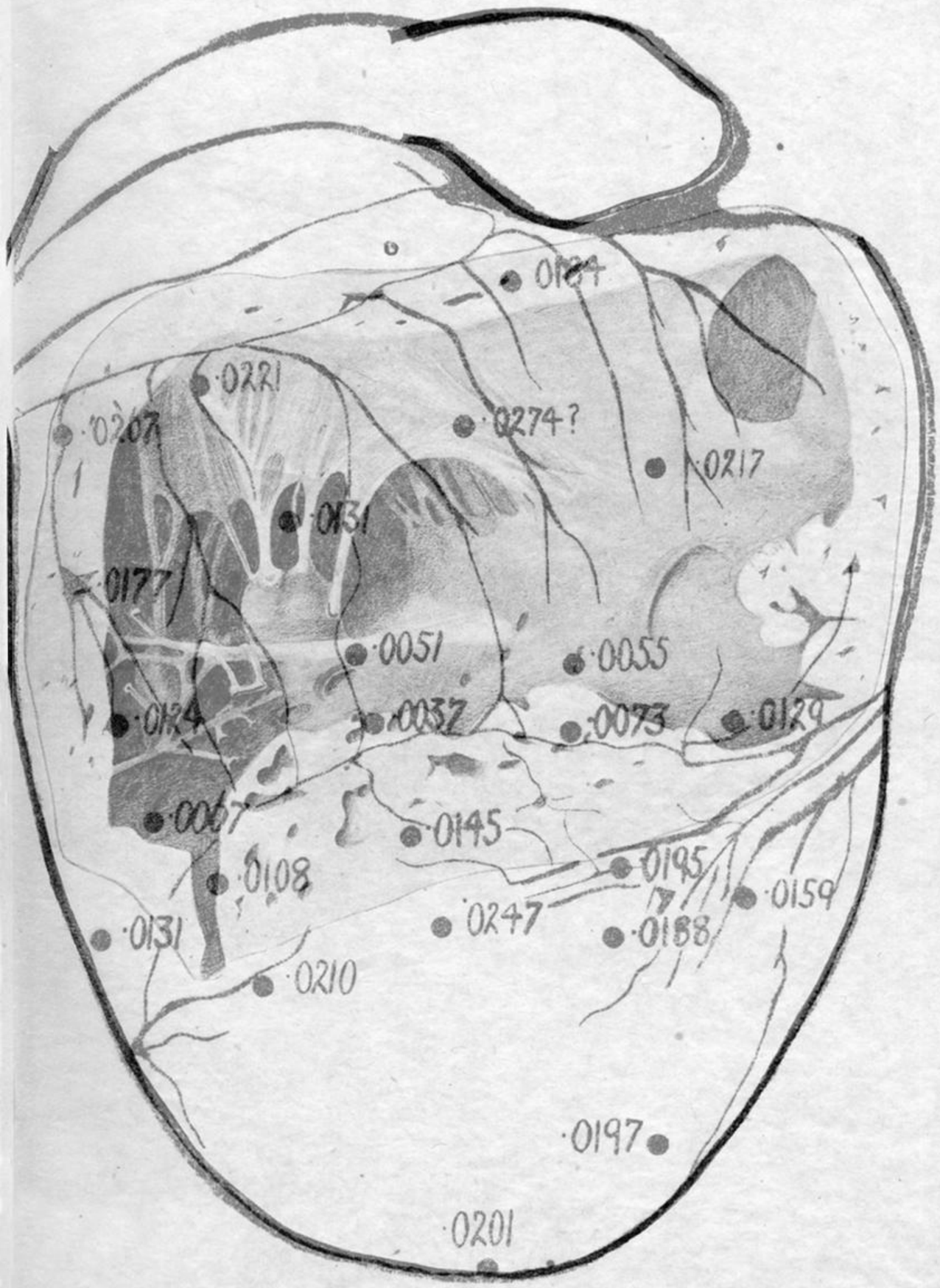

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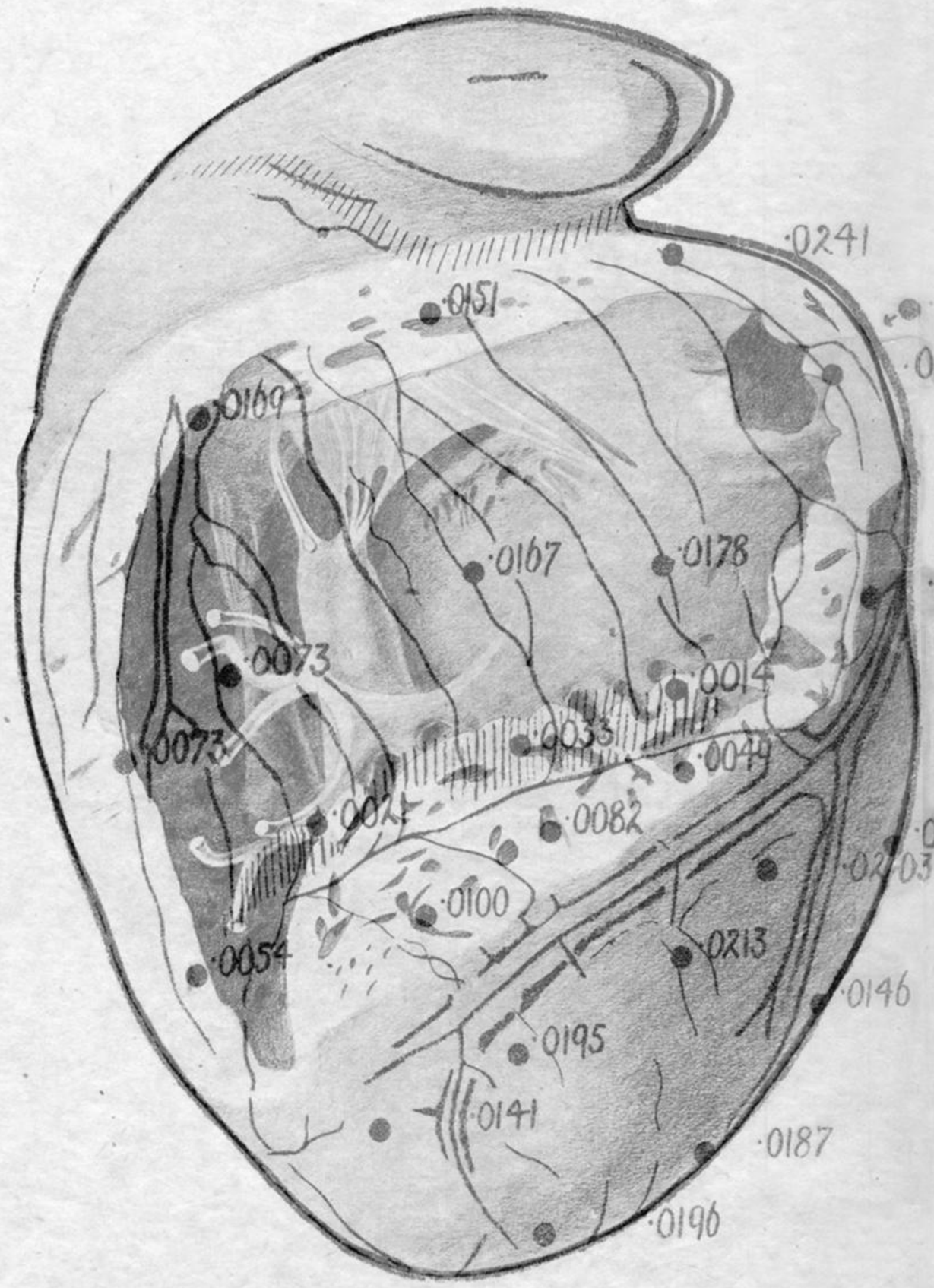

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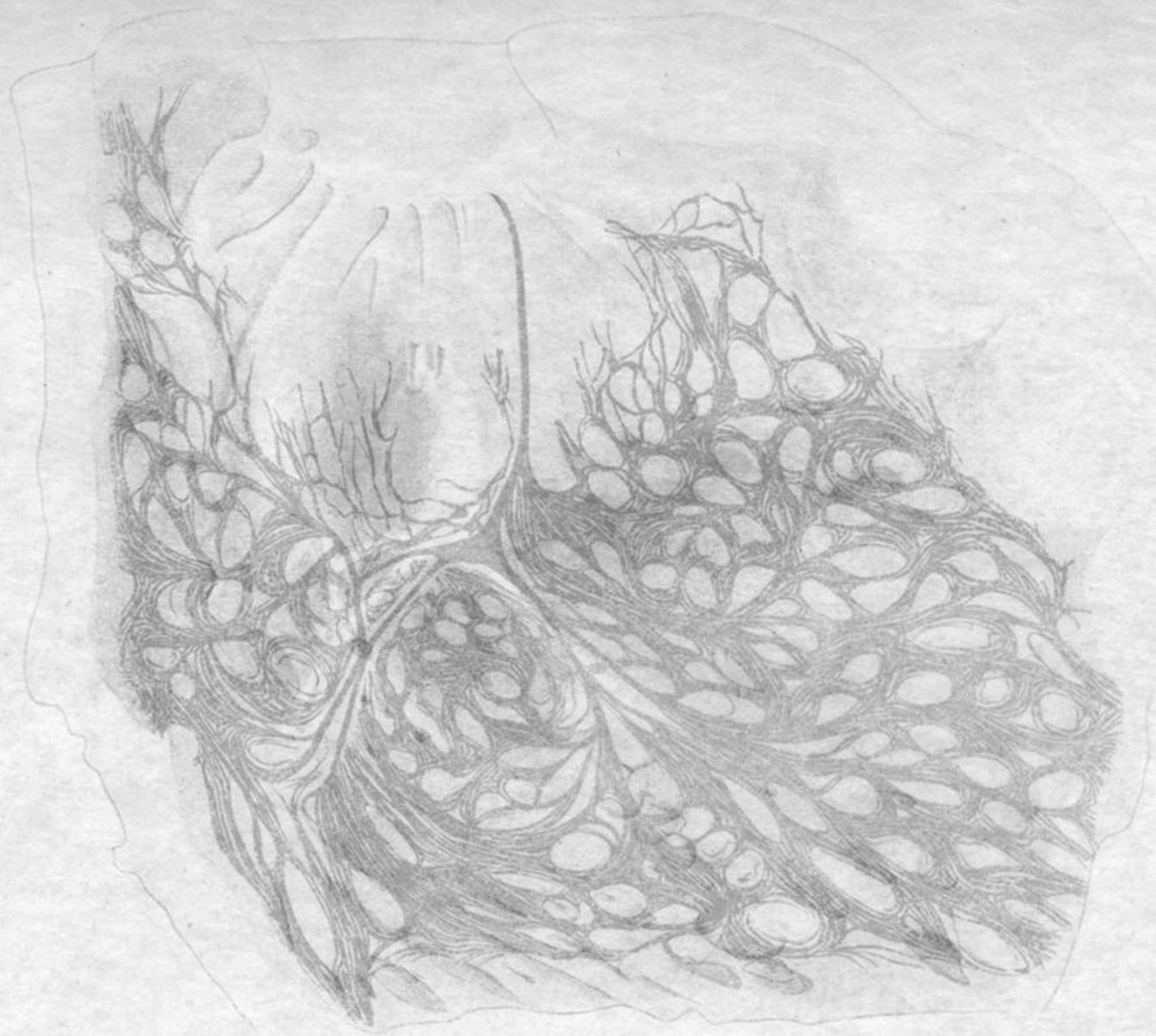

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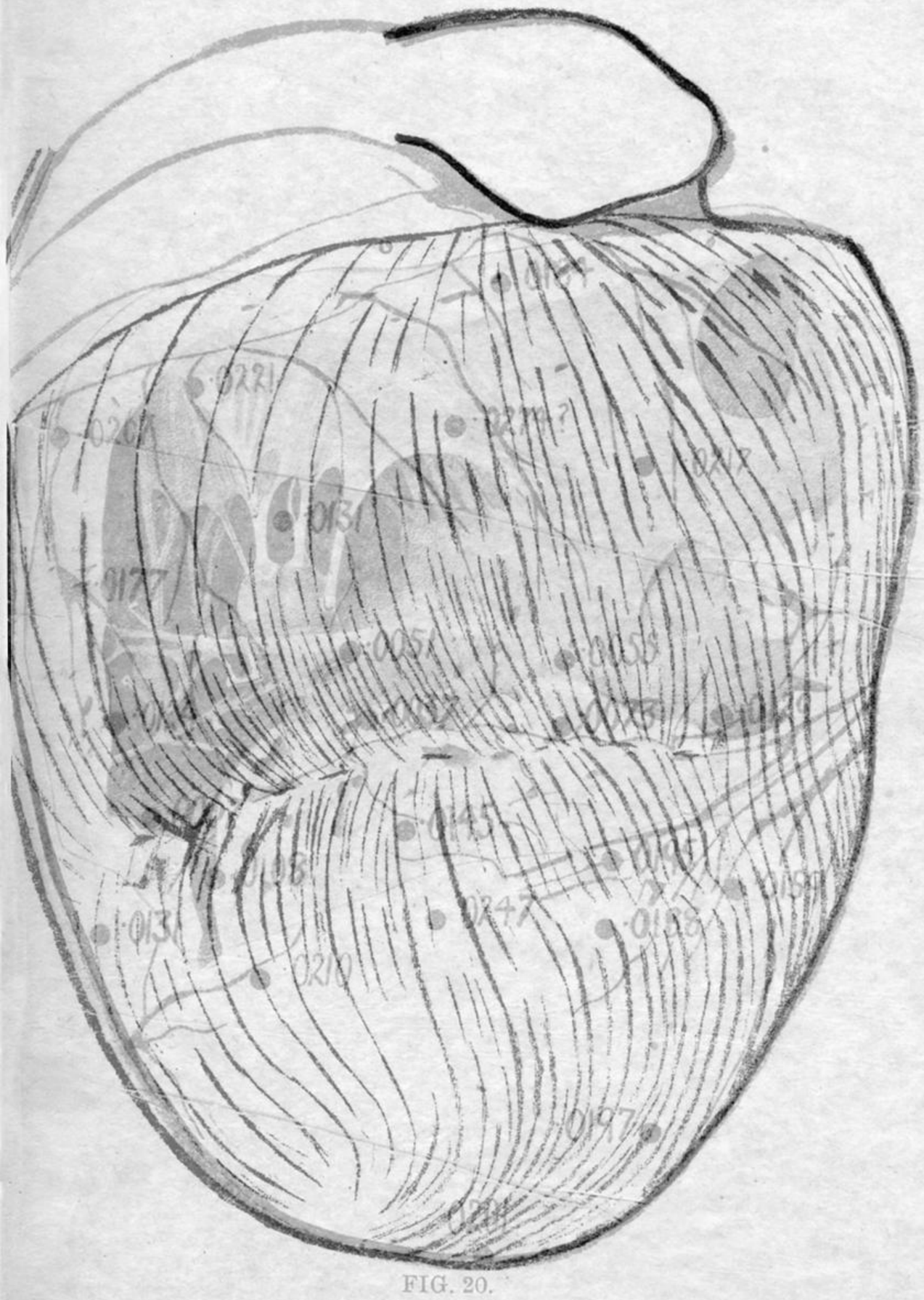

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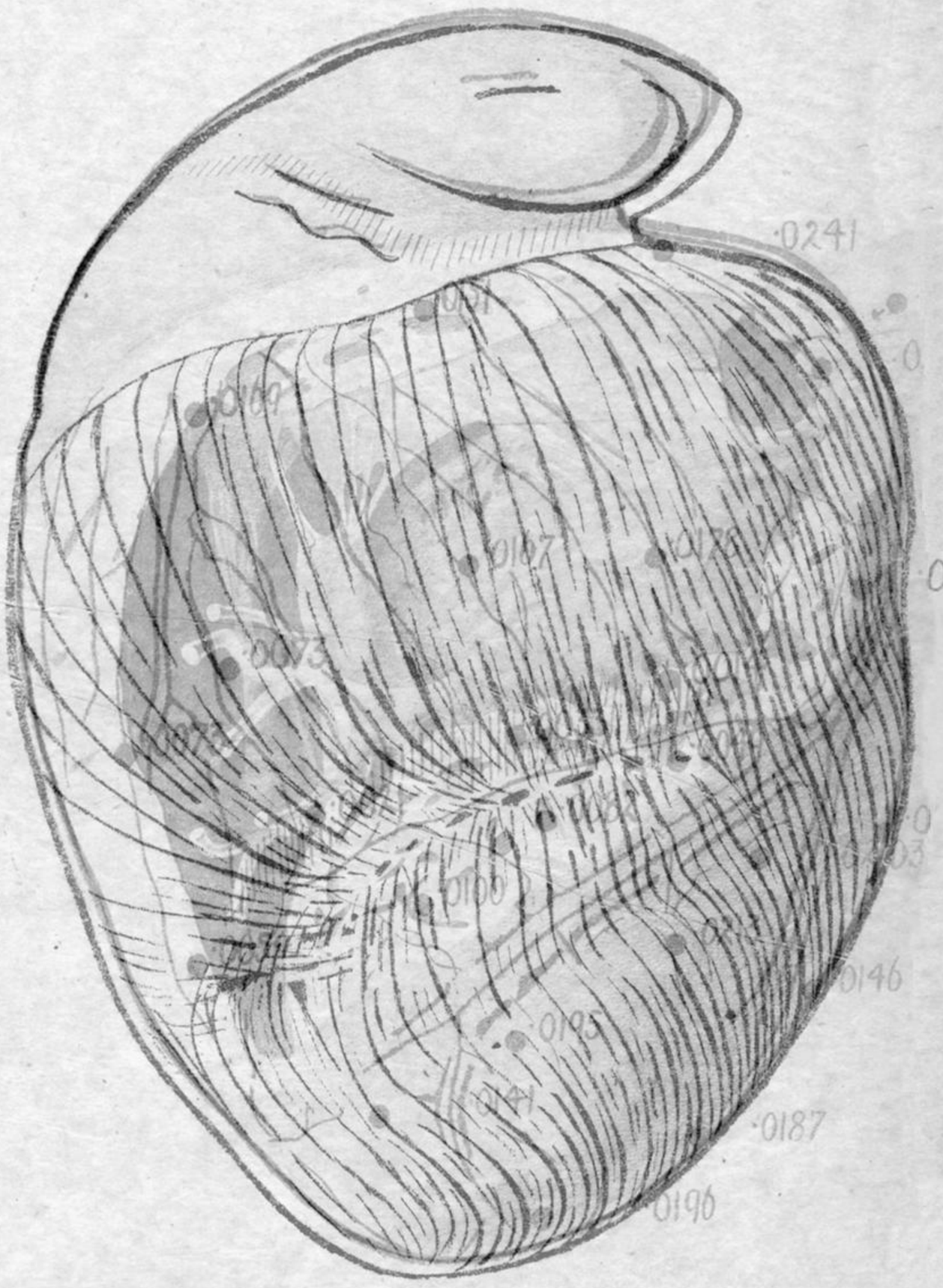

FIG. 21.

Plate 8.

Fig. 19.—A projected drawing (twice natural size) of the interior of the right ventricle of a dog. The outer wall has been freed along the posterior interventricular groove and between the tricuspid and pulmonary valves and thrown downward. The ensheathed branches of the right division of the bundle are seen. The tracing is a drawing of the right branch of the bundle, the Purkinje arborisation and network, displayed by special carmine staining.

Fig. 20.—A projected drawing ($1\frac{2}{5}$ natural size) of the cavity of the right ventricle (Dog G.K.) showing the relations of the large papillary muscle and free arborisation to the trabeculated region and surface. The overlying tracings are projections of the superficial muscle fibres and of the surface of the heart with the contacts and readings of the experiment.

Fig. 21.—A similar projection ($1\frac{2}{5}$ natural size), with similar overlying tracings of the heart of Dog G.L.

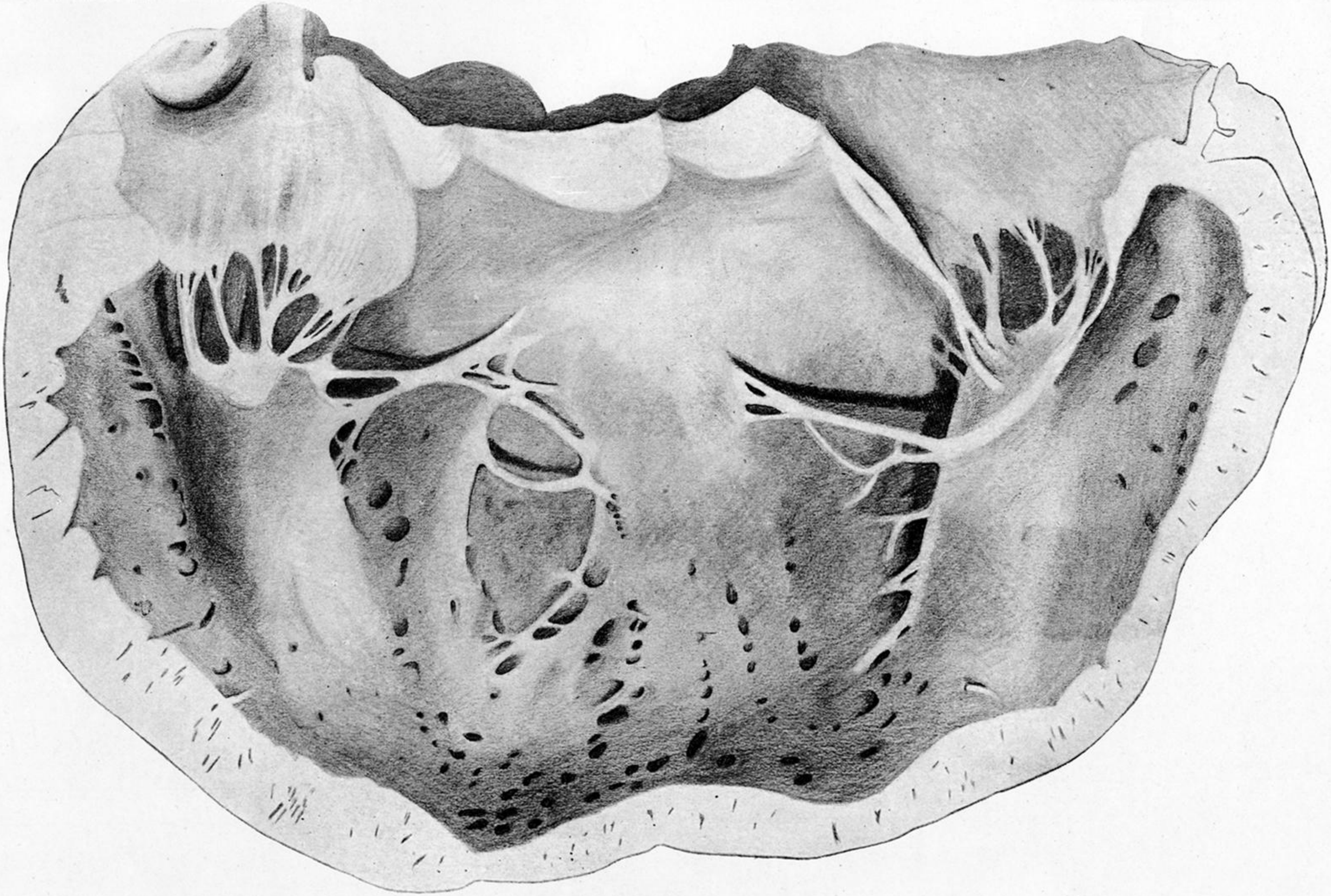

FIG. 22.

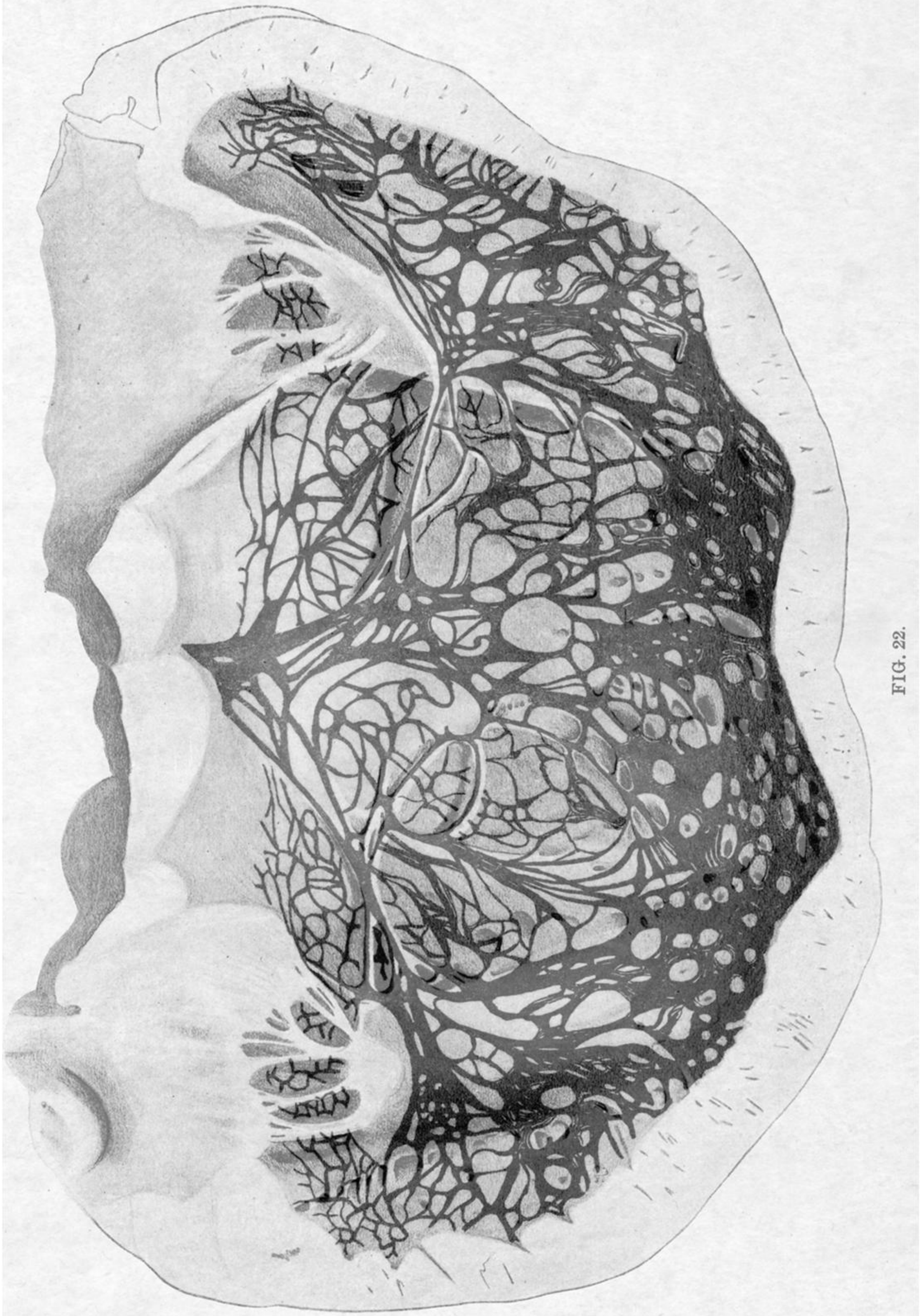

FIG. 22.

PLATE 9.

Fig. 22.—A projection (twice natural size) of the cavity of the left ventricle of a dog, opened between the two papillary muscles, and through the mitral valve. The tracing shows the left branch of the bundle, its arborisation and network. Prepared in similar fashion to fig. 19.

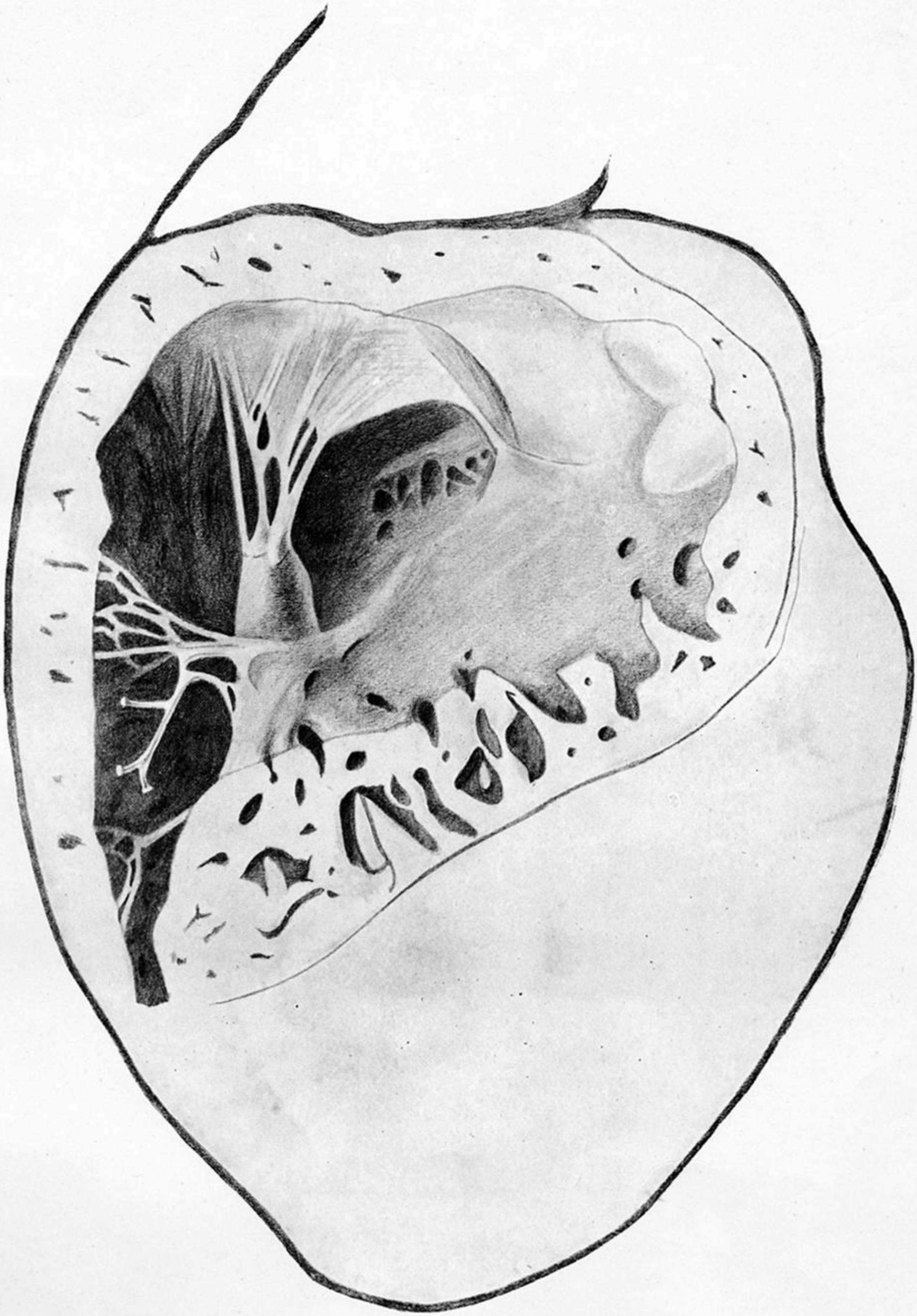

FIG. 23.

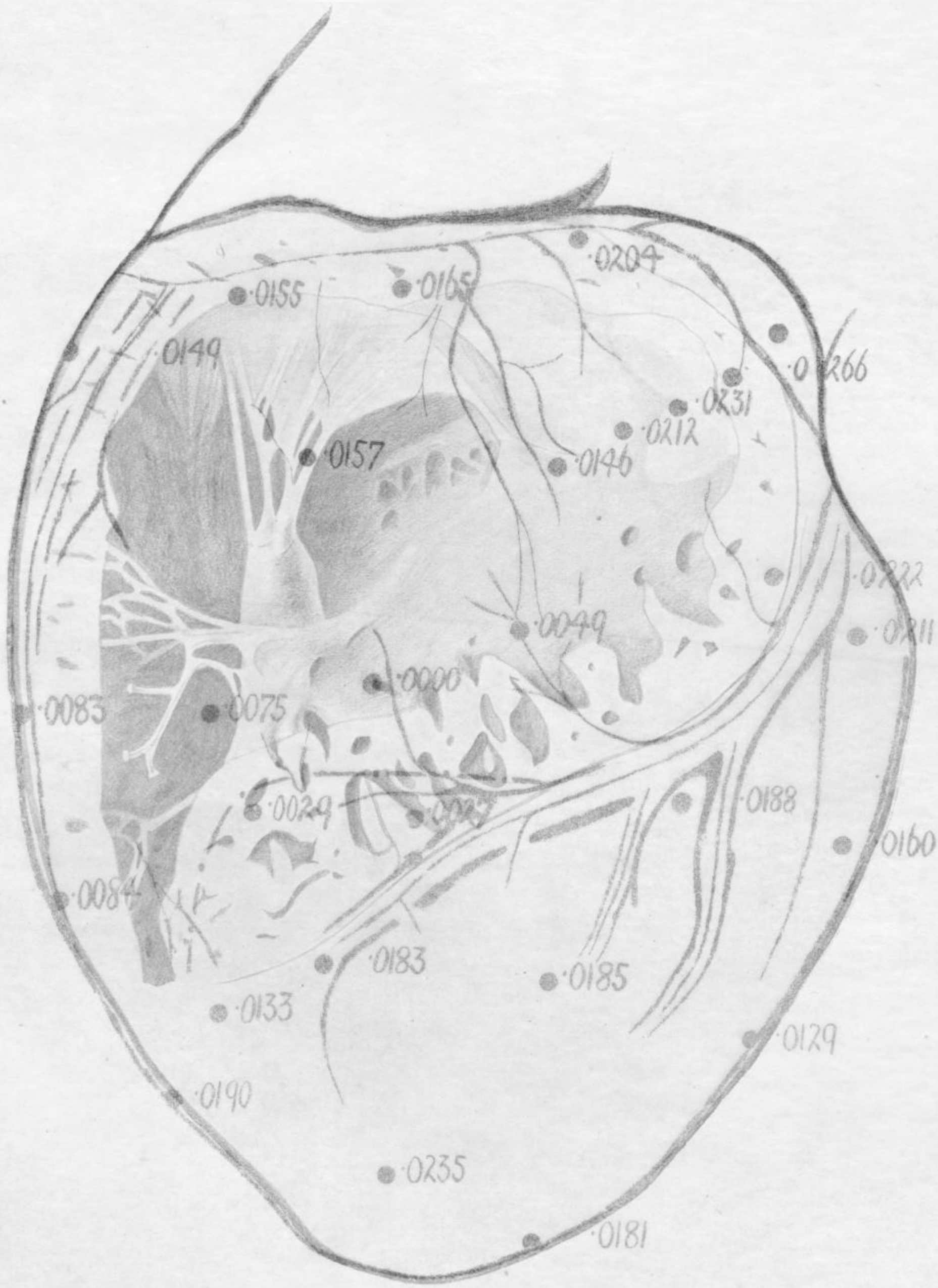
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FIG. 23.

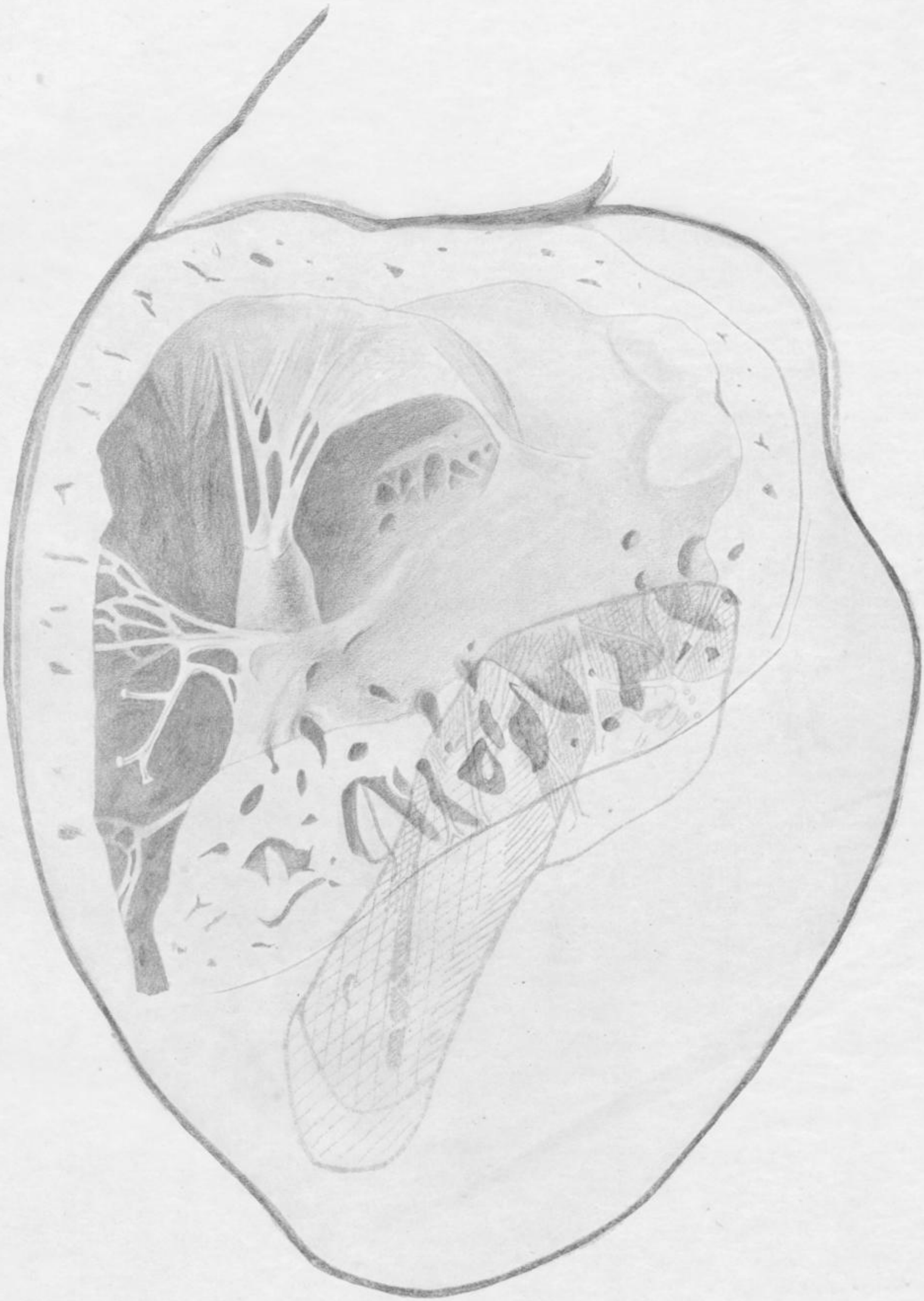


FIG. 23.

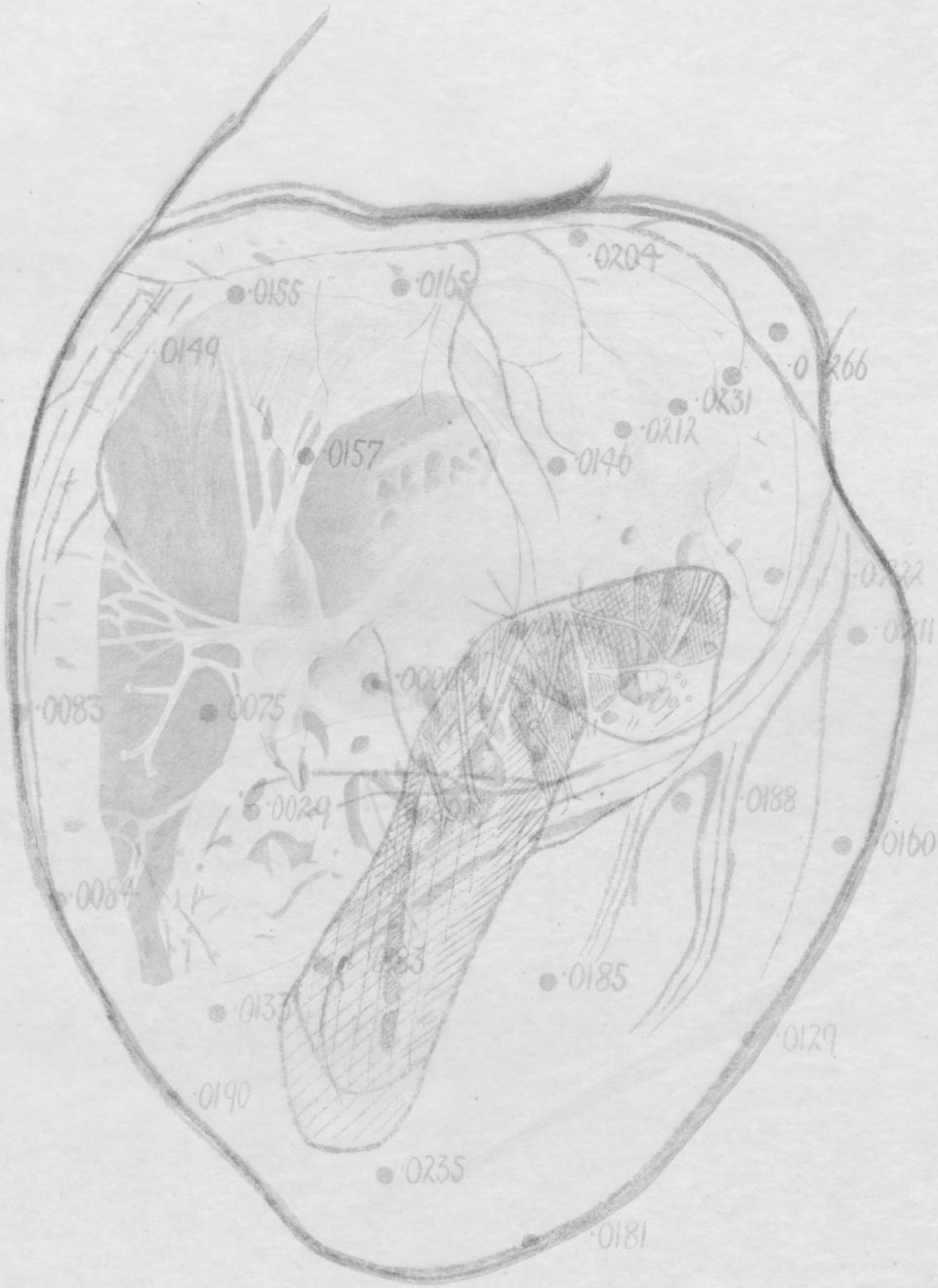


FIG. 23.

Plate 10.

Fig. 23.—A projection ($1\frac{2}{5}$ natural size) of the heart of Dog G.M., similar to that of fig. 20. The overlying tracings are projections of the cavity of the left ventricle, showing the relations of the papillary muscles to the surface, and of the surface of the heart with the contacts and readings of the experiment.

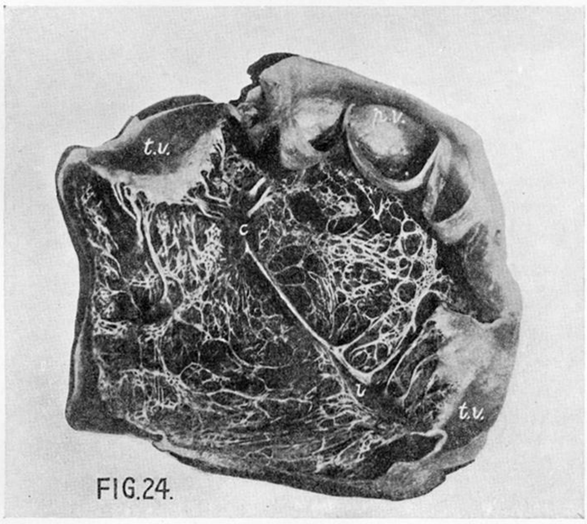
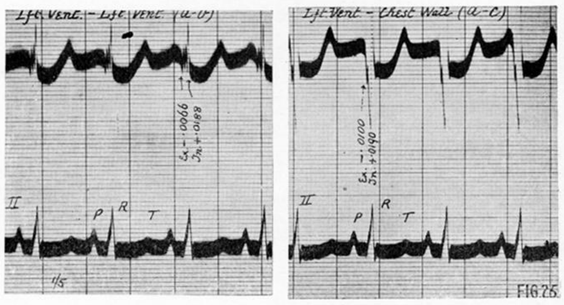


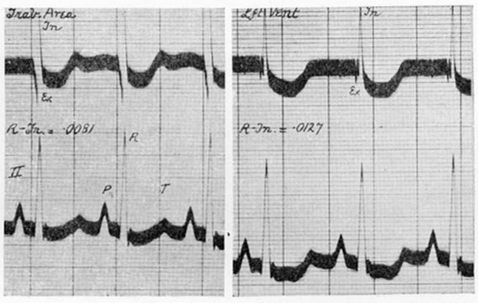
FIG. 24.

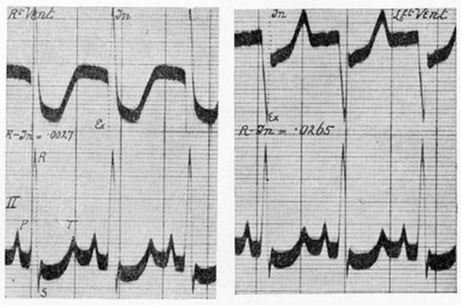


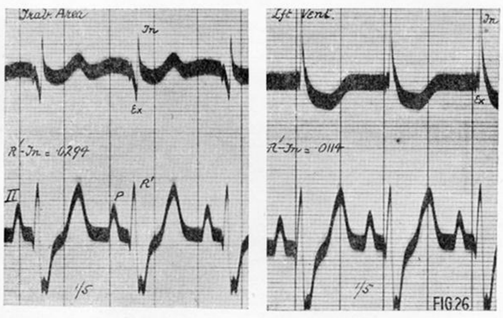
A. B.

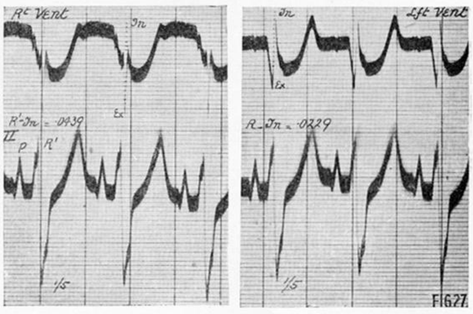
FIG. 25.

A. B.









C. D.

FIG. 26.

C. D.

FIG. 27.

Plate 11.

Fig. 24.—A photograph ($\frac{3}{7}$ natural size) of the interior of the right heart of an ox, in which the sheaths of the Purkinje network have been injected. *p.v.* = pulmonary valves; *t.v.* = tricuspid valve segments; *c.b.* = moderator band. To show the extent of the network.

Fig. 25.—Dog G.J. Two curves ($\frac{2}{3}$ natural size), each simultaneous with the standard electrocardiogram, showing the effects of changing a distal contact. The leads are illustrated in fig. 4 in the text.

Fig. 25 A.—The upper curve is from two contacts (*a*–*b*) on the left ventricle.

Fig. 25 B.—The upper curve is from point *a* on the left ventricle to the left chest wall (*c*). The form of curve in changing the distal contact from *b* to *c* alters considerably. The relation of the first extrinsic deflection to *R* (−0·0066 and −0·0100) varies; the relation of the intrinsic deflection to *R* (0·0188 and 0·0190) remains unchanged. *Ex.* = extrinsic; *In.* = intrinsic deflection.

Ordinates (upper curves), 5 scale divisions = 8·4 millivolts.
Abscissæ = 0·2 second.

Fig. 26.—Dog G.P. Four figures ($\frac{2}{3}$ natural size) showing the effect of cutting the chief branch of the right division of the bundle.

Figs. 26 A and 26 C are simultaneous records from a point on the right ventricle (upper curve in each) and from Lead *II* (lower curve in each). Figs. 26 B and 26 D are similar records from a point on the left ventricle Figs. A and B were taken before and figs. C and D after section of the branch of the bundle. The corresponding contacts may be found by consulting fig. 12.

Ordinates (upper curves), 5 scale divisions = 10 millivolts.
Abscissæ = 0·2 second.

Fig. 27.—Dog G.R. Four records ($\frac{2}{3}$ natural size) arranged in precisely the same fashion as those of fig. 26; and exemplifying a similar experiment. The corresponding contacts may be found by consulting fig. 13.

Ordinates (upper curves), 5 scale divisions = 10 millivolts.
Abscissæ = 0·2 second.

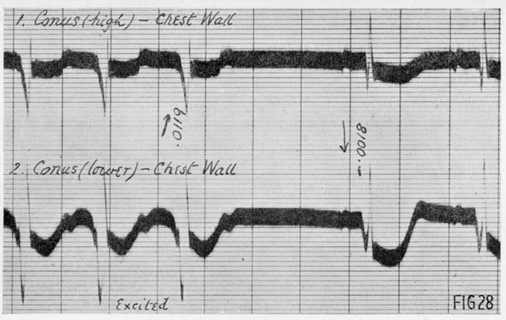


FIG. 28.

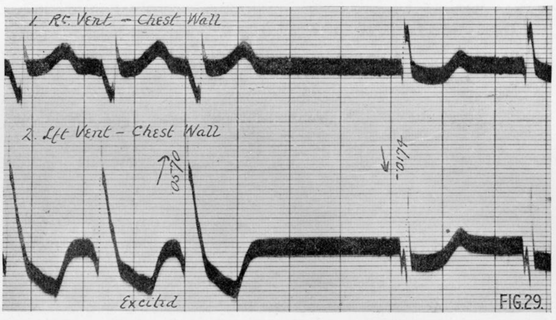


FIG. 29.

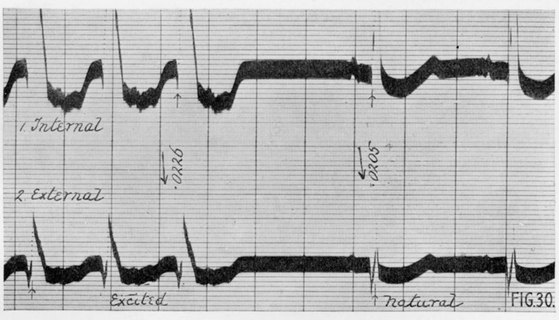


FIG. 30.

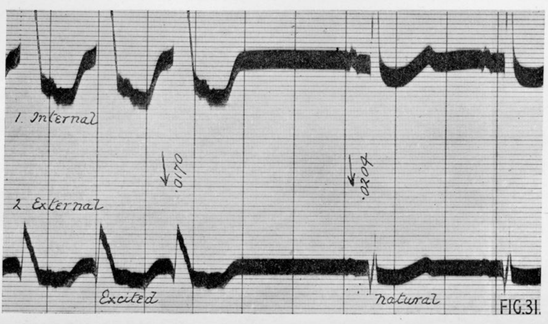


FIG. 31.

Plate 12.

Fig. 28.—Dog G.K. Two simultaneous curves (natural size) (1) from a conus to chest wall lead, and (2) from a point lower and more to the left on the conus.

As shown in the two cycles to the right of the figure, while the heart beat naturally, the excitation wave appeared almost simultaneously at the two conus points. While the heart responded to excitation at a point on the left ventricle in line with these contacts, the contact proximal to the point stimulated received the excitation wave 0·0119 second before the other (the distance of the contacts apart was 16 mm.).

Ordinates (both curves), 5 millimetres = 10 millivolts.

Abscissæ = 0·2 second.

Fig. 29.—Dog G.K. Similar curves from the same animal showing the slower conduction across the interventricular groove. (1) Curve taken from a lead from right ventricle to right chest wall. (2) Curve from a lead from left ventricle to left chest wall. The first three cycles were excited from the left border of the heart, at a point in line with the ventricular contacts; the latter lay 22 mm. apart, and the excitation wave took 0·0570 second to pass between them.

Ordinates (both curves), 5 millimetres = 10 millivolts.

Abscissæ = 0·2 second.

Fig. 30.—Dog G.O. Simultaneous curves (1) leading from an internal point near the base of the right ventricle to the right chest wall, (2) leading from an external point opposite the internal point to the left chest wall (see fig. 15).

While the heart beat naturally (last two cycles) the excitation wave at the internal point preceded that at the external point by an estimated time of 0·0205 second. While the heart beat in response to excitation at a point 15 mm. from the external contact, the excitation wave at the internal point preceded that at the external point by an estimated time of 0·0226 second.

Ordinates (both curves), 5 millimetres = 10 millivolts.

Abscissæ = 0·2 second.

Fig. 31.—Dog G.O. An exactly similar experiment to that illustrated in fig. 30, with the exception that the point of stimulation lay only 6 mm. from the external point. The excitation wave at the internal point now precedes that at the external point by a lessened interval, namely, 0·0170 second, because the external point receives the excitation wave directly and not through the Purkinje system.

Ordinates (both curves), 5 millimetres = 10 millivolts.

Abscissæ = 0·2 second.

Note.—The ordinates for all standard curves (Lead *II*) are on the scale of 3 centimetres (30 scale divisions in reduced figures) = 3 millivolts.