Bulletin No. 5

Further Contributions to the Study of

THE EFFECTS OF LUMBAR LESIONS

RESEARCH DEPARTMENT

Louisa Burns, M. S., D. O., *Acting Director*

FELLOWS

C. R. Atzen, M. D., D. O.
Avis G. Hoskins, D. O.
Earl R. Hoskins, B. S., D. O.
Jane Slosson, D. O.

THE A. T. STILL RESEARCH INSTITUTE
CHICAGO, ILLINOIS

1917

PREFATORY NOTE

The papers in this Bulletin must be regarded as a further contribution to the study of the effects of lumbar lesions upon the pelvic and abdominal organs, and not in any sense as a completed series of experiments. The complete determination of the effects of these lesions must depend upon experiments carried on through several years of time, upon a great number of animals, and interpreted into the language of human reactions by means of the clinic study of hundreds of selected patients. Because it is not justifiable to withhold knowledge which may render osteopathic practice more efficient for a longer time than is necessary to verify and interpret the conclusions resting upon the experiments, and because of our hope that this work may serve to stimulate further investigations along the same line, this work is presented to the profession at this time. A later report will be made, giving in greater detail the microscopic findings of the visceral abnormalities present in lesioned animals.

We now know that certain pathological changes in pelvic and abdominal viscera follow lumbar lesions; we have yet to learn how this is produced, what factors localize in any given animal the effects of the vertebral lesion, and to how great an extent the animal is able during the progress of some months or years to overcome the evil effects of the lesion.

All illustrations have been made from original slides and plates, none of which have been retouched or modified, except by the distinguishing markings, not possibly to be mistaken for changes in the structures portrayed.

The experiments upon white rats reported in this Bulletin were begun in September, 1915. Dr. Grace L. Smith and Dr. Zuie McCorkle of Chicago gave constant help during the winter of 1915-1916 in this work. Dr. J. H. Edmiston was a member of the Institute staff during that year and carried the work alone for several months. In the summer of 1916 and during the winter of 1916-1917 Dr. C. R. Atzen, Dr. Jane Slosson, Dr. Avis G. Hoskins and Dr. E. R. Hoskins carried on the work of experimentation.

LOUISA BURNS,
Acting Director.

July 1, 1917.

Table of Contents

List of Tables

Illustrations and Charts

FURTHER CONTRIBUTIONS TO THE STUDY OF THE EFFECTS OF LUMBAR LESIONS

Previous Experiments.

The following early work was reported in the Journal of the American Osteopathic Association. This includes all experimental study of the effects of lumbar lesions so far as we have been able to find the published reports.

Nov. 1, 1906. "Notes from the Laboratory of the Pacific College." Experiments performed upon a pregnant cat indicate that a lumbar lesion may be responsible for habitual abortion.

Oct., 1907. "Viscero-somatic and Somato-visceral Reflexes." "Stimulation of the spinal tissues from the second lumbar segment to the lumbo-sacral region was followed by uterine contractions in the pregnant animal and by imperfect movements of defecation, urination and erection in twenty other animals. Stimulation of the ovaries and testes initiated muscular contractions near the tenth thoracic vertebra; stimulation of the cecum and appendix initiated contractions of the muscles near the thirteenth or fourteenth thoracic and the first lumbar segments. Stimulation of the rectum, bladder, cervix uteri, and prostate initiated contraction near the lumbo-sacral articulation." None of these reactions occurred after destruction of the sympathetic ganglia of the related segments.

March, 1908. "Preliminary Report upon the Functions of Certain Basal Ganglia." In pregnant animals, stimulation of the cerebral cortex did not cause any recognizable uterine contractions. Stimulation of the region of the red nucleus did initiate uterine contractions which were very regular and which continued until the death of the animal. No stimulation of any maternal tissue caused movements of the fetuses.

July, 1910. "The Immediate Effects of Bony Lesions." Lesions of the eleventh to fourteenth thoracic—eleventh and twelfth in the human—were associated with circulatory disturbances of the intestines, supra-renals, kidneys, ovaries, testes and sometimes the bladder. Clinic records show enuresis, ovaritis, orchitis, cystitis, dysmenorrhoea. First and second lumbar; dila-

tation of the vessels of intestines, kidneys, bladder, ovaries, testes, pregnant uterus, and supra-renals. Clinic records include dysmenorrhoea, hemorrhoids, appendicitis, habitual abortion. Third to fifth lumbar; dilatation of the blood vessels of the colon, bladder and all reproductive organs. The reactions are usually lost on section of the lumbar spinal cord at the level of the second lumbar spine. Clinic records include hemorrhoids, dysmenorrhoea, enuresis.

The work reviewed above was reported from the laboratories and clinics of the Pacific College of Osteopathy.

Later experiments performed at the A. T. Still Research Institute in the investigation of the effects of lumbar lesions were reported in the Journal of the American Osteopathic Association, October, 1916. "Preliminary Study of Lumbar Lesions in White Rats." Louisa Burns, Grace L. Smith, and J. H. Edmiston. Several pathological changes in the pelvic and abdominal viscera were described as occurring in lesioned white rats. These animals are included in a part of the reports in this bulletin.

THE PRODUCTION OF EXPERIMENTAL LESIONS

In this report the lesions have been produced upon animals in one of three ways; section of ligaments, forcible subluxation and tapping. A discussion of other methods will be found in Bulletin No. 4, "The Pathology of the Bony Lesion."

Section of the ligaments of the chosen vertebral articulations gave fairly good results. In the bony lesions found in human subjects, there is reason to infer weakness of certain ligaments as one of the factors which perpetuate the lesion and which also cause its recurrence after the bones have been placed in correct alignment. By section of certain articular and interspinous ligaments this weakness was imitated and a fairly good reproduction of the bony lesion as found in the human subject was produced.

In producing the lesion by forcible subluxation, great care was taken to prevent any injury to the articular tissues. The animals were not anesthetized, but were kept quiet. In many cases they did not struggle, though sometimes they tried to escape; no evidences of pain were noted, or else a very slight "squeak" announced the yielding of the tissues to the manipulation; this sounded more like an expression of surprise than an expression of pain. The entire discomfort was certainly negligible. The procedure was very much like giving an osteopathic treatment for the correction of a lesion of the opposite type. No great amount of force is necessary if the preliminary "trying out" movements have been carefully done, so that the direction of movement is determined. Only a slight additional force is then sufficient to cause subluxation which is visible in the X-ray plate.

Lesions produced in this gentle manner do not always remain present for any considerable time at first. The normal state of the ligaments and muscles, and the fact that the animal throws his weight alternately from one side to the other of his horizontal backbone, lead to the speedy correction of the lesion, as first produced, in a large majority of cases. It was not the purpose, at this time, to reproduce those lesions found in the human subject which are due to serious trauma, but to reproduce those lesions which are due to the strains or other injuries so slight as to receive no atten-

tion when they first occur, or which are the result of some small disturbing forces often repeated or long continued. Such lesions are found in a considerable proportion of patients, and our present work was devoted to the study of these types of maladjustment.

When an individual suffers from any disturbance of the support of the spinal column, a series of curves and individual subluxations is soon produced. These lesions are found in the human subject, but not in the typical form in laboratory animals, which carry their backbones in almost a horizontal position. In the human subject an innominate lesion, differences in the length of the legs, the wearing of shoes with high heels, a forced habit of gait, or lesions of the lumbar vertebrae, or any one of several other etiological factors, disturbs the proper balancing of the weight of the body. In order to maintain equilibrium, the muscles of the side opposite to the concavity and somewhat higher than the lesion contract, thus causing a curve. The necessity for maintaining a fairly erect position of the spinal column above this point leads to an upward prolongation of the series of curves and other lesions, until at last the necessity for maintaining correct bilateral vision causes the head to be held with the eyes level, even at the expense of occipital or upper cervical lesions.

In such a spinal column the articular surfaces of certain points along the spinal column receive the greatest amount of stress; usually these points mark either the crossing of the vertical line of the spine by a curve, or else the occurrence of a lesion involving a single vertebra. (It has often been doubted that a single vertebra alone can be responsible for a lesion; however, an examination of X-ray plates shows that in a spine apparently perfectly normal for its entire extent elsewhere, a single vertebra may be disturbed in such a way that its relations with its next neighbors above and below are disturbed, while these retain normal relationships with the rest of the spinal column. This is true of experimental animals, and also of certain human subjects.)

In such a spinal column, in the human subject, every step of walking, and still more in running or jumping, must be associated with stress upon the articular surfaces whose relations are out of plumb, even if ever so lightly, and this must cause sensory irritations of varying intensity. To imitate this condition in laboratory animals is a difficult problem. The best solution so far is that afforded by the "tapping lesion."

In producing a lesion in this manner, the animal is held perfectly quiet while very gentle taps are administered near the spinous process of the selected vertebra. The taps are not so directed as to affect the vertebral articulations directly, but only indirectly, through tension upon the fascia and the reflex contractions of the spinal muscles. The fact that no recognizable local injury is due to the taps is shown by the absence of pain, of local inflammatory symptoms, of hemorrhages in the subcutaneous connective tissues, or of changes in the skin or the hair of the tapped areas.

The fact that the tapping as administered does cause a bony lesion, is indicated by the usual diagnostic symptoms, and, still more certainly, by the study of the X-ray plates taken of the animals before the tapping was begun, and at various intervals during the process.

EVIDENCE OF THE EXISTENCE OF LESIONS

It is very apparent that any study made of the effects of vertebral lesions without a positive diagnosis in one group of animals of the presence of the desired lesion and in another group of similar animals of freedom from all lesions must be worthless. In this report the diagnosis of the existence of the lesion has been based upon the following evidence:

1. The most authoritative evidence is found in the X-ray plate. This method has been held as the court of last resort in all cases, and it is rare that the stereoscopic plate fails to show a lesion which is palpable to the fingers. It is true that bony maladjustments are sometimes absent in the plate when they are considered to be present by the evidence of palpating fingers; in such cases the X-ray plate nearly always shows the localized edema or fibrosis which is responsible for giving the deceptive findings on palpation. (Fig. 7, 8, 11, 16, 24, 27.)

2. It is not desirable to subject animals to too great dosage of the X-ray, lest the integrity of our studies should be lost. For this reason, especially in watching animals and in maintaining lesions, the other diagnostic findings were trusted. Palpation usually shows with a fair degree of accuracy the location of the lesion, and when several persons, judging independently, agree as to the fact and the location of a lesion its existence may be granted. But comparison of these decisions with X-ray plates shows that it is, as yet, impossible to decide absolutely and invariably between the lesion which is an actual bony maladjustment and the lesion which is a localized edema and fibrosis by any method of diagnosis other than the study of stereoscopic X-ray plates, taken carefully and examined in a good stereoscope in a properly darkened room. Of course, there are lesions that "he who runs may read," but these are not usually the lesions that give the most trouble in symptomatology, in diagnosis, or in treatment.

Palpation gives evidence of bony mal-adjustment if this is marked; of localized tension, which may be due to either edema or fibrosis, and of muscular contraction. Muscular contraction

may be recognized either by a generally increased tone of the spinal muscles, or by the occurrence of localized "knots" or "strings" in the irregularly contracted muscles of the deeper spinal layers.

3. Localized hypersensitiveness. In lesioned animals, especially the day after the lesion has been produced, and usually for as long as the lesion remains, palpation of the tissues around the affected vertebra causes some shrinking, or some reflex muscular contraction, or an effort on the part of the animal to escape. This hypersensitiveness is not always marked, but it appears to be always present in the early weeks of the lesion. In some animals, after the passage of a few weeks, the hypersensitiveness may disappear, though even then it may be elicited on prolonged handling. It is not possible to elicit this hypersensitiveness in normal animals, though non-lesioned animals who suffer from inflammatory visceral disease usually show this same hypersensitiveness in the region of innervation of the affected viscera. No such animals are included in this present report, however.

4. Visceral disturbances. This is not usually recognized in laboratory animals before the autopsies. In certain experiments visceral disturbances are studied before death, and the occurrence of atypical visceral events in any animal was followed by an X-ray examination. In the human subject the occurrence of related visceral disturbances gives more satisfactory evidence than laboratory animals, because it is much easier to secure symptoms and material for laboratory diagnosis from human subjects.

5. Rigidity. This is one of the most conspicuous symptoms of the vertebral lesion in the human subject. It is usually less widespread and less noticeable in laboratory animals, but can usually be found by careful palpation. (Fig. 26, 28.)

6. Other less constant signs of the bony lesion are found in changes in the skin and the hair of the affected area, positions assumed during rest, variations in physiological habits, and other evidences of disturbed function.

A PRELIMINARY STUDY OF THE MOTILITY OF THE GASTRO-INTESTINAL TRACT IN LESIONED ANIMALS

E. R. Hoskins, B. S., D. O.

This is a preliminary report of a study to determine the results of osteopathic lesions upon the motility of the entire gastro-intestinal tract.

In 1911 W. B. Cannon of Harvard University published an epoch-making monograph on the mechanical factors of digestion as studied in animals by the aid of X-radiance. He concerned himself with the rate and manner of passage in the various parts of the gastro-intestinal tract of bismuth impregnated meals of various compositions and consistencies and studied carefully the effect of various emotions upon such passage. In his series the work was done with bismuth sub-nitrate and he notes that after his work the animal usually died. The amount of X-ray given the animals is not stated but probably was quite large. The animals were fastened in holders and upon their backs and kept there until they had, outwardly at least, calmed themselves. The animals were fasted to clear the tract as far as possible before the ingestion of the meal.

We have begun a somewhat similar study of the gastro-intestinal tract in animals with especial regard to the effects of artificially produced osteopathic lesions upon the rate and the manner of the passage of food through the digestive tracts. This work has been so far largely done on cats—a few dogs, guinea pigs, rabbits and white rats also having been used. Other than supposedly normal animals have not as yet been studied except in cats. We are unable to keep dogs at present more than one day (they disturb the neighbors), so what work has been attempted on them is unsatisfactory. Dogs eat readily 150 gms. of a mixture of three parts of creamed salmon and one part of C. P. barium sulphate; are rather easily lesioned and are very satisfactory animals to work with.

Rabbits were difficult to persuade to eat any sort of barium mixture in any uniform quantity. It could be administered by

gavage through a soft rubber catheter but the attending excitement would nullify the results. The best results obtained so far have been by use of a mash of alfalfa leaves into which barium sulphate was carefully stirred. But as yet the amount we are able to introduce is not uniform enough to use satisfactorily.

Guinea pigs do fairly well on the alfalfa meal. They were tried on barium in milk but they seemed to relish the milk better after the barium had settled to the bottom. Pigs have so far refused to take milk after gum tragacanth and other emulsifying agents had been introduced. Some further modifications have yet to be made in technique of feeding rabbits and guinea pigs before their results will be at all accurate.

White rats eat with apparent zest the same mixture as used for dogs, 20 gms. of a 1 to 3 mixture of barium and creamed salmon. They are less satisfactory than the other animals because of their size.

The cats used were males, kept under observation from ten to thirty days before being used to be sure of a fairly normal animal, the animals were several times taken into the X-ray room before being given the meal while the transformer was in operation. Most of them were radiographed before this series of work was begun with them. Only two of the series demonstrated any fright. Most of them purred contentedly while being fluoroscoped and required no holding except gentle pressure with the fluoroscopic screen while being examined.

The meal consisted of a 1 to 3 mixture of barium sulphate and creamed canned salmon, 75 gms. being given in place of the ordinary breakfast. No fasting was used to clean out the tract. The work of flouroscoping would have been easier had this been done, but the purpose of the work was to record what actually goes on in the tract of the animal under every-day normal conditions in comparison with what happens as the result of spinal lesion—with as few outside complicating factors as possible. For the same reasons no cathartics of any sort were used. Animals No. 1, 2 and 3 were allowed to eat as much as they liked of the meal—the dish being weighed before and after they had satisfied themselves. No. 1 ate 225 gms., No. 2, 240 gms. and No. 3, 300 gms. The results of these cases show just what would be expected to take place in a human glutton. The work with these animals caused the standard meal to be made 75 gms. which was

found to outline the stomach well, yet was not great enough to distend the organ beyond its physiological power of contraction.

The animals were placed on a heavy cardboard over an opening in a lead lined box, large enough to contain an X-ray tube without sparking to the walls. The X-rays were directed upward from the tube within the box through the cardboard and animal to the flouroscopic screen held horizontally above the animal. The operator stood at the side of the box and looked downward on the screen. The current in the tube was controlled by a foot-switch beside the box. The tube was operated at a 7-inch back-up spark; ½ to 2 milliamperes of current being used from a Kelley-Koett transformer. The actual time necessary to locate the barium throughout the abdomen is very short after a little practice. A single glance is often sufficient, as the contrast given by small amounts of barium in the animals is striking. The animals were watched at close intervals until the stomach began to empty. Several animals were kept on the box and looked at in rotation. After the stomach began to empty they were looked at less often throughout the day until the small intestine was cleared. Then they were let go until 24 hours, and then were examined about once an hour until the colon was practically free of barium.

The following observations were made and timed in each animal:

1. The time after ingestion of meal until the first food passed out through the pylorus and into the duodenum.
2. The time necessary for the stomach to empty itself.
3. The time necessary for the first barium to reach the cecum.
4. The time to reach the hepatic flexure.
5. The time taken to cross the transverse colon to the splenic flexure.
6. The time taken to practically clear the tract of barium. Small flecks of very finely scattered barium were disregarded in this reading. (Fig. 1, 2.)

Peristalsis even in the smaller animals can be made out if studied carefully but as the purpose of this study was primarily to study passage relationships it was not observed carefully—the idea being to subject the animal to absolutely no more X-ray than was necessary. It is doubtful if any animal received more than 100 milliampere-seconds in all; most of them received less than 75 M. A. S. Some idea of this dosage can be obtained from the

fact that it is about the exposure necessary to make one radiograph of an average human shoulder. The animals did not appear to suffer any ill effects from the meal or the X-radiance except in the cases of those allowed to eat until satisfied. No. 3 had a marked diarrhoea at the end of 24 hours.

It was the plan to make the study of a series of animals which as far as careful examination could determine had no spinal lesions. Then these animals were to be lesioned and kept lesioned from 10 to 14 days and given another meal to compare with the first. This plan could not be carried out on all the animals on account of a scourge of pneumonia which started among them and which carried off especially the lesioned animals very rapidly. Some were treated osteopathically and the usual osteopathic results were obtained, but even a convalescent cat is worthless for this work. Hence there are only seven lesioned animals to report effects on.

The lesions were made by reversing the process by which a lesion at the twelfth dorsal and first lumbar would be corrected. X-ray pictures of these traumatic lesions show more or less edema followed by fibrous formation at the point worked upon. (Figs. 3, 4, 5.)

In the future other methods of making lesions will be tried and the effects from other segments of the spine be studied as well. (See tables on pages 20-27.)

The above results although perforce secured from so few animals are significant in being rather uniform and in all pointing in the same direction. It is noticed that as far as beginning to empty the stomach is concerned, over-eating, fear and excitement, and an osteopathic lesion—not necessarily in the stomach segments have about equal effects. This delaying of action on the part of the stomach from a lesion below that region specifically supplying it is in accordance with W. J. Mayo's statement that "The stomach is the alarm box of the abdomen."

Every radiologist sees many cases of big distended stomachs from chronic over-eating which often lie dormant during barium examination for the first two or three hours, emptying in probably five hours without any other demonstrable pathology. It is seldom in the X-ray room that even a patient who has the over-eating habit can be induced to take enough of a barium mixture to over-distend, but it is necessary to use a routine amount here

SERIES A—TIME FOR NON-LESIONED CATS

Cat No.	(1) Through Pylorus	(2) Stomach Empty	(3) Cecum	(4) Hep. flex.	(5) Splen. flex.	(6) Empty
(*3) 1	20 min.	4 hrs. 30 min.	3 hrs.	3 hrs. 30 min.	5 hrs.	36 hrs.
(*3) 2	18 min.	3 hrs.	2 hrs. 30 min.	3 hrs. 30 min.	6 hrs.	36 hrs.
(*3) (*) 3	30 min.	2 hrs. 55 min.	3 hrs.	5 hrs.	5 hrs. 50 min.	24 hrs.
4	8 min.	2 hrs. 45 min.	2 hrs. 45 min.	4 hrs.	6 hrs.	30 hrs.
5	4 min.	3 hrs.	3 hrs. 30 min.	5 hrs.	7 hrs.	30 hrs.
6	6 min.	3 hrs. 20 min.	3 hrs.	4 hrs.	6 hrs.	24 hrs.
7	5 min.	3 hrs. 15 min.	3 hrs. 15 min.	4 hrs. 30 min.	6 hrs.	32 hrs.
8	8 min.	4 hrs.	3 hrs. 45 min.	5 hrs. 30 min.	7 hrs.	36 hrs.
(*2) 9	19 min.	3 hrs. 45 min.	4 hrs.	5 hrs.	6 hrs. 30 min.	30 hrs.
10	10 min.	3 hrs.	3 hrs. 30 min.	5 hrs.	6 hrs. 45 min.	24 hrs.
11	10 min.	4 hrs.	3 hrs. 45 min.	4 hrs. 30 min.	6 hrs. 30 min.	30 hrs.
(*2) 12	20 min.	2 hrs.	3 hrs. 15 min.	4 hrs.	5 hrs.	24 hrs.
13	8 min.	3 hrs. 20 min.	3 hrs. 30 min.	4 hrs. 30 min.	6 hrs.	30 hrs.
14	6 min.	2 hrs. 45 min.	3 hrs.	4 hrs. 10 min.	5 hrs. 45 min.	24 hrs.
15	7 min.	3 hrs.	2 hrs. 45 min.	4 hrs.	6 hrs.	24 hrs.
16	11 min.	3 hrs. 20 min.	3 hrs.	3 hrs. 45 min.	5 hrs. 30 min.	24 hrs.
17	5 min.	2 hrs. 45 min.	3 hrs.	4 hrs.	5 hrs.	24 hrs.
18	7 min.	3 hrs. 15 min.	3 hrs.	4 hrs. 15 min.	6 hrs.	30 hrs.
19	8 min.	3 hrs.	3 hrs. 15 min.	4 hrs. 10 min.	5 hrs. 30 min.	32 hrs.
20	6 min.	2 hrs. 50 min.	3 hrs.	3 hrs. 45 min.	5 hrs.	30 hrs.
21	9 min.	2 hrs. 30 min.	3 hrs. 20 min.	4 hrs. 30 min.	6 hrs. 20 min.	30 hrs.
22	11 min.	2 hrs. 40 min.	3 hrs. 30 min.	5 hrs.	6 hrs.	24 hrs.
23	4 min.	3 hrs. 15 min.	3 hrs.	3 hrs. 30 min.	5 hrs.	24 hrs.
24	6 min.	3 hrs.	3 hrs. 20 min.	4 hrs. 15 min.	5 hrs. 30 min.	24 hrs.
25	7 min.	3 hrs.	3 hrs. 45 min.	4 hrs. 45 min.	5 hrs. 50 min.	27 hrs.
26	6 min.	3 hrs.	3 hrs. 15 min.	4 hrs. 30 min.	6 hrs.	24 hrs.
27	6 min.	3 hrs. 10 min.	3 hrs. 10 min.	4 hrs. 30 min.	5 hrs. 15 min.	24 hrs.

SERIES B—TIME FOR CATS OF ABOVE SERIES AFTER LESIONING BETWEEN THE TWELFTH DORSAL AND THE FIRST LUMBAR. SAME TECHNIQUE ADHERED TO IN BOTH SERIES

10	19 min.	5 hrs. 30 min.	5 hrs.	6 hrs. 30 min.	8 hrs.	36 hrs.
13	18 min.	4 hrs. 30 min.	5 hrs.	6 hrs. 15 min.	7 hrs. 30 min.	32 hrs.
16	21 min.	5 hrs.	5 hrs. 30 min.	6 hrs. 30 min.	7 hrs. 45 min.	30 hrs.
19	17 min.	4 hrs. 15 min.	5 hrs.	6 hrs.	7 hrs.	30 hrs.
20	15 min.	4 hrs. 20 min.	4 hrs. 45 min.	5 hrs. 30 min.	6 hrs. 45 min.	27 hrs.
21	17 min.	3 hrs. 50 min.	3 hrs. 15 min.	5 hrs. 45 min.	7 hrs.	30 hrs.
22	18 min.	3 hrs.	3 hrs. 15 min.	4 hrs. 30 min.	6 hrs.	27 hrs.

(*) Cat No. 3 had pronounced diarrhoea at end of 24 hours.
(*2) Cats Nos. 9 and 12 were "neurotic" individuals; were much excited at the beginning of the series; could not be quieted by petting.
(*3) Cats Nos. 1, 2 and 3 were not limited in amount of meal taken so stomach was greatly distended; too far for normal action.

AVERAGE TIME FOR ALL OF SERIES A, EXCEPT NOS. 1, 2, 3, 9, AND 12

(1)	(2)	(3)	(4)	(5)	(6)
Normals 7.2 min.	3 hrs. 12.5 min.	3 hrs. 12.5 min.	4 hrs. 48 min.	5 hrs. 50.7 min.	26 hrs. 46.2 min.

AVERAGE TIME FOR ALL OF SERIES B, BEFORE LESIONING

Normals 9 min.	2 hrs. 57.1 min.	3 hrs. 17.8 min.	4 hrs. 40 min.	5 hrs. 52.1 min.	27 hrs. 42.8 min.

AVERAGE TIME FOR SERIES B, 10 TO 14 DAYS AFTER LESIONING

18 min.	4 hrs. 20.7 min.	4 hrs. 49.3 min.	5 hrs. 41.4 min.	7 hrs. 08.6 min.	30 hrs. 17.1 min.

Actual Stomach Emptying Time (2) Minus (1)

Series A, except Nos. 1, 2, 3, 9 and 12,	3 hrs. 5.3 min.
Series B, before lesioning,	2 hrs. 48.1 min.
Series B, after lesioning,	4 hrs. 2.7 min.

Pylorus to Cecum, (3) Minus (1)

Series A, except Nos. 1, 2, 3, 9 and 12,	3 hrs. 5.3 min.
Series B, before lesioning,	3 hrs. 8.8 min.
Series B, after lesioning,	4 hrs. 31.3 min.

Cecum to Hepatic Flexures, (4) Minus (3)

Series A, except Nos. 1, 2, 3, 9 and 12,	1 hr. 35.5 min.
Series B, before lesioning,	1 hr. 22.8 min.
Series B, after lesioning,	52.1 min.

Hepatic Flexure to Splenic Flexure, (5) Minus (4)

Series A, except Nos. 1, 2, 3, 9 and 12,	1 hr. 2.7 min.
Series B, before lesioning,	1 hr. 12.1 min.
Series B, after lesioning,	1 hr. 27.2 min.

Cecum to Cleaning of Tract, (6) Minus (3)

Series A, except Nos. 1, 2, 3, 9 and 12,	23 hrs. 33.7 min.
Series B, before lesioning,	24 hrs. 25 min.
Series B, after lesioning,	25 hrs. 17.8 min.

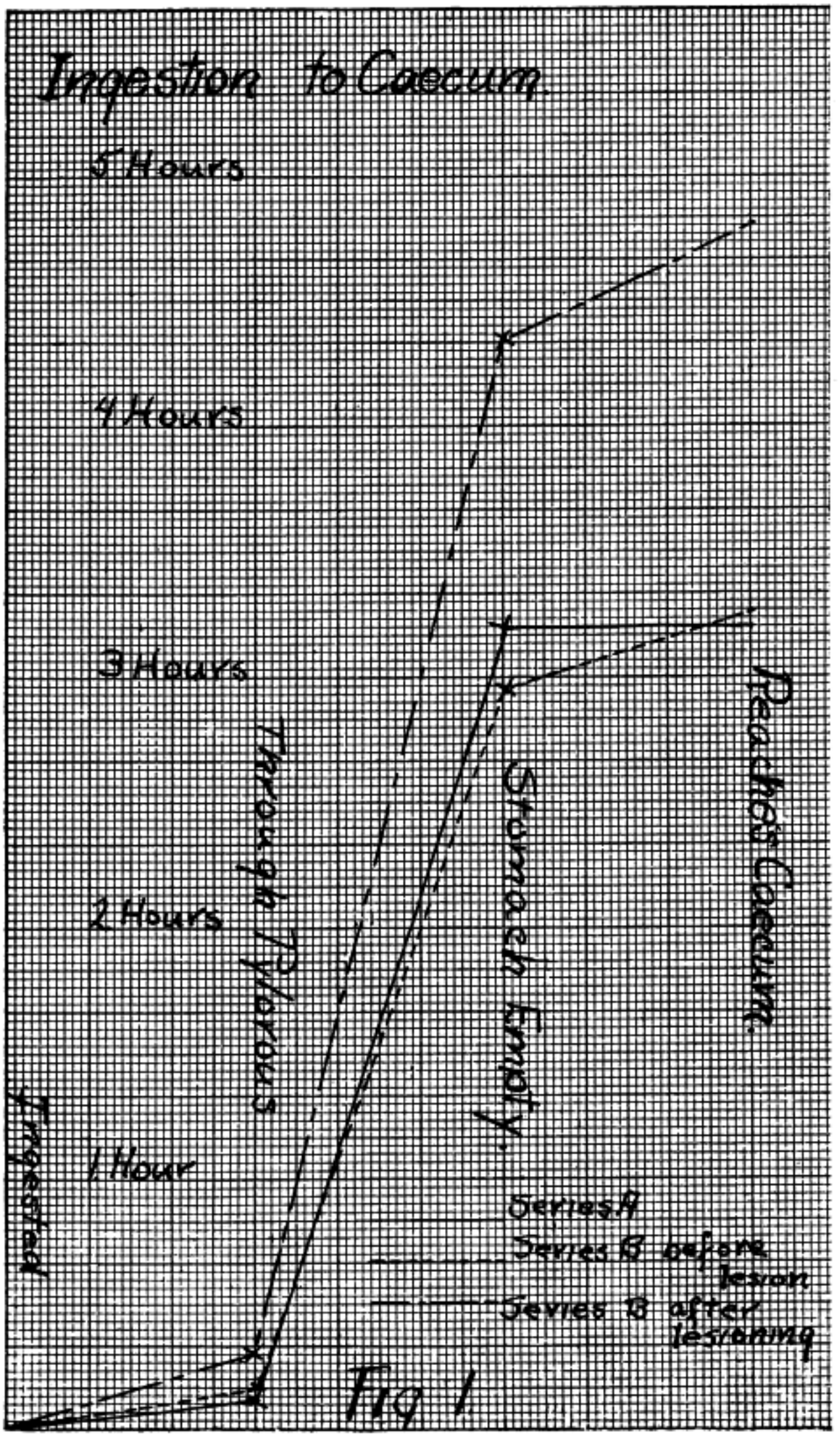
Ingestion to Caecum.
5 Hours
4 Hours
3 Hours
2 Hours
1 Hour
Ingestion
Through Villi Workers
Crosses Empty.
Ingestion to Caecum.
Series A
Series B before lesson
Series B after lesson
Fig 1

Fig. 1

Fig. 1. The results of the series from the ingestion of the meal until the first barium was seen in the cecum are plotted to make comparison of findings easier. The time was plotted as ordinates, allowing three large squares to the hour as shown. Arbitrarily three large squares were allowed to each observation in abscissae as shown.

The continuous line represents the average behavior of the cats in Series A, except Nos. 1, 2, 3, 9 and 12, as shown by the first table.

The dotted line represents the average behavior of the cats, which were afterwards successfully lesioned, before they were lesioned. The material for the second table is part of the first table. The tabular results as well as the plotted lines show that the animals which were later lesioned were normal; the two lines running quite close together.

The broken line represents the average behavior of the cats in Series B after they were lesioned. The persistent slowing of action is shown rather nicely.

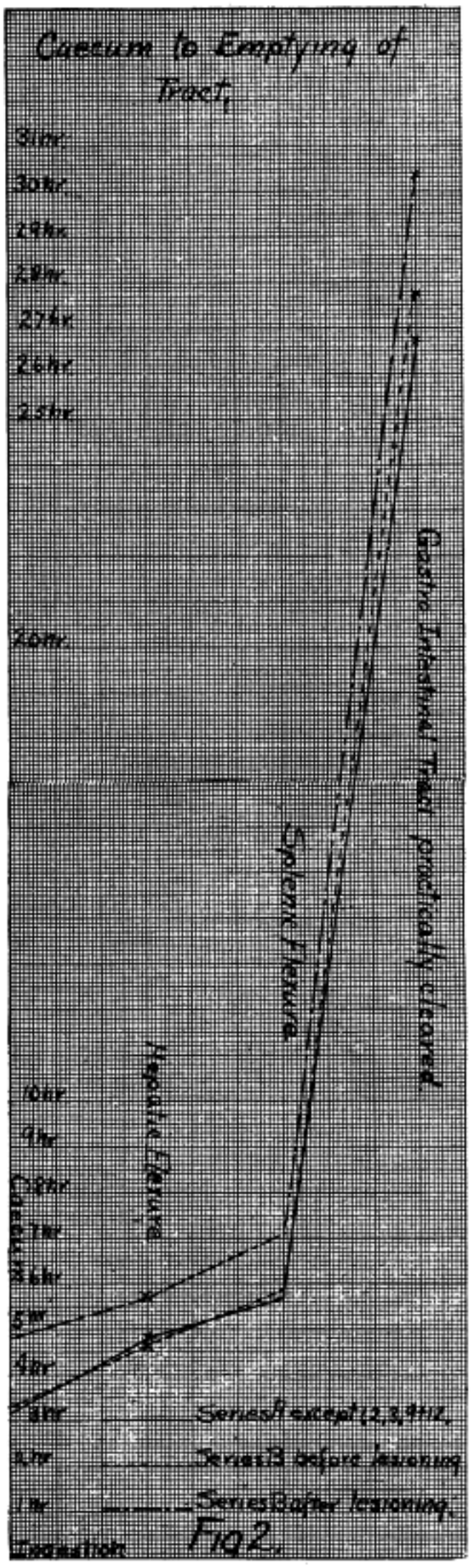
Caecum to Emptying of Tract T_1
31 hr.
30 hr.
29 hr.
28 hr.
27 hr.
26 hr.
25 hr.
24 hr.
Gastric Emptying Time (practically cleared)
Ileocaecal Sphincter Relaxure
Hepatic Flexure
0 hr
9 hr
10 hr
7 hr
6 hr
5 hr
4 hr
3 hr
2 hr
1 hr
Ileum
Series A except (2, 3, 9 hr.
Series B before lessoning
Series C after lessoning.
Fig 2.

Fig. 2

Fig. 2. This is a continuation of Fig. 1 on a smaller scale; necessitated by the thirty-one-hour interval required. The progress from the time that the first barium reached the cecum until the entire tract was practically emptied is shown in this figure. The lines (representing the same groups as in Fig. 1) are more nearly parallel showing that the effects of these dorso-lumbar lesions were more pronounced earlier in the tract.

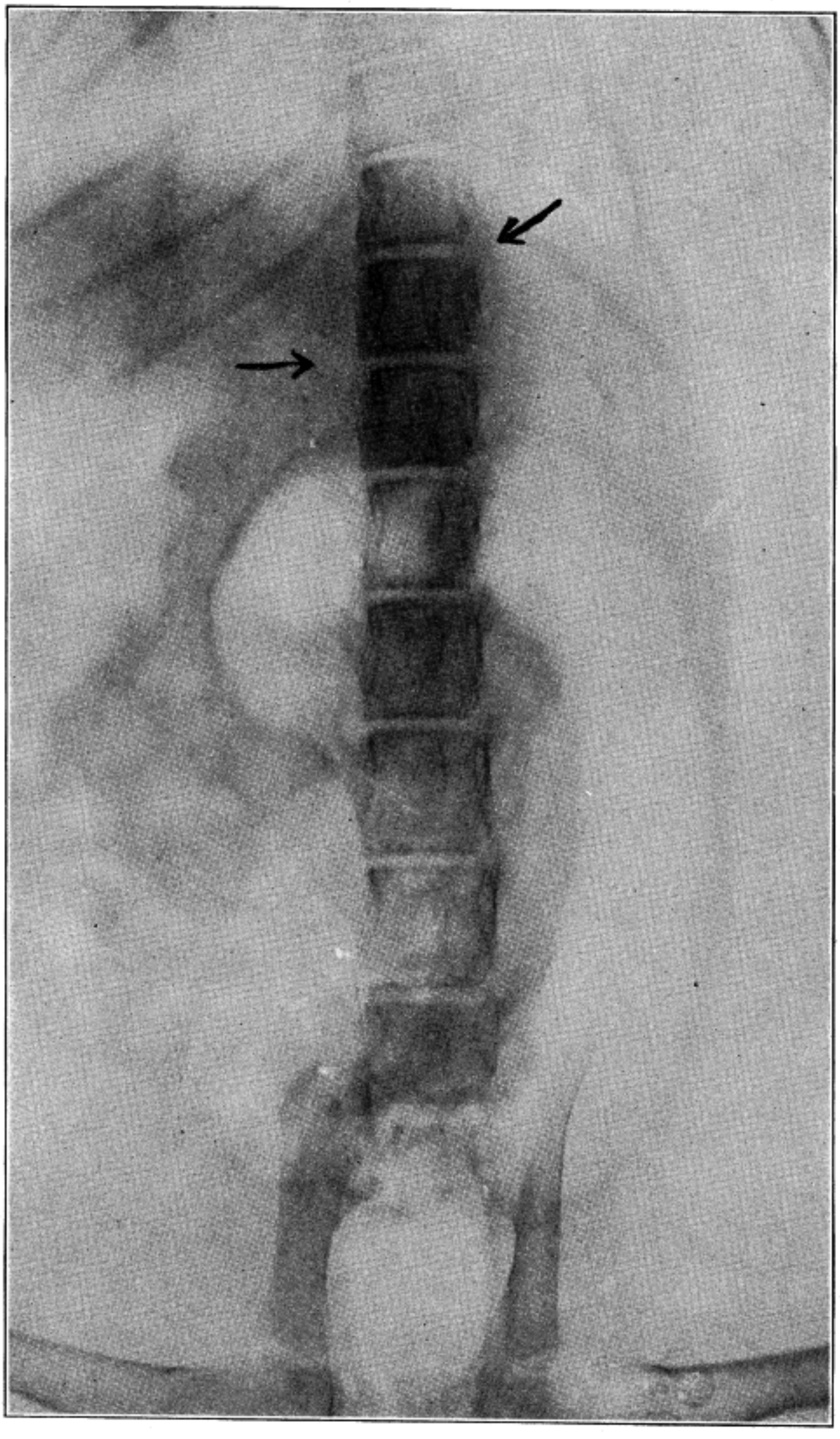

Fig. 3

Fig. 3. This radiograph of cat No. 10 was taken three days after the attempt at lesioning it. There is a diffuse edematous area quite pronounced between the last dorsal vertebra and the first lumbar. Similar but less pronounced area between the first and second lumbar vertebra.

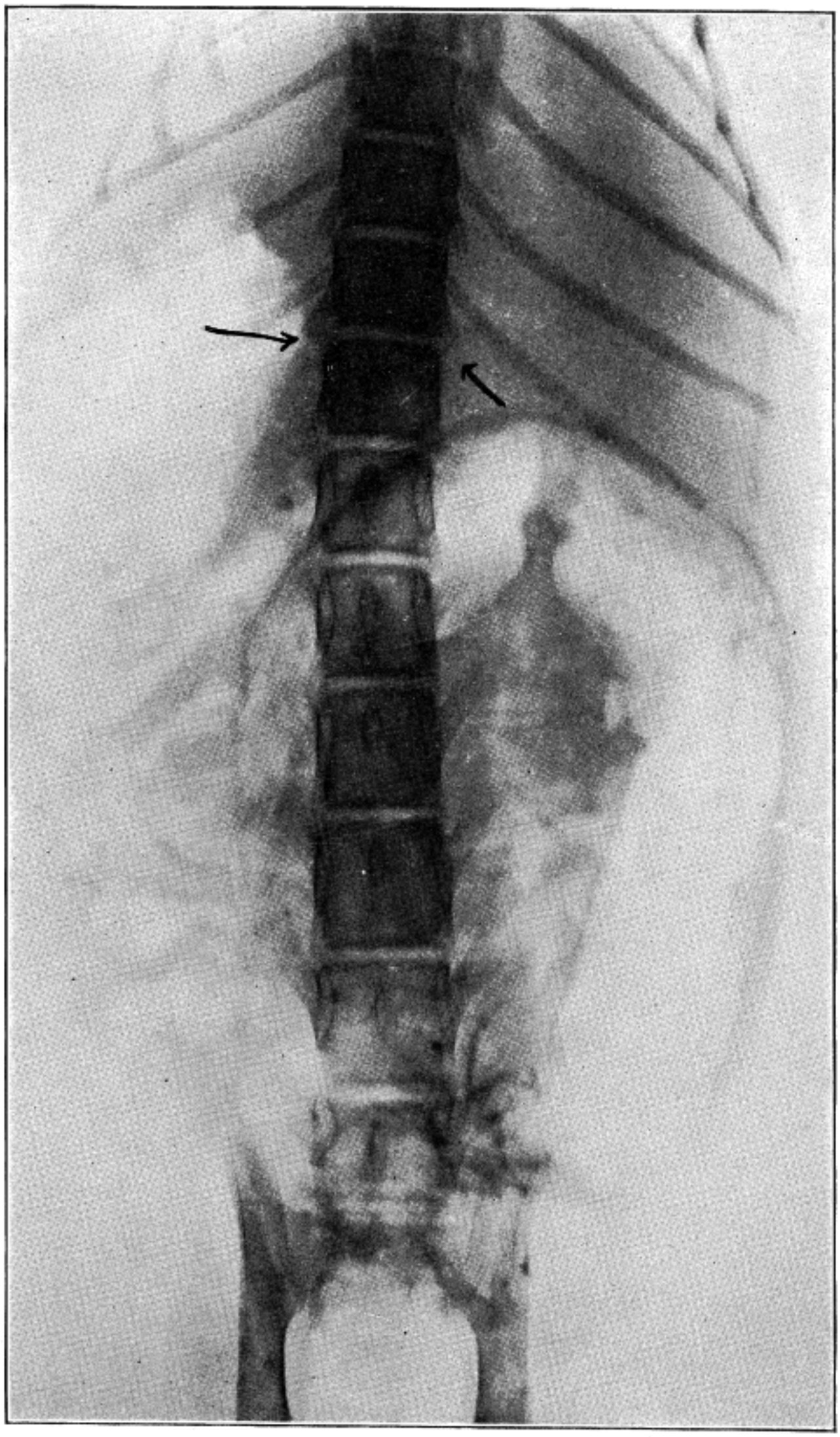

Fig. 4

Fig. 4. This radiograph of cat No. 16 was taken ten days after the process of lesioning was begun. The edematous areas as shown in Fig. 3 are partially absorbed and on their outer borders in the original plate is a faint "fibrous halo."

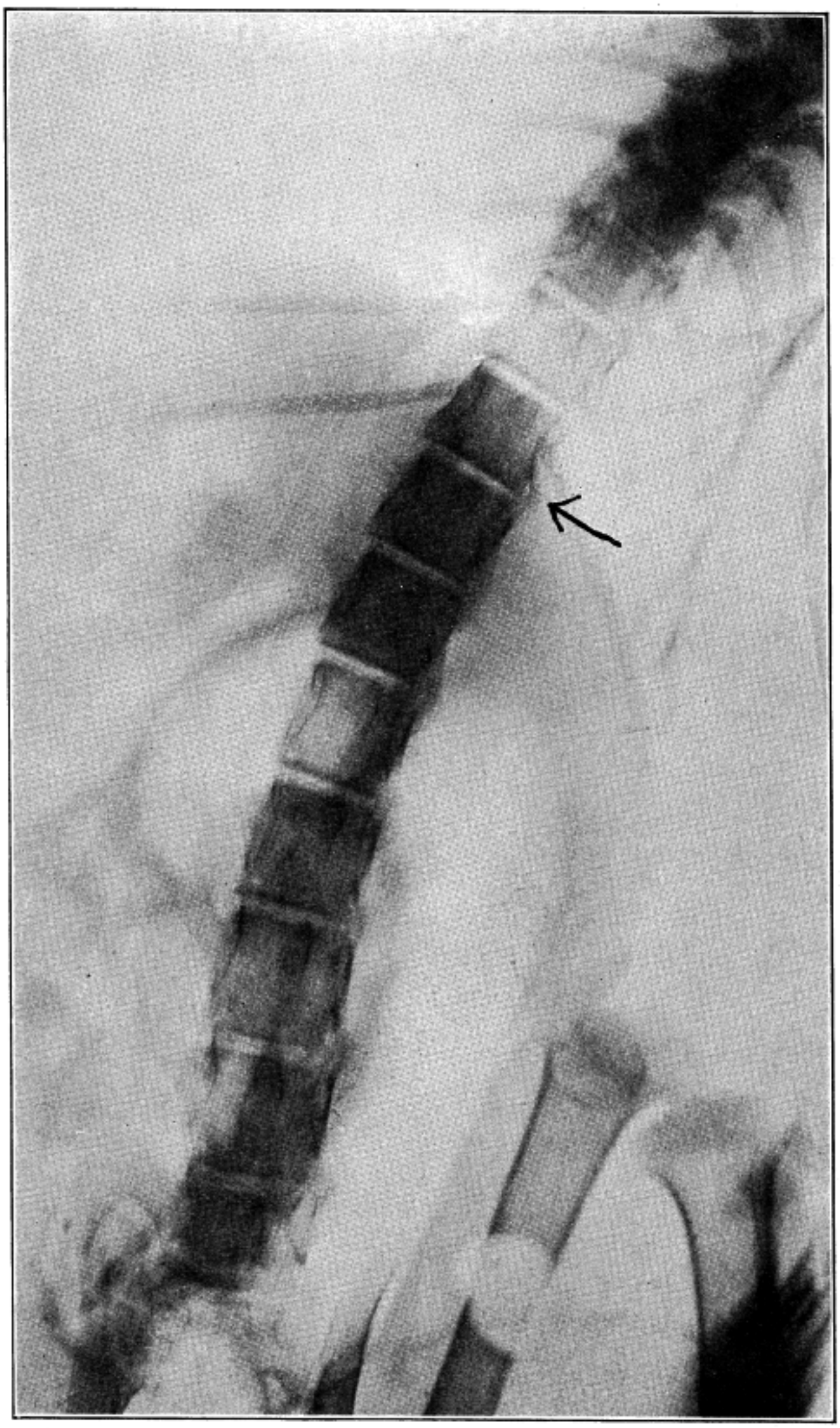

Fig. 5

Fig. 5. This radiograph of cat No. 19, taken ten days after lesioning shows the edematous formation with "fibrous halo" and limitation of motion in the lesioned area to lateral flexion. This rigidity was demonstrated in all other directions under the fluoroscope.

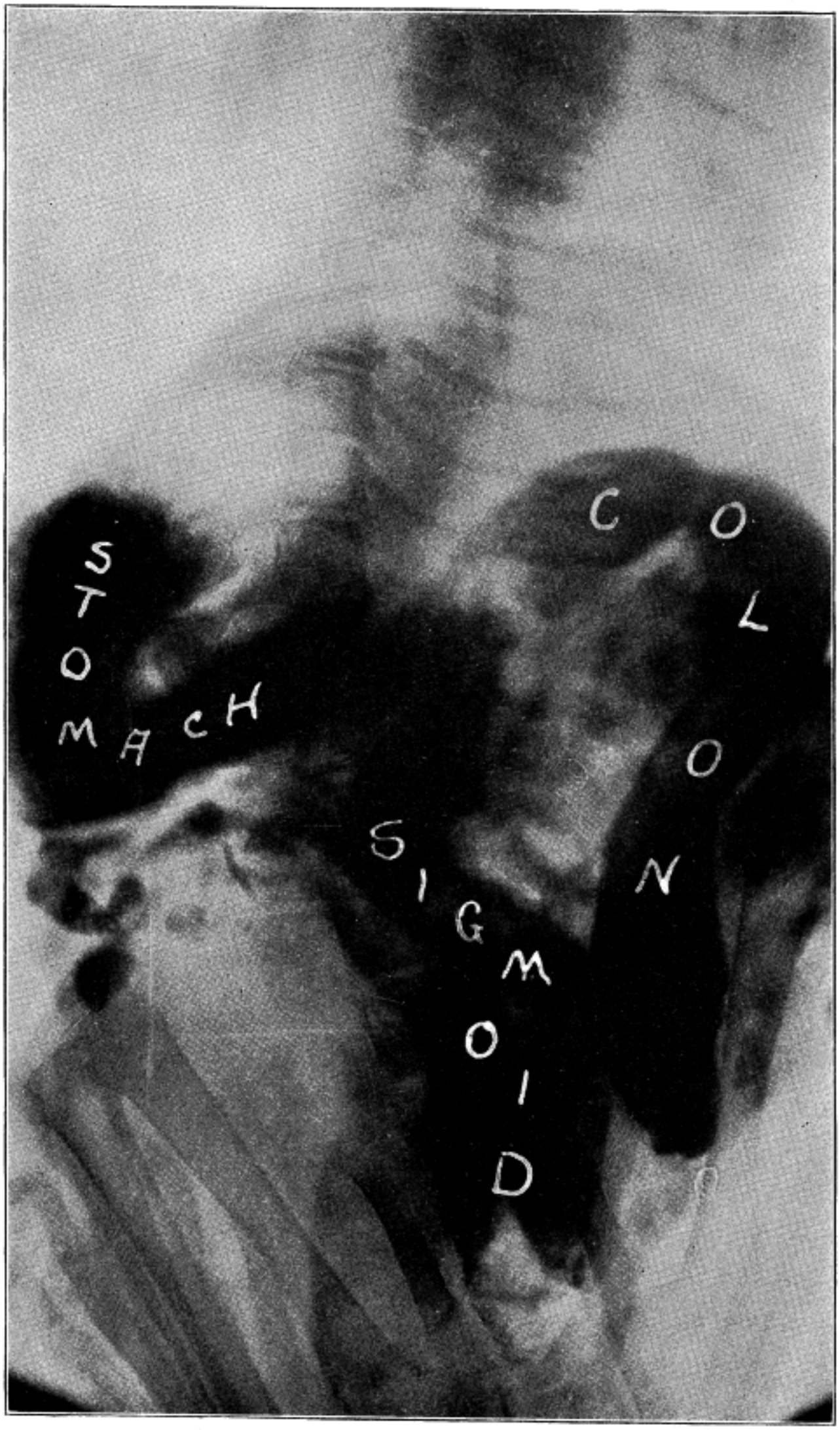

Fig. 6

Fig. 6. Radiograph of cat during the passage of the barium meal after lesioning. Barium is seen ready to leave the tract while the stomach still contains a considerable amount of barium. This cat is not included in the series as later work showed cat infested with intestinal worms.

to avoid lack of uniformity of results, just as is exemplified by Nos. 1, 2 and 3. There are many individuals who are expecting to be hurt during an X-ray examination or are of the so-called "high-strung" or neurotic type. Great care to overcome fear is essential to arrive at the real knowledge of the stomach's behavior. Sometimes if such a condition is coupled with actual structural pathology the symptom-complex will be exaggerated and emphasize the structural condition. These cases must needs be checked under more favorable circumstances. A number of such cases are on file at the Institute and at all roentgen laboratories.

There is a difference of about an hour in the stomach emptying-times before and after lesioning. The food as given was a fairly well-balanced meal, as far as actual fats, proteids and carbohydrates content go, and as the cats were fairly well nourished it must be assumed that that time is sufficient for their stomachs to complete the gastric digestion of such a meal. The effect of the barium itself can be considered practically negligible as barium given in a water suspension passes out of the stomach almost immediately along with the water and passes on out through the tract unchanged itself. The actual time difference is shown to be 4 hrs. 2.7 min. minus 3 hrs. 5.3 min. or 57.4 minutes and for the same animals after lesioning, 4 hrs. 2.7 min. minus 2 hrs. 48.1 minutes or 1 hour 14.6 min. This is too great to be a coincidence.

In the normal animals barium reached the cecum about as the stomach was emptied. In the lesioned series there was an average of about thirty minutes longer time required. The actual interval from the time the first barium reached the duodenum until it was seen in the cecum was 3 hrs. 5.3 minutes in the 22 normal animals; 3 hrs. 8.8 min. in the normal animals which were later lesioned and 4 hrs. 31.3 in the lesioned animals. The passage from the two sets of averages was delayed 1 hr. 26 minutes in the first series and 1 hr. 22.5 minutes in Series B after lesioning. This is the greatest difference in tiine found in the study of the series. This is to be expected from the location of the lesion. (Fig. 6.)

The progress through the ascending colon was faster in the lesioned series than in the normals. This is the only place in the tract that the lesion accelerated the passage. It is quite probable that had there been more animals in the series some information might have been gained on the subject of diarrhoeas accom-

panying lumbar lesions. It is useless to try to draw conclusions as to the relative irritative and inhibitory effects as yet. The progress through the transverse colon was retarded but not in so marked a degree as it was earlier in the tract.

So far this work has shown that a lesion at the dorso-lumbar junction has a marked tendency to retard the progress of food through most of the gastro-intestinal tract, increasing the tendency to stasis although in some parts of the tract probably tending toward diarrhoea. The most pronounced disturbances were manifested in the upper rather than the lower part of the tract.

PROTOCOLS OF LESIONED FEMALE GUINEA PIGS MATED WITH NORMAL MALES.

Avis G. Hoskins, D. O.

On Jan. 23, 1917, a normal male, two normal females, and three lesioned females were put in a cage to begin an experiment on effect of lumbar lesions on pelvic viscera. On Feb. 2 and 16, 1917, five more lesioned females were added to the series. March 3, 1917, five more lesioned females were added and four more females added for controls. These pigs, normal and lesioned with the one normal male, were all kept in the same cage until the work was completed, July 9, 1917. During this time the normal controls experienced normal pregnancies. Each pig was put into a separate cage at about the time for the birth of the young and was left with the babies until it was thought the babies could be weaned, then the mother was put back in the cage to be mated again.

All pigs were kept two weeks for adjustment to their new surroundings and for observation before being added to the series. Three people examined each pig and all agreed that the pigs were normal when added to the series.

The lesioning was done by tapping. They were given about 200 taps every day for three weeks, then every other day for two weeks and twice a week for one week. Lesions were demonstrated by palpation, the diminished spinal flexibility, hypersensitiveness of surrounding tissues, and by the examination of the X-ray plates. (Figs. 7, 8.)

The pigs were all kept in normal condition except as stated in the protocols. No infectious disease appeared among any of the guinea pigs during this time.

No. 1. Brown pig. Black spots on left side and over left eye. Lesioned by tapping, Jan. 23, 1917. Tapping was continued for three weeks every day—then was repeated every other day for two weeks—then twice a week for a week or so, and X-rays were taken every week for the first three weeks. These show bony lesions involving second and third lumbar vertebrae.

April 9, 1917, this pig had a miscarriage. Two fetuses were found which were probably between sixteen and twenty days old.

They were dark purple in color and had likely been dead several days before the miscarriage. Pig died April 13, 1917, and a post-mortem showed the following:

Ovaries: Left ovary contained a few small cysts. Right ovary had large cyst.

Uterus: Right horn contained a fibroid tumor, a little larger than a lima bean.

No. 2. White pig, brown on right side and on left hind quarter. Lesioned by tapping, Feb. 2, 1917. X-ray pictures were taken every week for first three weeks.

March 28, 1917, this pig was taken from the cage, given ether anesthesia and posted to study the lesion and effects of the lesion. A distinct rotation of the second lumbar to the right was found and a lack of motion in all directions was noted.

The viscera appeared normal excepting the following:

Kidneys: Large and filled with blood.

Uterus was congested.

Ovaries appeared normal.

No. 3. Dark red pig, white right front paw, white spot in forehead.

This pig was pregnant when purchased and gave birth to two babies the next day. The pregnancy seemed to be normal and she was left with the babies until Feb. 16, 1917, when she was put with the lesioned series, and lesioned by tapping in area of second and third lumbar.

The night of July 2, 1917, she gave birth to two babies. Both were males and were found dead in the cage the morning of July 3, 1917. (This was her first pregnancy since the normal pregnancy the first of February, 1917.) The pig became pregnant about May 10, 1917, and pregnancy seemed normal until birth.

Pig A had been born alive, and was of normal size. It was still damp when found and the amnion still covered the hind part of the body and front paws.

The placenta was very dark purple with hard tumor mass in center.

The lungs floated in water.

The viscera were normal.

There was slight hemorrhage from the circle of Willis with blood over base of brain and in both orbits around eye ball.

Pig B. This little pig was much larger than normal, at least 1/3 larger than his brother. He was enveloped in a caul, and had a double placenta, completely separated, with two cords running to a single umbilicus. One placenta was of normal size, and the other was about 1/3 less than the normal size.

This pig was born dead; the lungs sank in water.

The viscera were anemic in appearance and showed no duplication of parts.

The stomach, bladder and thorax all contained a bloody fluid, the stomach and bladder being quite distended with this fluid.

There was hemorrhage at the base of the brain extending downward toward the medulla and cord.

The mother guinea pig seemed ill after the birth of the babies. She ate very little, and lay quiet most of the time. The fur was rough, and she grew worse from day to day.

The lesions were palpable, and the lumbar region extremely hypersensitive. The X-ray plate shows the lesions. (Fig. 8.)

The mother was allowed to live until July 12, 1917, when she was given chloroform anesthesia and posted. The post showed large bloody kidneys and large spotted liver.

The ovaries and the right horn of the uterus were more congested than is usual ten days after the birth of young. The walls of the right horn were relaxed and atonic, resembling the normal uterus during the first week after birth of young. The left horn contained a tumor mass about as large as a pea. The veins and arteries to the uterine wall underlying this mass were very large and full of blood, resembling the same vessels during pregnancy. The uterine wall was thickened for a considerable distance around the edge of the tumor; the longitudinal diameter of the thickening was about an inch; the entire circumference of the uterine horn was involved in the thickening. The entire mass was excised, and about one-third cut off for immediate examination; the rest was placed in Zenker's fluid and mounted in paraffine for cutting.

For description of the sections see page 134.

No. 4. Small brown and black pig.

Lesioned by tapping Feb. 2, 1917. On March 25, 1917, this pig was found in cage dead and badly mutilated. It had been killed by a wild rat. This pig had shown no signs of pregnancy up until time of death.

No. 6. Lesioned by tapping March 3, 1917. Black pig with white spot on breast. March 28, 1917, pig was found dead and body had been partly eaten. The pig had been killed by wild rats. No signs of pregnancy were found.

No. 7. White pig, with brown right shoulder and leg. Lesioned March 3, 1917, in second and third lumbar by tapping. March 20, 1917, the pig killed during night by wild rats. No signs of pregnancy were found.

All the guinea pigs were then placed in cages raised from the floor.

No. 5. Black and white spotted female.

Lesioned Feb. 16, 1917, by tapping. Lesion in second and third lumbar as shown by X-ray.

This pig had had a normal pregnancy, giving birth to two pigs Feb. 9, 1917. The wild rats got the babies.

June 7, 1917, chloroform anesthesia was given and a post was done before allowing the pregnancy to terminate in a normal manner for the sake of securing photographs. (Fig. 9.)

The pig appeared in good health. When the abdomen was opened and viscera examined the liver was spotted over its entire surface. The kidneys were large and full of blood.

The uterus was very dark purple in color, very friable, and contained one fetus of enormous size with a single placenta attached which was somewhat abnormal in shape and color. The ovaries appeared normal.

On the inner wall of the uterus about two inches from the placenta were noticed two dark, hard, pale green tumors about the size of a lima bean. (Fig. 9.) These were coin-shaped, five-sixteenths inch in diameter, elevated about one-sixteenth inch above uterine surface. Each tumor was surrounded by an indistinct, sticky, greenish exudate. An area of similar exudate was found upon the uterine wall below the attachment of the placenta. The exudate consisted of pus and disintegrated blood. A part of one of the tumors was teased apart for microscopic study and was found to contain chorionic villi. They are probably the remains of the placentas of three fetuses that had died very early in the pregnancy.

The viscera of the fetus were examined and appeared normal.

This fetus had evidently been carried for some time longer than the length of a normal gestation period, as shown by the

X-ray. (Fig. 11.) The X-ray taken of this fetus was compared with X-rays taken of normal fetuses at the end of normal gestation period, and the following differences were noticed: The length of the body from occiput to coccyx was 5¾ inches, as compared with the 3¾ inches of a normal fetus. (Fig. 12.) The femur was twice the length of the normal fetus, and there was advancement of ossification of the knees, ankles, pelvis, spine, shoulder, elbow, wrists, head and scapula. The fontanels were practically closed.

For the sake of comparison, a photograph was taken of a normal full term guinea pig. The uterus, placentas, and baby pigs were normal in every recognizable respect. Labor was just beginning when the chloroform was administered. (Fig. 10.)

No. 8. White female with brown hind quarters.

Lesioned March 3, 1917, by tapping. Lesion involved from 1st to 3rd lumbar.

July 8, 1917, a post was done under chloroform anesthesia. Three fetuses (apparently about a month's development) were found, all in the right tube. They all appeared normal; two placentas were round and normal, one was reniform but not apparently diseased.

Mother pig seemed normal excepting for two white papillary projections from the right and one from the left ovary. Other viscera were normal. The right ovary was put in Zenker's fluid for examination later.

No. 9. White head and shoulders, rest of body solid red. Lesion produced Feb. 2, 1917, included area from first to third lumbar.

July 8, 1917, a post was done under chloroform anesthesia. Two fetuses were found in each uterine horn. Both appeared to be normal, with normal placentas. The period of gestation did not exceed 16 to 20 days.

No. 10. White pig, black spots on right side of face, including right ear. Lesioned in upper lumbar by tapping Feb. 16, 1917. On April 14, 1917, this pig was injured by wild rat and a pus pocket developed in the belly wall. Pig died April 17, 1917, from septicemia. No pregnancy occurred.

No. 11. Light brown and white spotted pig.

Lesioned Feb. 2 in upper lumbar region by tapping.

On May 10 this pig gave birth to two babies. These were found in the cage still wet and both dead. A post of the two babies showed nothing abnormal in the viscera, the lungs floated in water so they had been born alive and had died very shortly after birth. They were both under size and thin, but perfectly developed.

On July 5 this pig was given chloroform anesthesia and posted. She was pregnant, the pregnancy being of about 25 days' duration. Two fetuses were found in the right horn and one in the left. All were alive and apparently normal.

The uterine wall seemed to be thinned and the placentas stood up quite prominently. Uterus was dark purple in color.

The left ovary appeared normal, but the right was badly congested and a cyst was found about the size of a half-pea.

Both kidneys were large and very full of blood.

A vaginal discharge was noticed.

No. 12. Brown pig with black face. Lesioned Feb. 3, by tapping. Died June 15, of septicemia from two dead fetuses of about 25 days gestation.

Controls.

The six normal controls of this series all became pregnant shortly after being mated. The birth of the young was normal and they were each left with their respective families until the young could be safely weaned when they were placed in cage from which they came.

A post-mortem was done of all the above pigs on July 6, 7 and 8 for the sake of photographs. They were all pregnant. Two of them were in the latter part of the pregnancy (Fig. 10), the other four at what seemed to be about a 25 or 30-day pregnancy. (Fig. 14.)

The maternal viscera of all appeared normal. The placentas were of normal size, shape and color.

The young seemed to be in good condition and with perfectly normal viscera. See table on page 69.

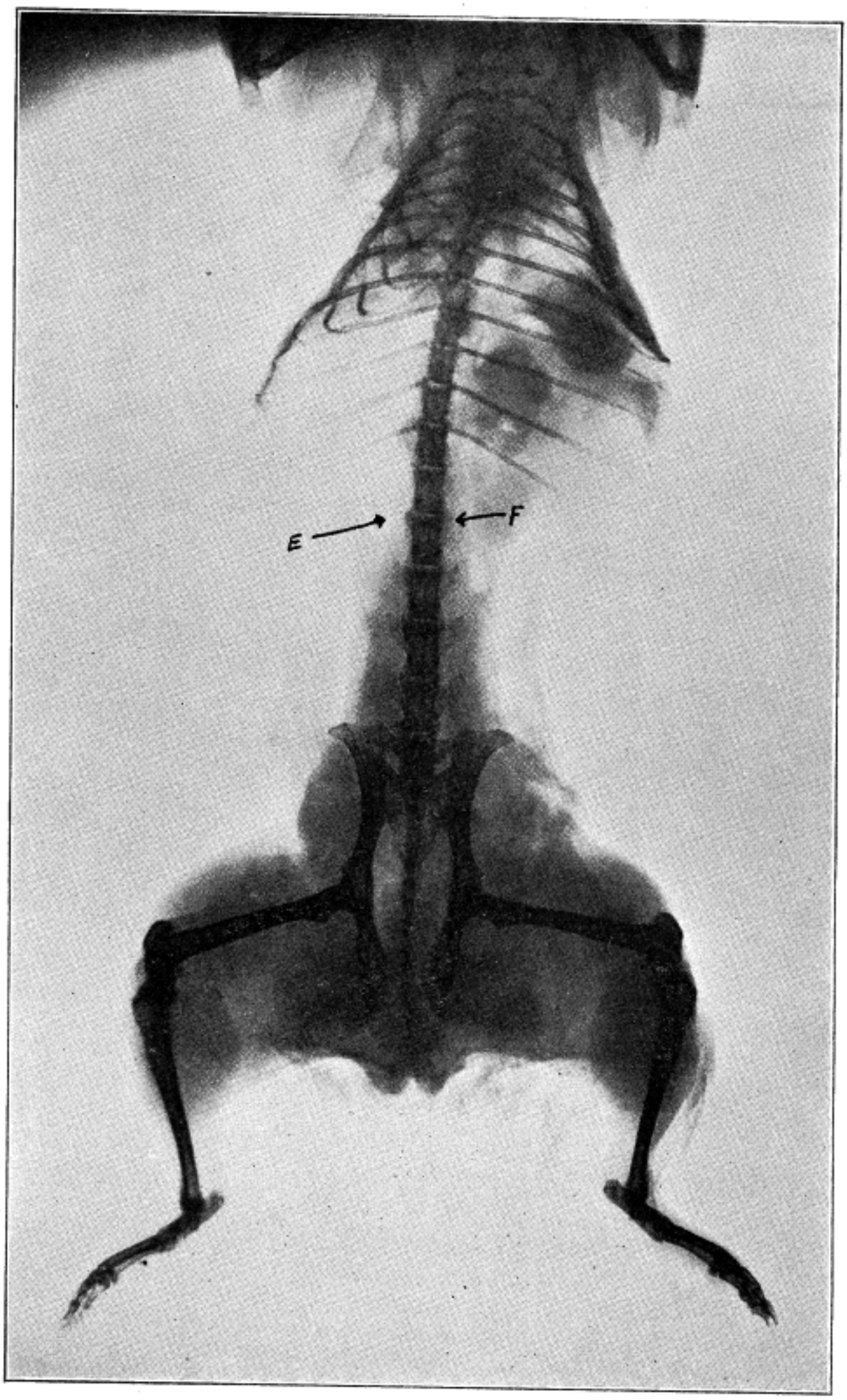

Fig. 7

Fig. 7. X-ray plate of guinea pig, showing milder degree of lumbar lesion, associated with slight edema and fibrosis.

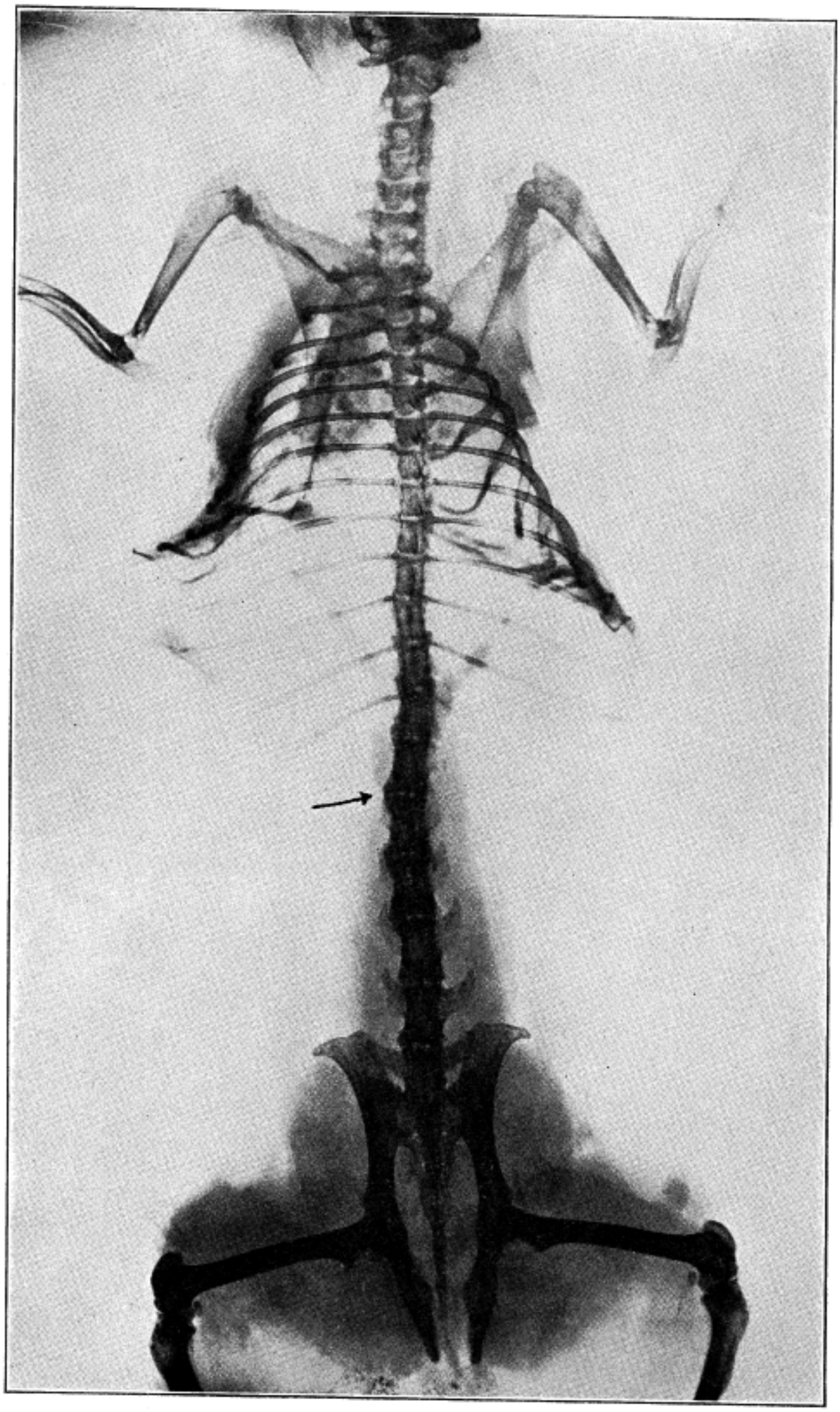

Fig. 8

Fig. 8. Radiograph of guinea pig No. 3. The arrow shows the primary lesion, produced by tapping, and shown in earlier X-ray plates. The lesions above this are of later development. This is the pig in which the malignant uterine tumor was found. (See also Figs. 37, 38, 39.)

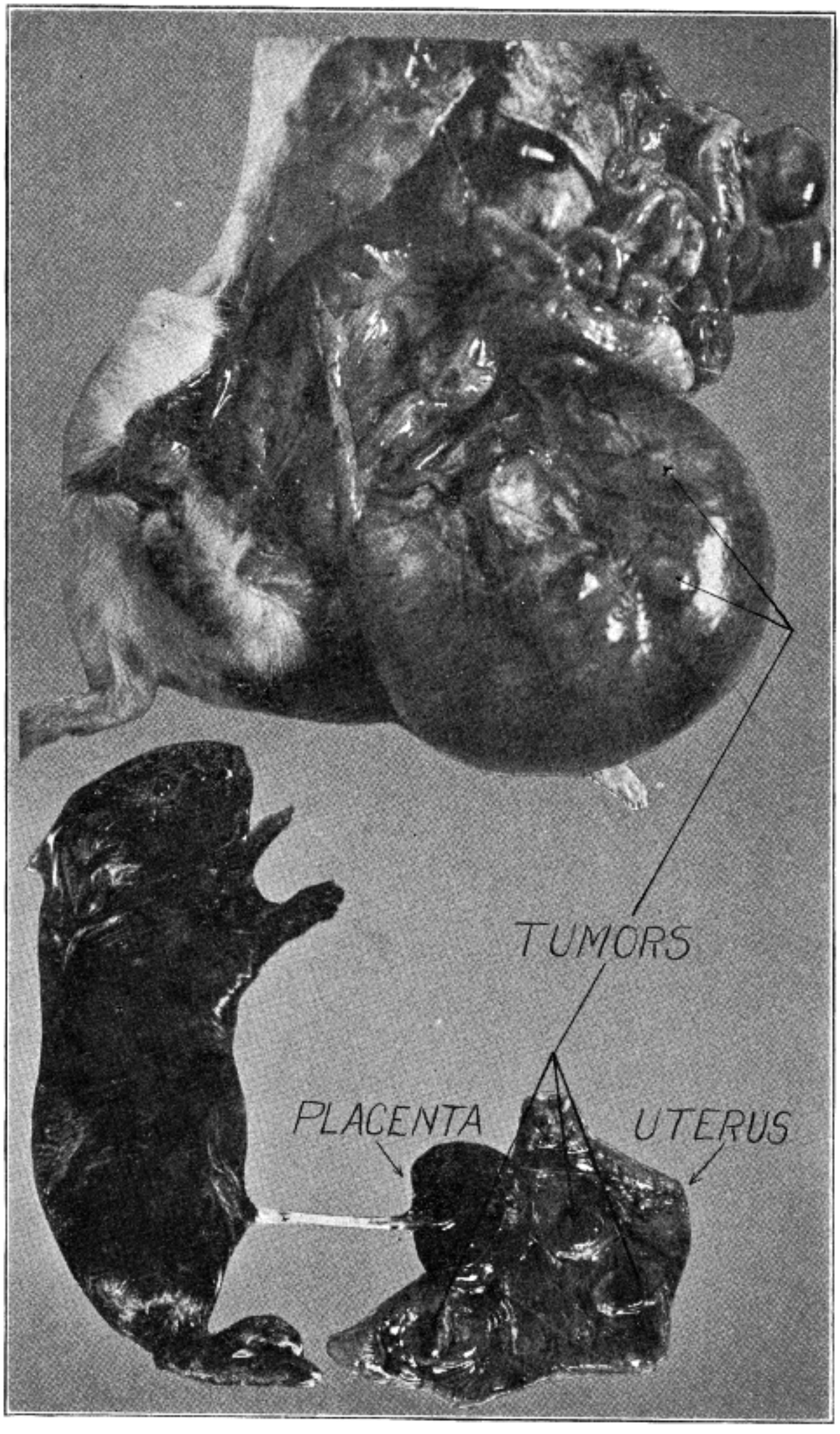

Fig. 9

Fig. 9. Guinea pig No. 5. The upper photograph shows the pig after the abdomen was opened, with the uterus drawn to the left side. The right horn of the uterus is empty, and is slightly congested, with relaxed walls, as is usual in the empty horn of guinea pigs in which a single pregnancy occurs. The lines indicate the positions of two of the discoid tumors. The location of the third is not visible upon the outer wall of the uterus from this angle. The size of the uterus should be compared with the normal uterus at full term in Fig. 10.

The lower photograph shows the fetus and the inner wall of the uterus. The cord was not broken. The pig was dead when it was removed from the uterus. The lines indicate the positions of the tumors. The blood supply to the two tumors shown in the upper photograph, in which the placental origin of the tumors is clearly evident, was not so profuse as is the case in malignant neoplasms.

See also Figs. 11, 12, 13.

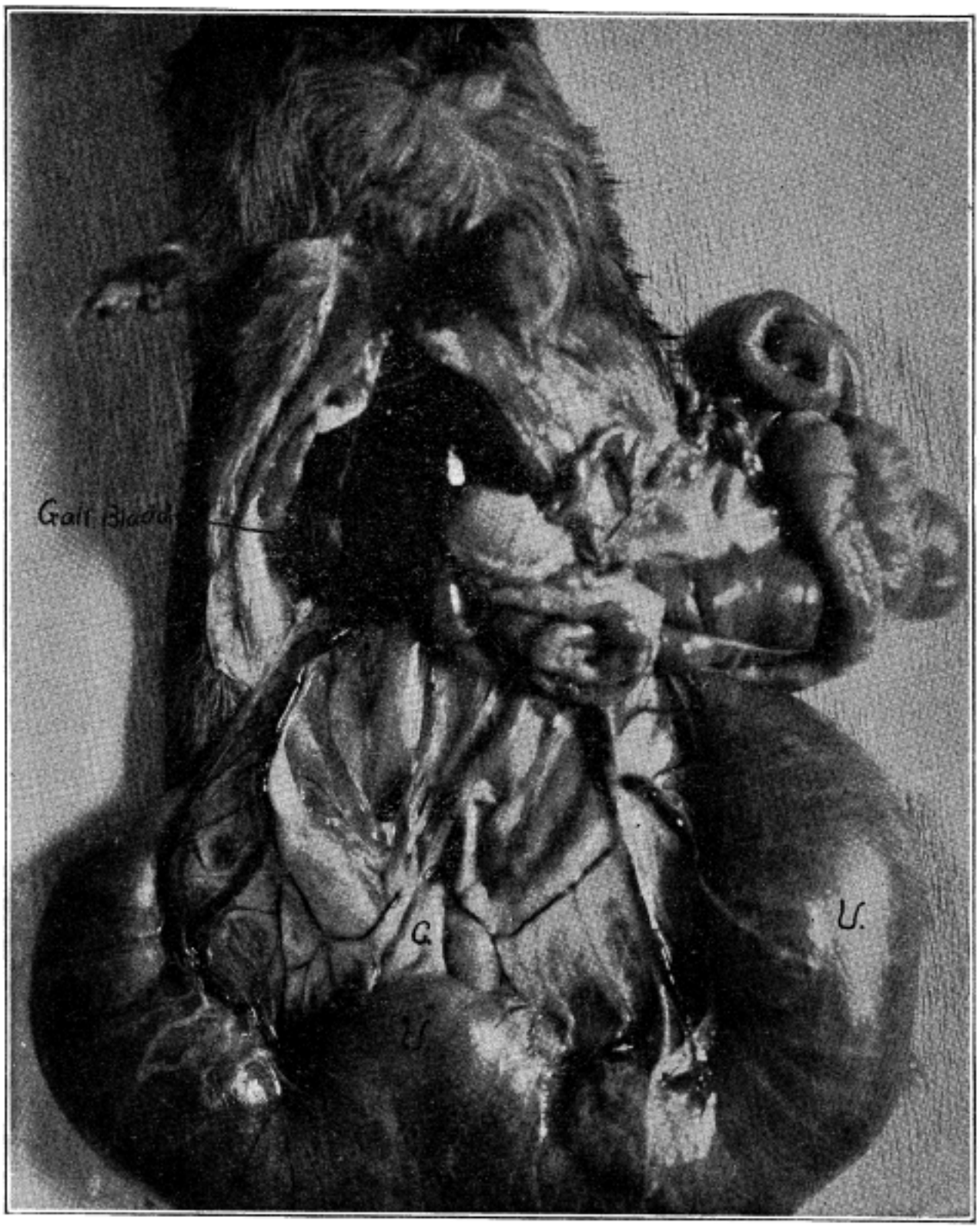

Fig. 10

Fig. 10. Normal, full term pregnant guinea pig.
U. Uterus, with a fetus in each of its two horns.
C. Cervix of uterus.
The gall bladder is shown and named.

The uterus is drawn downward in order that the size may be seen, and also the anatomical relations.

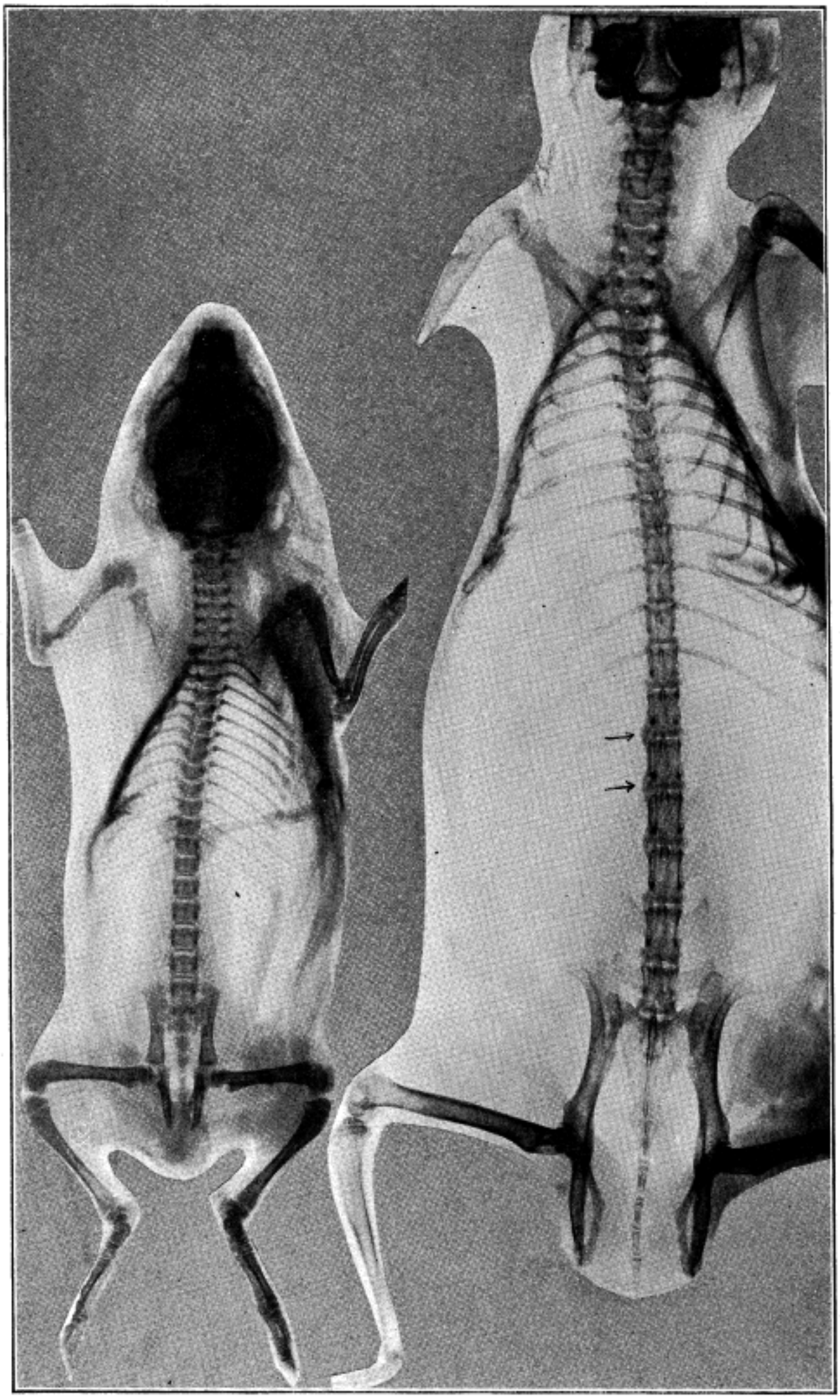

Fig. 11

Fig. 11. X-ray plate of guinea pig No. 5 and the fetus removed from the uterus. The two were taken upon the same plate, at the same time, but the exposure was slightly less for the fetus than for the mother, in order that the bones might be clearly shown. The disproportionate sizes of mother and fetus are evident. The arrows indicate the vertebral lesions. See also Figs. 9, 13.

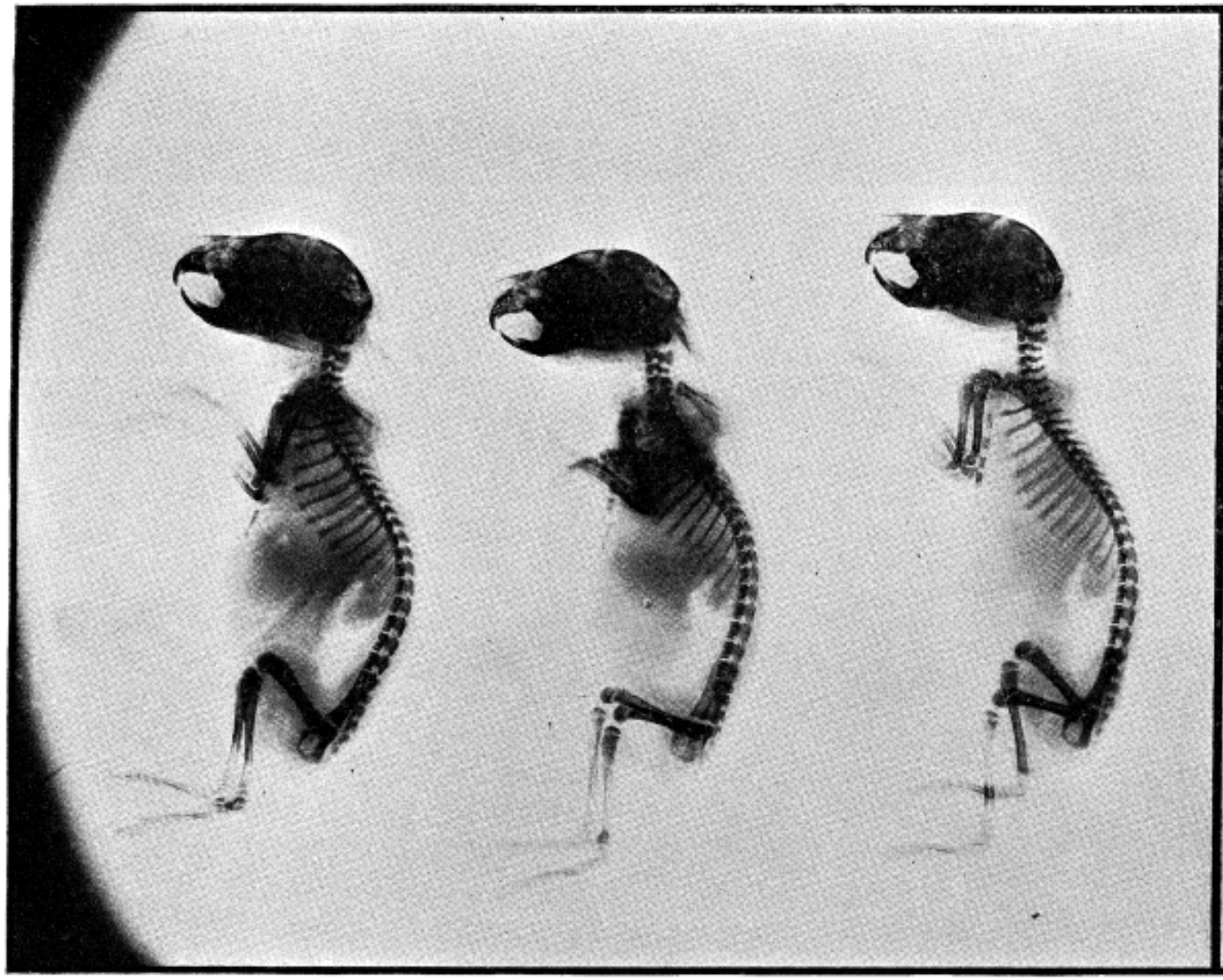

Fig. 12

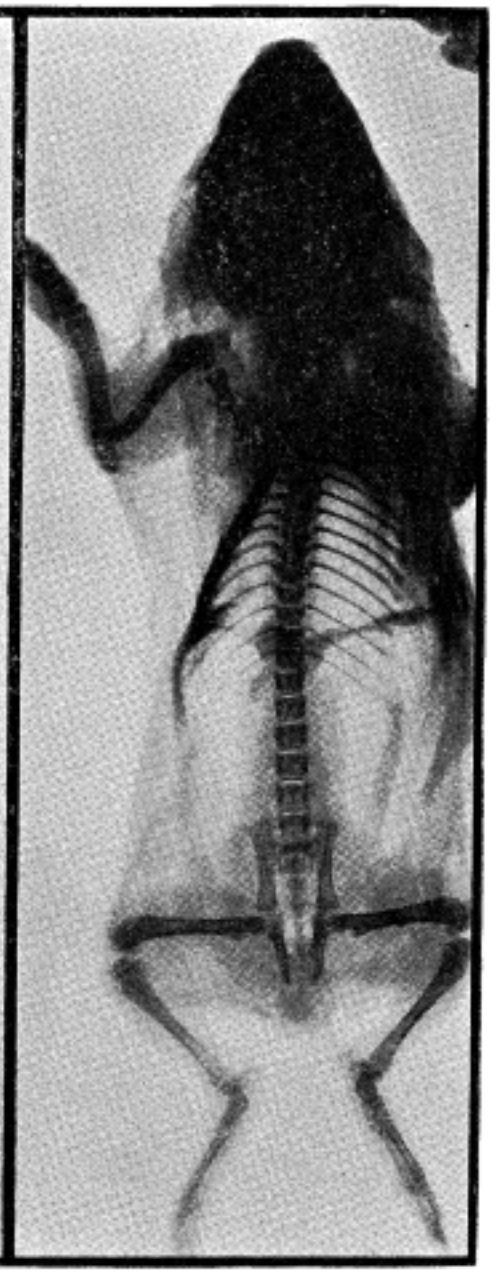

Fig. 13

Figs. 12, 13. X-ray plates showing three normal full-term guinea pig fetuses and the abnormally large guinea pig fetus shown in Figs. 9, 11.

Note the differences in size and in the ossification of the bones.

Fig. 14

Fig. 14. Normal guinea pig of 25-30 days' gestation. (The gestation period of the guinea pig is 57-60 days.) One of the pigs removed from this uterus is shown in the lower part of the plate.

The uterus was drawn downward and the intestines drawn upward, so as to show the anatomical relations. Two fetuses occupy each horn of the uterus—four in all. The attachment of the placentas is shown by the swollen areas. The two horns unite just above the uterine cervix.

The rectum is separated from the cervix by the pouch of Douglas. The right ovary can be seen lying with the parovarium just above the upper extremity of the right horn of the uterus.

PROTOCOLS OF LESIONED FEMALE WHITE RATS MATED WITH NORMAL MALES

Nov. 12th, 1915. Three adult female white rats were taken from a group of normal individuals. They were examined and no evidences of any pathological condition were discovered. The spinal column and neighboring tissues were carefully palpated. After this examination gave no evidence of spinal lesions already present a lesion was produced in each. The lumbar spine was held firmly between the thumb and fingers of both hands and tension was thrown upon the transverse process of the second lumbar vertebra. Tension was thrown upon these bones with the pressure exerted in such a manner as to rotate the vertebra upon an axis running through the opposite articular surfaces. This process was repeated until a change in the spinal tissues became evident on palpation. Examinations of the spinal column were made by three persons and in each case they agreed upon the diagnosis without previous consultation. The spinous processes of the affected vertebrae were approximated and a slightly anterior condition of the bodies could be palpated through the abdomen. No anesthetic was necessary since the operation did not appear to be at all painful. When the animals were placed in cages they seemed to walk with some difficulty for a few minutes, probably as a result of being held for some time. This effect passed off in a very few minutes and the animals seemed to be as well as ever. A normal male was placed in the cage with the three females. The cage was marked "Lesioned Nov. 12."

Nov. 15, 1915. Thirteen females were lesioned after the method employed Nov. 12. The lesion which was planned is a rotation of the lumbar region so that the spinous process of the third lumbar vertebra is turned toward the right. In such small animals the exact localization of the lesion is very difficult. One female seemed to be partly paralyzed in the hind quarters for three days. Three families each consisting of three of these lesioned females with one normal male were formed and were placed in cages. The cages were marked "Lesioned Nov. 15."

Nov. 22, 1915. Lesioned rats were examined. In about half of these the lesion was found present. Those which showed

no evidence of lesion were subjected to a repetition of the manipulation described for Nov. 12, 1915.

Nov. 29, 1915. The rats were examined. In a few the lesion had become corrected; these were lesioned again.

Dec. 4, 1915. A new family of three lesioned females with one normal male was formed in the same manner. The cage was marked "Lesioned Dec. 4."

Dec. 14, 1915. The rats were examined. The lesions persisted in all but three. These were lesioned.

Dec. 30, 1915. Three female white rats, two of which appeared to be pregnant, were lesioned and placed in a cage with a normal male. Two females which show no symptoms of pregnancy were lesioned, and were placed in cage with normal male. The cage was marked "Smith." Rats previously lesioned were examined.

Jan. 13, 1916. The rats were examined, and lesions produced in a few whose lesions had not persisted.

Jan. 20, 1916. The rats were examined; every one was found with lesion. A new family was formed in the same way as before. The cage was marked "Lesioned Jan. 20, 1916."

Jan. 27, 1916. The rats were examined. Lesions persisted in all.

Feb. 3, 1916. Two lesioned rats in the "Nov. 15, 1915," cage were found dead. Lesions were present in both. Autopsy shows pneumonia to be cause of death. Ovaries and tubes congested in one. Ovaries cystic in both. The ileo-cecal region shows adhesions and exudate.

Feb. 10, 1916. One lesioned female rat in the "Dec. 4" cage was found dead. Autopsy shows pneumonia to be cause of death. Ovaries cystic, uterus congested, kidneys large and full of blood, ileo-cecal region shows adhesions and exudate. Lesions persist in all others. Female from normal cage showing no signs of pregnancy was lesioned to take the place of dead rat. All cages emptied, scrubbed and disinfected.

Feb. 17. Rats were examined for lesions and one, the last lesioned, found normal. The lesion was again produced as before.

March 12. The lesions were found present in all rats. One rat (cage marked "Smith") bore five young and died after few days. Cause of death could not be found. One placenta was

adherent. The kidneys were large and red. The baby rats were eaten by the older rats.

March 23, March 30, April 6. The usual examinations for lesions were made.

April 13. One rat in cage "Smith" bore four young. She and all four babies died before morning. No cause of death could be found. Lungs were normal. Ovaries were congested and cystic; kidneys were large and full of blood. Another normal rat was lesioned and placed in same cage.

April 20, April 27, May 4. The usual examinations were made.

May 17, 1916. The usual examinations were made.

May 17, 1916. The usual examinations were made. One rat, lesioned Dec. 4, 1915, appeared ill. Hyersensitiveness and muscular tension were present throughout the lumbar region. The vertebral lesion was not recognizable.

May 19, 1916. White rat, female, lesioned Dec. 4, 1915. found dead this morning. Autopsy shows probable cause of death to be a perforating ulcer in the ascending colon, about one-half inch in length, about the ileo-cecal valve. Another ulcer in the ileum one-half inch above the valve did not penetrate.

Both ovaries and all the lymphoid and cellular tissues around the ovaries and uterus were congested. The lymphoid tissue appears decidedly increased in amount. This congestion is most apparent upon the right side. The left ovary shows an area which is whitish and appears purulent. Both ovaries and the uterous were removed and placed in Zenker's fluid, then were cut in paraffin.

The bladder and other tissues which lie within the true pelvis appear normal. A single cyst was found upon the lower edge of the right lobe of the liver. This contains a small tape worm. No gross changes appeared in the spinal cord. All other organs appear normal.

May 26, June 1, June 8, June 15. The usual examinations were made.

June 22, 1916. One white rat only appeared to have become pregnant after the lumbar lesions had been produced in the female rats, Nov. 15, 1915. This rat now appeared to be about at full term and was placed alone in a cage. She appeared to be very sick, moped and the hair was rough. She entered the

box which had been placed in her cage for a nest, and ate no food. The lesion in the lumbar region is palpable, and this region is hypersensitive.

June 26, 1916. The rat which had been isolated since June 22, on account of apparent pregnancy, was found dead this morning. The lumbar lesion was found on palpation. The vagina was dilated and moist, as if in labor. The abdomen was soft and as large as full term pregnancy.

The abdomen was opened, and was found partly filled with blood, partly coagulated and partly thin and watery, and very dark in color. Both horns of the uterus were empty, but their veins were greatly dilated. The ovaries were found with some difficulty; both were slightly congested, but were otherwise normal in appearance.

The right ovary lay upon the wall of a great cyst which filled almost the entire abdomen, and to which the great size of the abdomen was evidently due. The cyst occupied the position of the parovarium. The upper and posterior aspect of this cyst presented a large ulcer-like opening, which was closely related to an area in the cecum which appeared to be about to break through. The edges of the ulcer were very friable. The wall of the cyst was dense and thick and very heavily supplied with dilated veins and arteries; these resembled the vessels of the normally pregnant uterus.

The cyst was filled with a jelly-like material, slightly blood-stained, and partially separated into compartments by trabeculae of connective tissue. The cyst was lined with a spongy layer which stripped away from the cyst wall easily. On microscopic examination this lining is found to bear a marked resemblance to placental tissue. No embryo could be found, although the cyst-contents were thoroughly examined.

The cyst was attached to the right kidney, on a line just below the hilum. The part of the kidney thus included within the cyst had become completely eroded, so that the cyst lining was attached to this eroded surface.

The ileo-cecal region was involved in a mass of adhesions, with cysts and enlarged lymphatic glands. An ulcer in the cecum involved both the anterior and the posterior wall, but this had not yet penetrated the wall. It appeared to be intimately associated with the ulcer in the wall of the cyst.

A small tapeworm occupied one of the cysts around the cecal region. Two fairly large tapeworms occupied cysts in the liver.

The stomach and intestines were completely empty. The heart had stopped in systole, and presented a rugged appearance. The lungs, thyroid, suprarenals, digestive tract, and other viscera not mentioned, all appeared normal.

The cause of death appears to be the rupture of the cyst at the ulcerated area. The diagnosis of extra-uterine pregnancy is probably correct.

July 1, 1916. A rat of the group "Lesioned Nov. 15, 1915," has been ill for several days. Ether anesthesia was given and the rat died. Autopsy immediately. The lungs were collapsed and the pleural cavity contains about two cubic centimeters of partly coagulated blood, but less than was present in the pleural cavity of the control rat which died the same day of pneumonia. The lungs were sprinkled with small hemorrhagic points—ecchymoses. Death was due to pneumonia.

Both ovaries were slightly congested, with small, yellowish areas, much larger than the usual corpus luteum of the rat. The horns of the uterus present a number of ecchymotic areas, resembling those of the lungs. (This condition is not found in non-lesioned rats with pneumonia.)

The ileo-cecal region presented evidences of old inflammatory states. Adhesions and partly-organized exudate occupied the abdominal cavity around the cecum. Enlarged lymph nodes were abundant around this area.

Later in the day another rat was found dead in the same cage. The pleural cavity was found partly full of partly coagulated blood, and death was probably due to pneumonia. The lungs were very pale, except for a few ecchymoses. The uterus and ovaries were congested and the ovaries slightly cystic. The stomach and intestines contained the normal amount of food remnants. The heart ceased in diastole. The kidneys were large and red. Other viscera appeared normal.

July 13, 1916. A female white rat from cage marked "Lesioned Nov. 15," appears to be in reasonably good health, except for slight hyperemia of the skin and loss of fur over the right shoulder.

The lesion involving second and third lumbar was recognizable on palpation.

Under ether anesthesia, abdomen and thorax were opened. One large tapeworm was found in the liver; both ovaries and uterus was intensely congested; (Fig. 16, 17) the ileo-cecal region was involved in large mass of adhesions and cysts. The kidneys appeared congested. Other viscera were normal.

All the remaining lesioned rats were moved into new cages without markings except the word "Lesioned."

During July and August the remaining rats were examined at intervals of ten days or two weeks. The lesions remained present. The lesioned animals bore no young, while the females in the control cages bore young normally.

Oct. 15. Eight females were lesioned and placed in families.

Oct. 31. Two more females were lesioned as before, and added to the group. They were placed in a cage with one normal male.

Nov. 14. Two more females lesioned and added to group. They were placed in cage with one normal male.

Nov. 21, Nov. 28. The usual examinations were made.

Dec. 5. The usual examinations were made. One male died of pneumonia. Autopsy showed characteristic lung changes, all other viscera normal.

Dec. 8. Female white rat died during the night. No pulmonary congestion. Fur and fat normal. Liver contained two worm cysts not larger than are often found in rats apparently normal and healthy. These cannot be cause of death. Both horns of uterus congested; left cystic; right ovary and horn of tube were drawn into mass of adhesions including cecum and adjacent intestines, all adherent to abdominal wall and including the skin of the abdomen. This abdominal condition seems to have been the cause of death, and it probably resulted from fighting. This rat was omitted from the table.

Dec. 10. Another female found dead. Lungs showed pneumonia, and this appeared to be cause of death. Stomach contained normal food remnants. The colon was distended. The kidneys were congested. The liver contained three worm cysts. The uterus and ovaries were congested. Other viscera were normal in appearance.

Dec. 14. The prevalence of pneumonia among the animals led to the immediate death of all, followed by careful disinfection of the animal quarters. Six lesioned females remained;

these were anesthetized and killed. No indication of pregnancy was found. In each case there was congestion of the ovaries. In two the left side was most pronounced, in three the right side most, in one both sides equally. The kidneys were normal in all six of these rats. The cecum showed slight inflammatory conditions in three rats. Worm cysts were found in every liver. Other viscera were normal, except that beginning pulmonary congestion was noted in two.

The presence of the worm cysts in the livers of one dozen of these animals adds a complicating factor. These rats were about equally divided between the controls and the lesioned cages, and the presence of a few of the worms did not seem to affect the general health of the controls, nor to give rise to any acute inflammatory changes in any of the viscera of the controls; it may be inferred that these did not materially affect the results of the lumbar lesions. Since the effects of the lesions in the rats in whom worms were found were, so far as we could determine, identical with the effects of the same lesions in rats in whom no worms were found, these rats may properly be included in the tables.

Pneumonia has been a constant danger to laboratory animals. When it appears, the disease spreads with remarkable speed over the other cages. Osteopathic treatment to the pneumonic animals seems to prolong their lives, but it permits the sick animals greater opportunity to spread the infection. No rat recovered after the symptoms became recognizable, though temporary improvement was several times noted. Convalescent rats are worthless for laboratory purposes. When an epidemic of pneumonia gains foothold, the immediate death of all animals, followed by thorough hot scrubbing and sunning of the cages, is the wisest course of action.

WHITE RATS WITH VERTEBRAL LESION FROM SECTION OF LIGAMENTS

Sept. 29, 1915. Two dozen rats were purchased and were kept under observation for ten days.

Oct. 9, 1915. Three normal females were anesthetized and the sacro-iliac articulation on the right side was cut through. All three made good recovery from the operation.

Oct. 16, 1915. X-ray examination shows slight separation of the sacro-iliac surfaces, which was the lesion expected from the operation of October 9.

Oct. 25, 1915. Two normal female white rats were anesthetized and the spinous process of the fourth lumbar vertebra separated from its neighbors. The wounds healed nicely.

Nov. 3, 1915. One of the females which had had an incision separating the spinous process of the fourth lumbar from its neighbors was anesthetized and the skin opened in order to see whether this had been successful in producing a bony lesion. The rat died almost at once under the anesthetic. Examination shows that the previous operation had been successful. The spinous process had been separated from its neighbors and the wound had healed well. The injured vertebra was much more freely movable than its neighbors and the entire condition seems to make a very good imitation of the bony lesion.

Autopsy. The uterine horns show areas of a whitish color which may be the site of beginning pregnancy. Ovaries and uterus were placed in Zenker's fluid for microscopic examination. The liver contains five large yellow cysts like those found in other rats, containing small tape worms. One of these cysts with the adjacent liver was also placed in Zenker's fluid. The kidneys of this animal were large and bloody and were prepared for microscopic examination. The slides prepared from this kidney are identical in appearance with the slide shown in Fig. 19.

During November and December these rats all succumbed to pneumonia.

The autopsy showed pneumonic lungs in all, and these resemble the lungs of control animals which also died of pneumonia

during the same weeks. No indications of pregnancy appeared in any of these rats at any time after the operation.

In the lesioned animals congested uterus and ovaries, large red kidneys and either congestion or adhesions in the ileo-cecal region were constant.

In non-lesioned animals dead of pneumonia, ovarian and uterine congestion and ileo-cecal inflammation were invariably absent. The kidneys in these animals were pale and rather smaller than normal. Pregnancy was occasionally present. The fetuses appeared normal, though slight abnormalities would probably escape notice in these sick rats.

PROTOCOLS OF RAT FEMALE CONTROLS

Families of three or four non-lesioned female rats with normal male were kept in cages in the stack also occupied by lesioned rats. During the month beginning November 12, 1915, there were two of these families, during December; five other families were added to these. No fatalities occurred during the two months.

January 20, 1916, two rats died—one male and one female. Autopsy showed pneumonia. Ovaries of female, kidneys of both, ileo-cecal region in both were all normal. The cages were emptied, scrubbed, sunned and disinfected.

Jan. 30. One rat gave birth to five young, and another to four young, all normal.

Feb. 17. Two rats bore seven young each, all normal. Mothers normal.

Feb. 24. One rat bore five young, and one three, all normal. Mothers normal.

March 2. One rat died. Autopsy showed death to be due to pneumonia. Many worms were encysted in the liver.

March 9. One rat bore eight young; all normal. Mother normal.

June 7, 1916. Female rat was found dead in control group. The thorax contained coagulated blood, and pressure upon the lungs caused dark blood to exude. No ruptured blood vessel or other traumatic injury could be found. The stomach, cecum and intestines were almost empty of food remnants, and contained much gas; they were otherwise normal. The pelvic organs were normal. No worms were found in the liver. The cause of death was pneumonia.

A normal rat in the control group gave birth to six young, all apparently healthy and perfect.

June 28, 1916. Rat dead in cage, "Normal, Nov., 1915." Left side of head and part of right; nearly all of the thoracic muscles on the left side and part of the right side of the thorax have been eaten, apparently by the others in the cage. Rat is female; five fetuses were in the uterus; all viscera appear normal, except

a single large worm-cyst in the liver. No cause of death was found, probably the animal was killed in a fight.

During April, May and June fifteen females of the controls have borne normal young. During the time of November 15 to July 15 every normal adult mated female gave birth to normal young at least once. Nine females gave birth to a second litter. From three to seven were born in each litter, with an average of four +.

During July and August fifteen females were anesthetized and used for various laboratory purposes. In each case the pelvic organs were examined. In nine cases pregnancy was well established, and appeared to be normal. No cystic ovaries were found, and the circulation through ovaries and uterus seemed to be perfectly normal for the stage of pregnancy present at death. In two rats there was no pregnancy, and the uterus presented the usual appearance of normal post partum relaxation and hyperemia. In four rats no signs of pregnancy were found; the uterus and ovaries were normal in every recognizable respect.

October 15, 1916. Fifteen females were grouped in families for controls.

These died or were killed in December during an epidemic of pneumonia. Twelve were found pregnant with three to six fetuses each.

TABLE SHOWING RELATIVE FERTILITY IN LESIONED FEMALE RATS AND GUINEA PIGS

Animals	Average time mated	Number of births	Number of young	Animals pregnant at autopsy	Number of fetuses found
40 lesioned rats	4.3 mo.	2, both followed by death of mother.	4 died soon after birth; 5 appeared normal.	1 ectopic gestation.	None.
40 normal rats	4.5 mo.	42 normal.	163 normal.	9	41, which appear to be normal.
10 lesioned guinea pigs	2.8 mo.	2 births, 1 miscarriage at about 18th day.	1 stillborn; 3 died soon after birth	3	1 abnormal, 5 which appear normal.
6 normal guinea pigs	6 mo.	6 births	15, all normal.	6 (25 to 30 days gestation).	18, all appear normal.

SUMMARY OF PROTOCOLS OF LESIONED MALE RATS

June 1, 1916. Ten male rats have been kept under observation for two weeks, during which time they seem normal.

Lesions of the second or third lumbar vertebra were made in all. This was done by the following method: We held the upper part of the body firmly; then by grasping the transverse processes of the third and giving first a few trying-out twisting movements, it was usually easy to produce a traumatic lesion. The spinous process of the third lumbar vertebra was rotated slightly to the right and very slightly upward.

Erection occurred in eight of the ten rats; this persisted for two days in one rat, for one day in one rat, and for several hours in six rats. Retraction of one testicle occurred in all; the testicle descended again almost immediately.

One rat was apparently paralyzed in the hind quarters for a few hours after the lesion.

Nine rats seemed uncertain in gait and presented evidence of slight incoördination of hind leg movements for several hours after the lesion was produced. The leg muscle symptoms are more pronounced in the males than in the females.

June 2. Incoördination persisted to some extent in all the rats.

June 9. The rats appeared to have recovered complete use of legs. Lesions persisted. During June and July the rats were examined twice each week. The lesions persisted. Examination of the lumbar spine usually caused erection and sometimes retraction of the testicle, this more often on the right side than the left.

During July the rats were anesthetized and killed. The testicles were invariably deeper pink than in normal rats of the same class. The walls of the scrotum were relaxed. In five cases the scrotum contained a bloody fluid.

In all cases the kidneys were congested—large and full of blood. In all cases the ileo-cecal region was congested, with some fibrinous exudate in the neighboring tissues.

Five normal male rats from the cages of controls were killed and dissected in the same way and at the same time. In one of these some very slight ileo-cecal congestion was found. No other pathological conditions were found in the control animals.

MALE CONTROLS

During the time intervening between September 29, 1915, and December 14, 1916, four dozen male white rats spent variable periods of time in the cages included as controls. The normal males mated to the lesioned females may also be included as control animals. The number of male controls in the cages varied, as some animals were occasionally taken from these cages to be used for laboratory purposes.

The causes of death in these rats include:

Anesthesia, given in preparation for experiments which killed the animals: twenty-seven rats.

Fighting among themselves: three rats.

Senility: one rat.

Pneumonia: ten rats.

Over-grown tooth which prevented the rat from eating: one rat. The rat died from starvation.

Anesthetised when pneumonia became apparently about to be fatal: three rats, Dec. 14, 1916.

Autopsies: The pneumonia rats were examined in seven cases. The lungs were hemorrhagic; hepatization sometimes occurred, but death often preceded the coagulation of the blood in the lungs. Slides were prepared from these lungs. The kidneys in rats dying of pneumonia sometimes showed slight congestion, but not usually marked inflammatory conditions. (Lesioned rats dying of pneumonia showed very serious kidney involvement.) The testicles, bladder, cecal region, intestines, appeared normal in all control rats dying of peumonia.

All rats examined from one lot of two dozen contained tapeworms in the liver. These were present in controls as well as lesioned rats.

Controls killed for other laboratory purposes were examined always. No diseases were found except as stated above and in the protocols elsewhere.

Congestion of the reproductive glands and kidneys, and of the ileo-cecal region, were not found in the controls except under unusual circumstances.

THE ILEO-CECAL REGION IN LESIONED ANIMALS

White, or albino, rats were chiefly employed in this group. The non-lesioned white rat occasionally has an inflammatory condition in the neighborhood of the ileo-cecal valve.

Of fifty-five lesioned white rats, one had an ectopic gestation, and the ileo-cecal region was included within the mass of adhesions; this rat is omitted from the count. Another died from an intestinal ulcer; this also is omitted from the count. This leaves fifty-three lesioned white rats, in whom lesions were known to be present from one to eight and one-half months. In a large proportion of rats the lesions were present about two months.

Four of these rats showed no evidences of ileo-cecal inflammation. These had been lesioned one month, three and one-half months, four and one-half months and eight and one-half months respectively. In eleven rats the ileo-cecal region included congested areas. This congestion was usually found chiefly near the valve. In forty-four rats an exudate was found; this was confined within the meshes of connective-tissue overgrowth in some, and occupied the folds of the peritoneum in others. This exudate was small in quantity. was slightly gelatinous, and was not analyzed. Thirty-eight of the rats had adhesive bands, which connected the loops of the intestines in an abnormal manner, and amongst which the exudate was often confined. In only one rat was this mass distinctly cyst-like. In one rat the lymph nodes were very distinctly enlarged. (In most of the rats with ileo-cecal pathology, the lymph nodes showed some evidences of enlargement, but the physiological variations are so marked that slight enlargements were not recorded.)

In one group of guinea pigs the condition of the appendix was not recorded. In another group of nine guinea pigs, including both sexes, the ileo-cecal region was uniformly congested, and adhesions were present in varying degree. In normal rats and guinea pigs no adhesions are found. In non-lesioned animals congestion and exudate, and occasionally adhesions, are not so very rarely found; in other words, the bony lesion does not appear to be the sole cause of this condition among the animals mentioned.

Fig. 15 shows a normal male guinea pig with the intestines drawn out to expose the ileo-cecal region. In rodents this part of the intestine is much longer than in the higher mammals. The great length of the appendix is shown in the photograph. (In birds the corresponding structure is called the "third gut" and may be longer than the entire small intestine and colon together.) The ileo-cecal region bears marked resemblance to the corresponding neighborhood in the higher mammals and man.

In four rabbits kept lesioned for two to three months, the ileo-cecal region showed some evidences of congestion. This was much less marked than in the white rats, however. In six non-lesioned rabbits, one had very slight congestion, five showed no evidences whatever of congestion or of adhesions.

In eight cats with lesions present when they were brought for examination, some congestion and adhesions were found. In non-lesioned cats—several dozen (exact number not recorded) —these inflammatory bands were found rather rarely. Congestion was not found in cats in whom neither bony lesions nor visceral disease were present.

Dogs appear less prone to ileo-cecal disease from the effects of upper lumbar lesion. Sixteen lesioned dogs showed no sign of ileo-cecal congestion or adhesions; two dogs showed congested areas around the ileo-cecal valve, and in two the entire neighborhood was the seat of acute inflammation.

Findings.

The lumbar lesion must be considered a disturbing factor in the circulation and the secretion of the organs of the ileo-cecal region. The fact that lesions in the thoracic and in the lower lumbar spine are often mentioned in clinic reports should restrain any opinion as to the relative importance of these various groups of lesions until experiments have been completed concerning the effects of these upon the circulation and the nutrition and the immunity of the intestinal tract throughout its entire extent.

The place of upper lumbar lesions in the predisposing etiology of appendicitis has been recognized by many osteopathic physicians, and reports have been published in all the important periodicals giving cases which show this relationship. Other lesions are also important in this disease, according to the literature, and these are usually given between the mid-thoracic and the lower

lumbar spinal column, with the lower ribs and the cervical region also mentioned rather prominently.

Lumbar lesions are often found associated with visceroptosis in human patients. Fig. 16 shows a prolapsed colon in a patient with several lesions of the lumbar region.

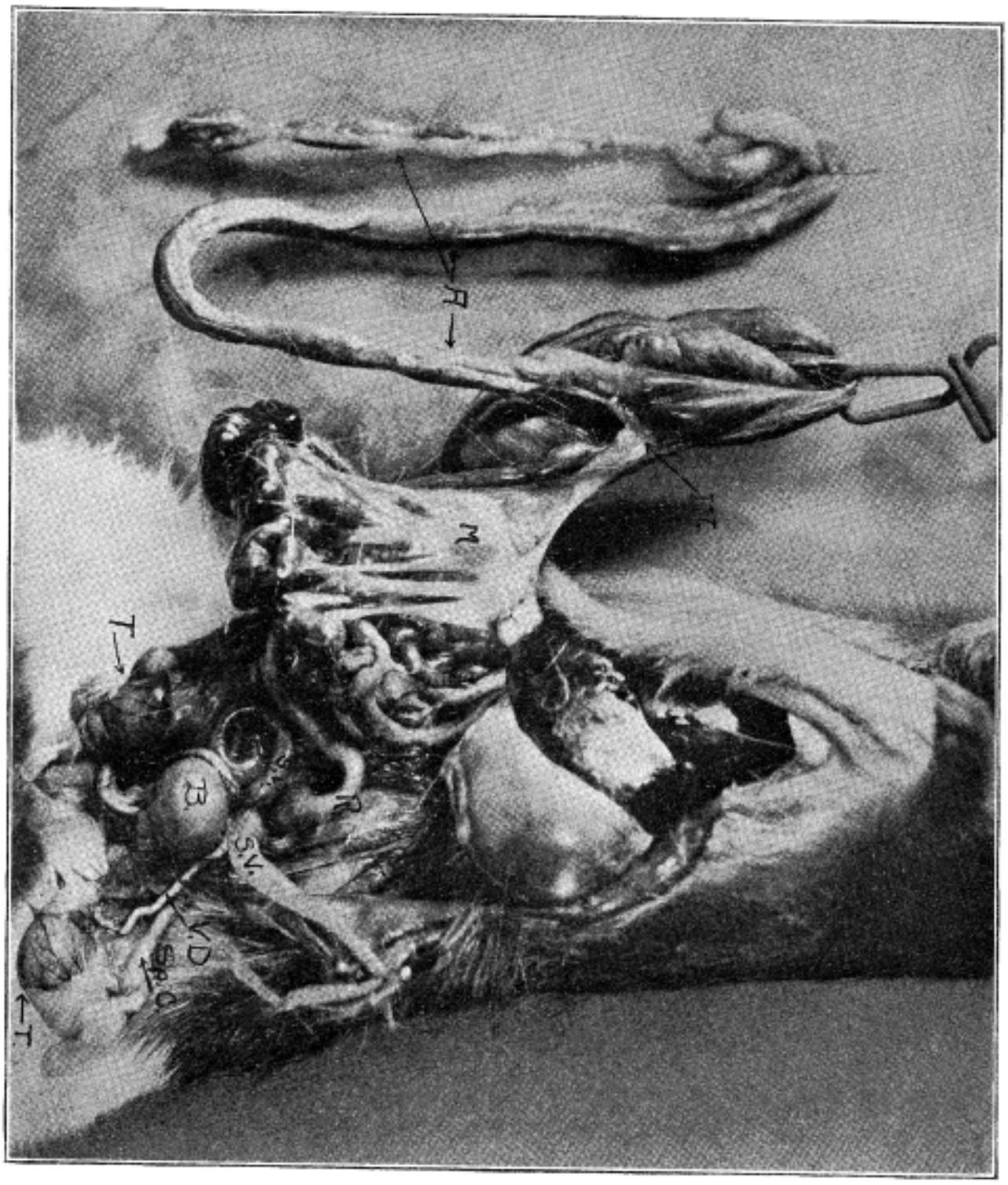

Fig. 15

Fig. 15. Adult male guinea pig with the abdomen opened and the intestines drawn out to show the ileo-cecal region.

I.V. External aspect of the region of the ileo-cecal valve.

A. Appendix, or "third gut." It arises from the cecum, which in the guinea pig is scarcely distinguishable from the colon. The colon is almost hidden by the small intestines. Note the length of the appendix.

M. Mesentery, from the folds of which the small intestines are hanging.

R. Rectum.

S.V. Seminal vesicles. The right is in its normal curled position at the side of the bladder; the left has been drawn out so as to show its length.

V.D. Left vas deferens, drawn out with the testicles. The right vas deferens can be seen at the side of the bladder.

Sp.C. Left spermatic cord, drawn outward with the testicle.

T. Testicles. The right lies just within the abdominal ring, in the position to which it is drawn after sudden production of lumbar lesions, and under certain other conditions. The left testicle has been drawn outside of the body, so as to show the anatomical relations. Note the blood vessels upon the surface.

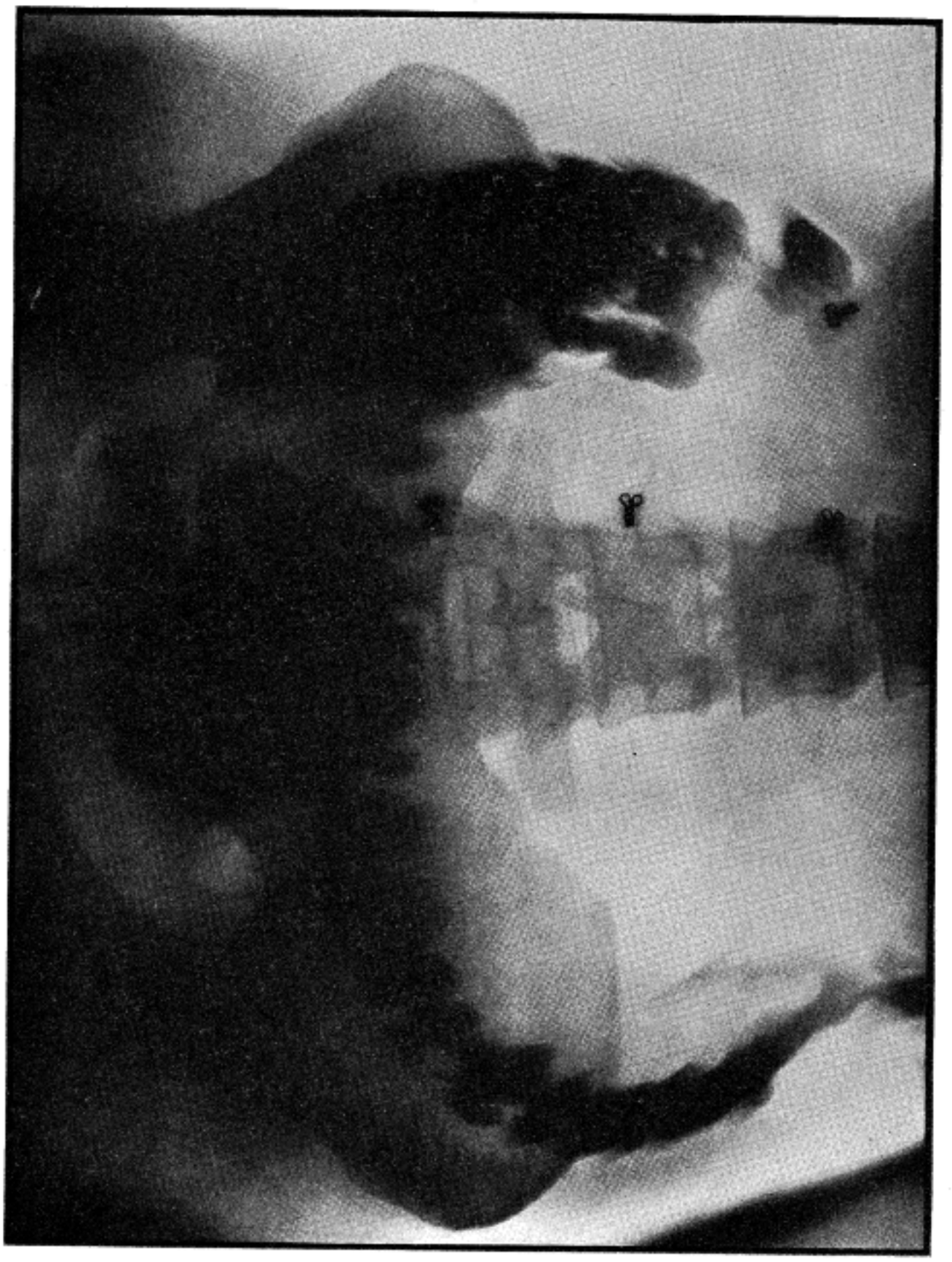

Fig. 16

Fig. 16. Radiograph showing ptosis of transverse colon; patient in horizontal position. Bismuth sub. carb. enema. Dilatation and sluggish movement at hepatic flexure (which was location of pain). Innominate lesion, with slight irregularities involving the entire lumbar region.

VISCERAL PATHOLOGY IN LESIONED ANIMALS

While the number of animals studied so far is too small to warrant any definite statements regarding the exact place of lumbar lesions in the etiology of diseases of the abdominal and pelvic organs, yet these records may serve as a beginning in the accumulation of the statistics necessary to a better understanding of the causes of these most serious disorders.

The findings here reported include a few animals in whom the lesion was present at the first examination. For the most part, however, the lesions were produced experimentally.

Of groups of animals watched for some weeks and known to be normal as far as could be determined by careful examination, about half were lesioned, after the methods already described. After lesioning, the animals were kept under as nearly normal conditions as possible, as were also the non-lesioned animals used as controls. The lesioned and non-lesioned animals were always kept under exactly the same conditions, and were often in the same cages.

After varying lengths of time the animals were killed, or they died, and they were then examined carefully. Specimens of tissue were put up in paraffine and cut, and were made the subject of microscopic study. From these specimens the microphotographs illustrating Bulletins No. 4 and No. 5 were prepared.

The first effect produced upon the viscera by a lesion suddenly produced is usually a slight and temporary constriction of blood vessels. In some animals this period is lacking, or is so short as to be imperceptible.

This temporary constriction is followed by a period of hyperemia, and this by rather marked congestion of an active type. During the early stages of congestion the activity of the organs may be increased; secretions are increased in quantity, and the muscular contractions of hollow viscera are increased in speed and sometimes in vigor.

The congestion passes, somewhat gradually, into that somewhat resembling the "passive congestion" of older pathologists. The organs are heavy and of a dull purplish color, and their

functions are usually diminished. Edema and hemorrhagic areas are found in the sections prepared for microscopic study.

The blood vessels appear engorged, and hemorrhage per diapedesin occurs. In the ovary the ovum cells show some cloudy swelling, and the Graafian follicles appear to be rather more edematous than is usual. (Fig. 17, 22, 23.)

The hemorrhagic areas appear very large in the uterine tissues. This hemorrhage is almost if not quite always per diapedesin, and the blood is found of varying shades of reddish brown and yellow, according to the time passed since its extravasation. The superficial cells in the uterine lining may be variously disintegrated and degenerated, in a way which is never found in the uterus of the non-lesioned, healthy animal. (Fig. 18.) The gross appearance of the heavy, edematous, dark-colored uterus of lesioned animals is easily recognized.

The kidneys present characteristic changes. The glomeruli are edematous. The cells show varying degrees of cloudy swelling. Hemorrhagic areas appear in the tubular region and among the glomeruli. These hemorrhages are small and rarely they are organized. (Fig. 19, 20, 21.)

The presence of tumors in lesioned animals is interesting, and suggest further investigations along the same line. (Fig. 9, 37, 38, 39.)

The nature of the vertebral lesions in the animals used during the last year, and whose history provides the materials for this and the preceding Bulletins, is shown in the X-ray plates. (Fig. 3, 4, 5, 7, 8, 11, 24, 25, 27, 29, 30, 50.)

The lack of flexibility in lesioned animals is difficult to illustrate. (Fig. 26, 28.)

The pathologic findings in animals with lumbar lesions are tabulated.

Table Showing Abnormalities of Certain Viscera in Animals with Lesions of First to Third Lumbar Vertebrae

Animal	Time Lesioned	Cause of Death	Ovaries	Uterus	Placenta	Ileo-cecal Region	Kidneys	Scrotum	Testicles	Other Organs Affected
Rat 1	1 mo.	Ether.	Normal.	Normal.	None.	Normal.	Large, red.			
Rat 2	1.5 mo.	Pneumonia.	Congested.	Congested.	None.	Congested.	Large, red.			
Rat 3	1.5 mo.	Pneumonia.	Congested.	Congested.	None.	Congestion and exudate.	Large, red.			
Rat 4	2 mo.	Pneumonia.	Congested.	Congested.	None.	Congestion.	Large, red.			
Rat 5	2 mo.	Pneumonia.	Congested.	Congested.	None.	Congestion and exudate.	Large, red.			
Rats 6, 7, 8, 9, 10	1.5 mo.	Ether.				Congestion and exudate.	Large, red.	Walls relaxed, contain bloody fluid.	Slight congestion.	
Rat 11	1 mo.	Ether.				Congested.	Congestion.	Walls relaxed.	Slight congestion.	
Rat 12	2½ mo.	Pneumonia.	Congested and cystic.	Congested.	None.	Adhesions and exudate.	Large, red.			
Rat 13	2½ mo.	Pneumonia.	Congested.	Congested.	None.	Congested.				
Rat 14	2 mo.	Pneumonia.	Cystic.	Congested.	None.	Adhesions and exudate.	Large, red.			
Rat 15	3½ mo.	Died after giving birth.	Cystic.	Usual post-partem.	Not found.	Normal.	Large, red.			
Rat 16	4½ mo.	Died in giving birth.	Congested and cystic.	Usual post-partem.	Not found.	Normal.	Large, red.			
Rat 17	5½ mo.	Intestinal ulcer.	Right congested, left contains abscess.	Congested.	None.	Adhesions and exudate.	Normal.			Lymphoid masses around both ovaries. Intestinal ulcers.
Rat 18	7½ mo.	Rupture of sack of ectopic gestation.	Slightly congested right, upon sack.	Congested.	Lines the sack.	Involved with other tissues in sack.	Left, large; right, red, included in sack.			Ulcer in intestines.
Rat 19	8½ mo.	Pneumonia.	Slightly congested; cystic.	Accymotic.	None.	Adhesions, exudate; enlarged lymph nodes.	Slightly hyperemic.			Lungs show usual changes.
Rat 20	8½ mo.	Pneumonia.	Congested with small cysts.	Congested.	None.	Normal.	Large, red.			
Rat 21	9 mo.	Ether	Deeply congested.	Deeply congested.	None.	Adhesions and cysts.	Large, red.			

TABLE SHOWING ABNORMALITIES OF CERTAIN VISCERA IN ANIMALS WITH LESIONS OF FIRST TO THIRD LUMBAR VERTEBRAE—*Continued*

Rats 22-55, including males and females	2-3 mo.	Ether, chloroform or blow on head.	Congested or cystic; rarely normal.	Congested or normal.	None or adherent or normal.	Adhesions and exudate always.	Large, red always.	Walls relaxed always.	Retracted or congested or normal.	
Guinea pig 1	3½ mo.	Died after miscarriage.	Cystic.	Contained tumor.			Large, red.			
Guinea pig 2	2 mo.	Chloroform.	Normal.	Congested.			Large, red.			Liver spotted.
Guinea pig 3	4 mo.	Chloroform.	Normal.	Contained tumor.			Large, red.			Liver spotted.
Guinea pig 5	4½ mo.	Chloroform.	Normal.	Venous congestion, walls friable.	Two or three degenerated placental masses.		Normal.			
Guinea pig 8	4 mo.	Chloroform.	Contained tumor.	Normal pregnancy.	Three normal.		Large, red.			
Guinea pig 11	5 mo.	Chloroform.	Left normal, right cystic.	Venous congestion, thinned over placentas.	Invading uterine walls.		Large, red.	Congested.		
Guinea pig 12	1 mo.	Ether.					Large, red.	Retracted.		Priapism.
Guinea pigs 15-23	Present when given.	Chloroform.				Congestion and adhesions.	Large, red.	Congested.		Priapism.
Rabbits 1-4	2-3 mo.	Blow on head.	Congested.	Congested.	None.	Congested.	Large, red. (Fig. 21)			Left suprarenal congested in one.
Cats 1-5	Present when given.	Chloroform or ether	Cystic.	Relaxed and congested.	None.	Congested.	Normal or congested.			
Cats 6-8	Present when given.	Chloroform or ether				Congested.	Large, red.	Relaxed.	Epididymus cystic.	
Dogs 1-7	Present when given.	Chloroform or ether	Congested or cystic.	Relaxed and congested.		Normal.	Normal.			
Dog 8	Present when given.	Ether.	Congested and cystic.	Pregnant.	Cystic.	Congested.	Large red.			
Dog 9	Present when given.	Chloroform.	Right, large cyst; left, normal.	Normal.	Normal, 1 fetus.	Congested.	Normal.			
Dogs 10-14	Present when given.	Chloroform or ether.				Normal.	Normal.	Relaxed edematous.	Very pale.	Priapism.
Dogs 15-16	Present when given.	Cyanide.				Inflamed.	Large, red. (Fig. 20)	Relaxed.		

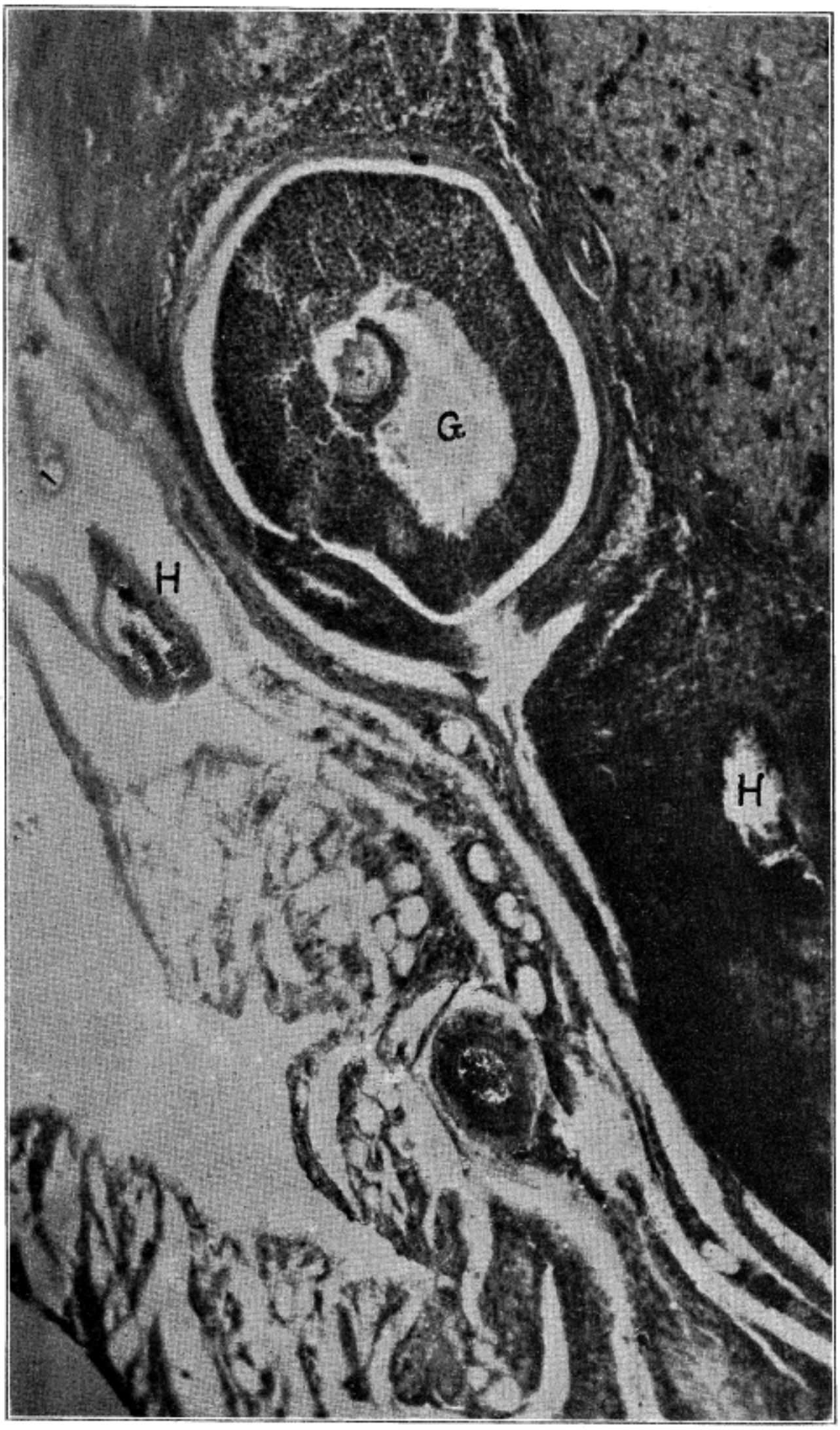

Fig. 17

Fig. 17. Microphotograph; ovary of white rat with upper lumbar lesion.

G. Graafian follicle containing ovum.

H. Hemorrhagic areas.

The blood vessels are engorged and hemorrhagic areas are to be found scattered through the mass of the ovary, especially in the central part of the lobules.

Fig. 18

Fig. 18. Microphotograph; uterus from same lesioned rat from which ovary in Fig. 17 was taken. Longitudinal section; central cavity is shown.

H. Hemorrhagic areas, some fresh, some old blood. Blood vessels engorged; some edematous areas.

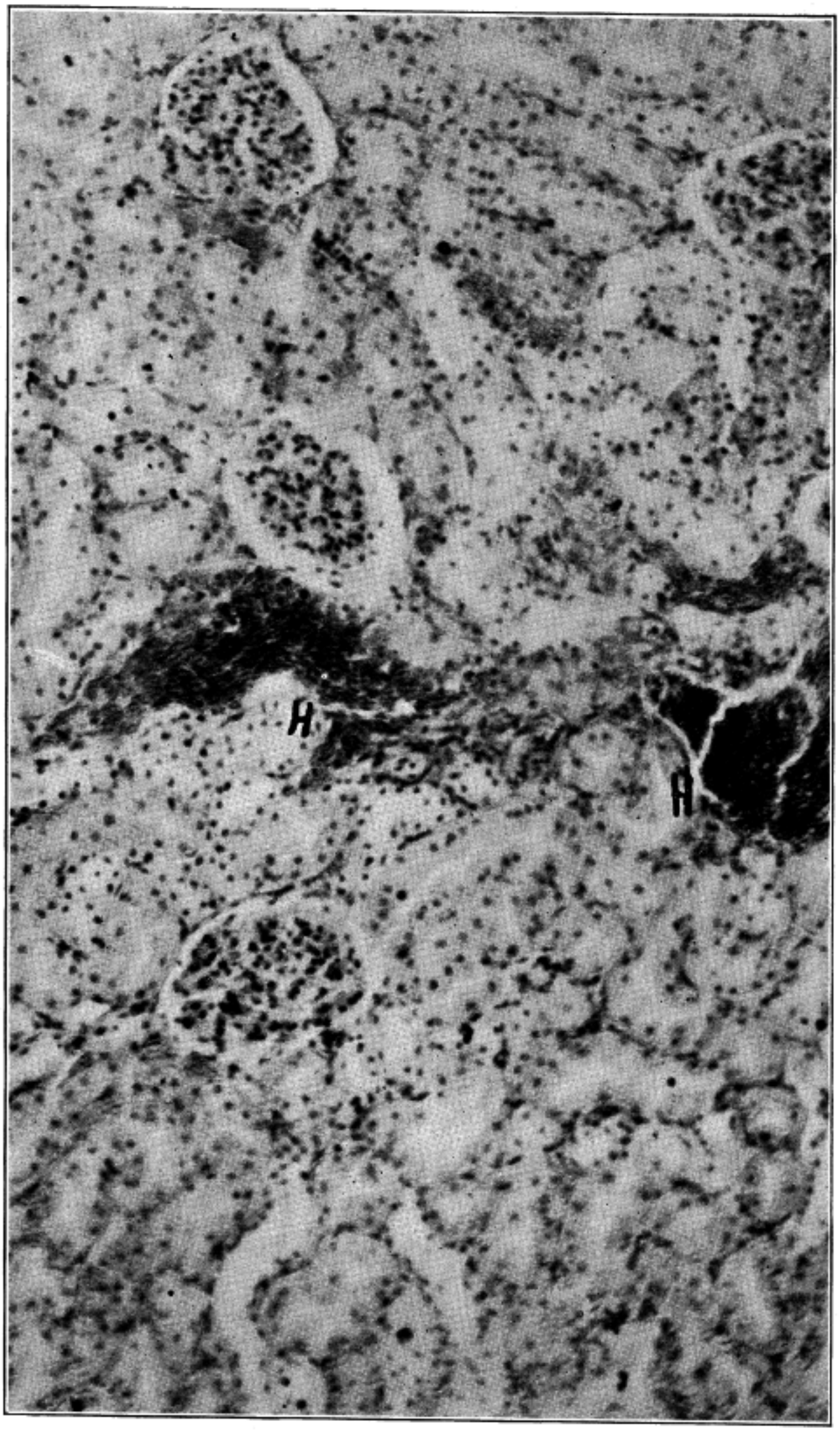

Fig. 19

Fig. 19. Microphotograph; kidney from white rat with upper lumbar lesion.

H. Hemorrhagic areas.

Small hemorrhagic areas are scattered throughout the section.

Glomeruli show hemorrhages and diapedesis.

Cells show cloudy swelling.

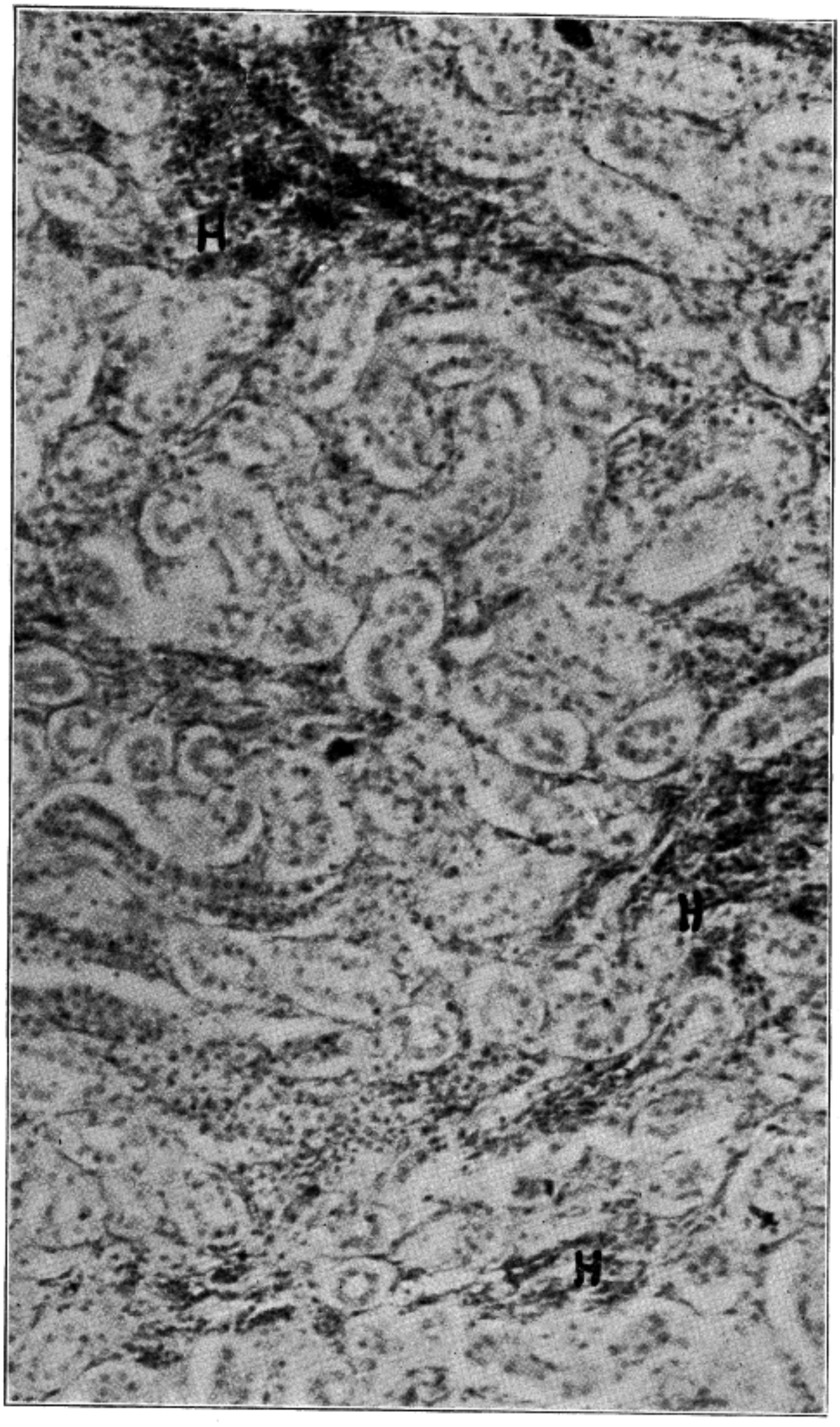

Fig. 20

Fig. 20. Microphotograph; kidney from dog with dorso-lumbar lesion, present when the dog was brought to the Institute.

H. Hemorrhagic areas. Diapedesis is present throughout the section.

Edema and cloudy swelling can be seen. The bladder contained urine in which albumin was heavy and casts plentiful.

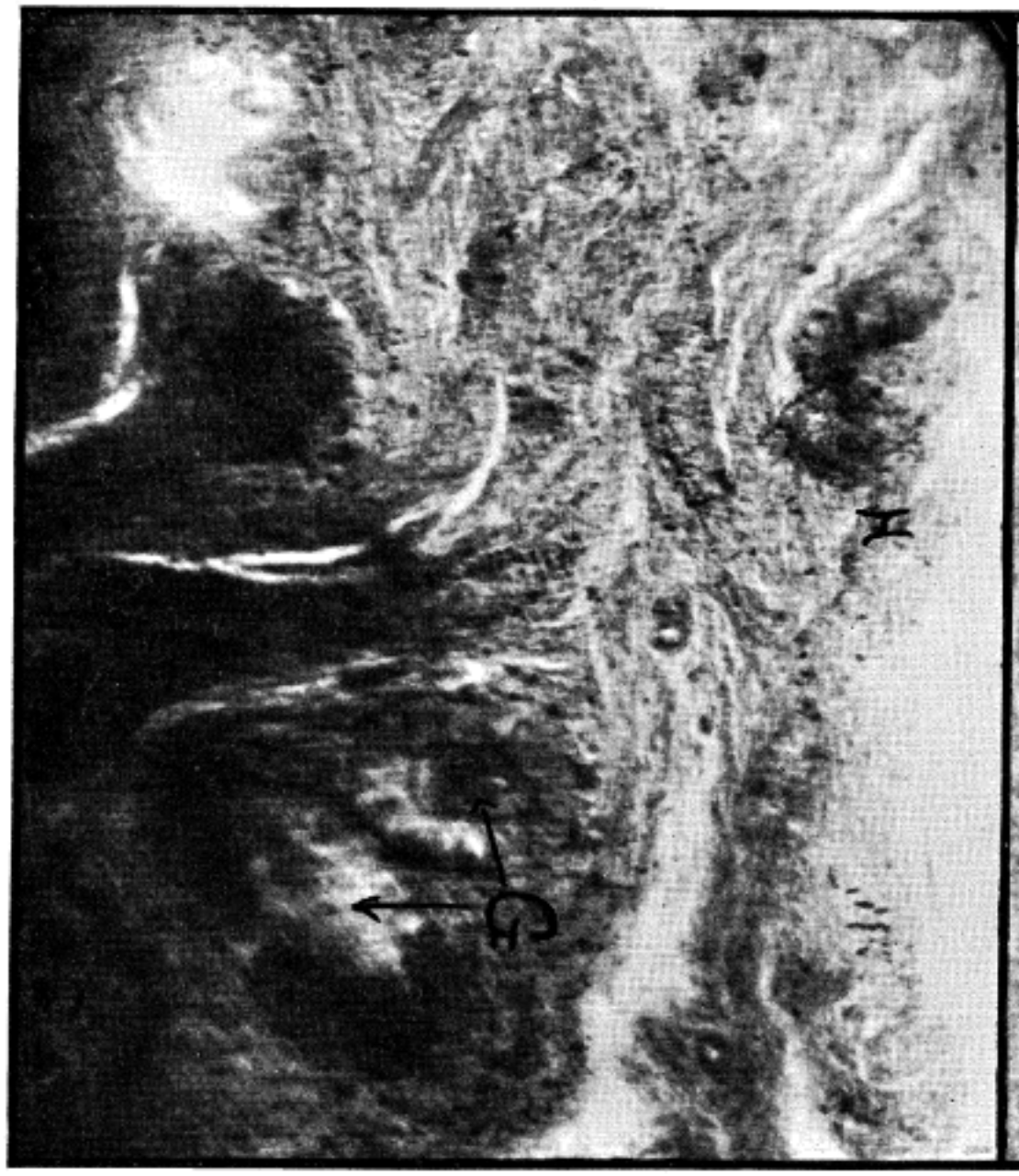

Fig. 22

Fig. 21

Fig. 21. Microphotograph of kidney of rabbit with upper lumbar lesion.

H. Hemorrhagic area. Hemorrhages and diapedesis are uniformly scattered over the sections.

Glomerular edema is more marked than is usual in the kidneys of lesioned animals.

Fig. 22. Microphotograph of ovary of dog with upper lumbar lesion.

G. Graafian follicle. Some diapedesis can be seen; this is not found normally before the escape of the ovum.

H. Hemorrhagic areas. These occur occasionally throughout the sections.

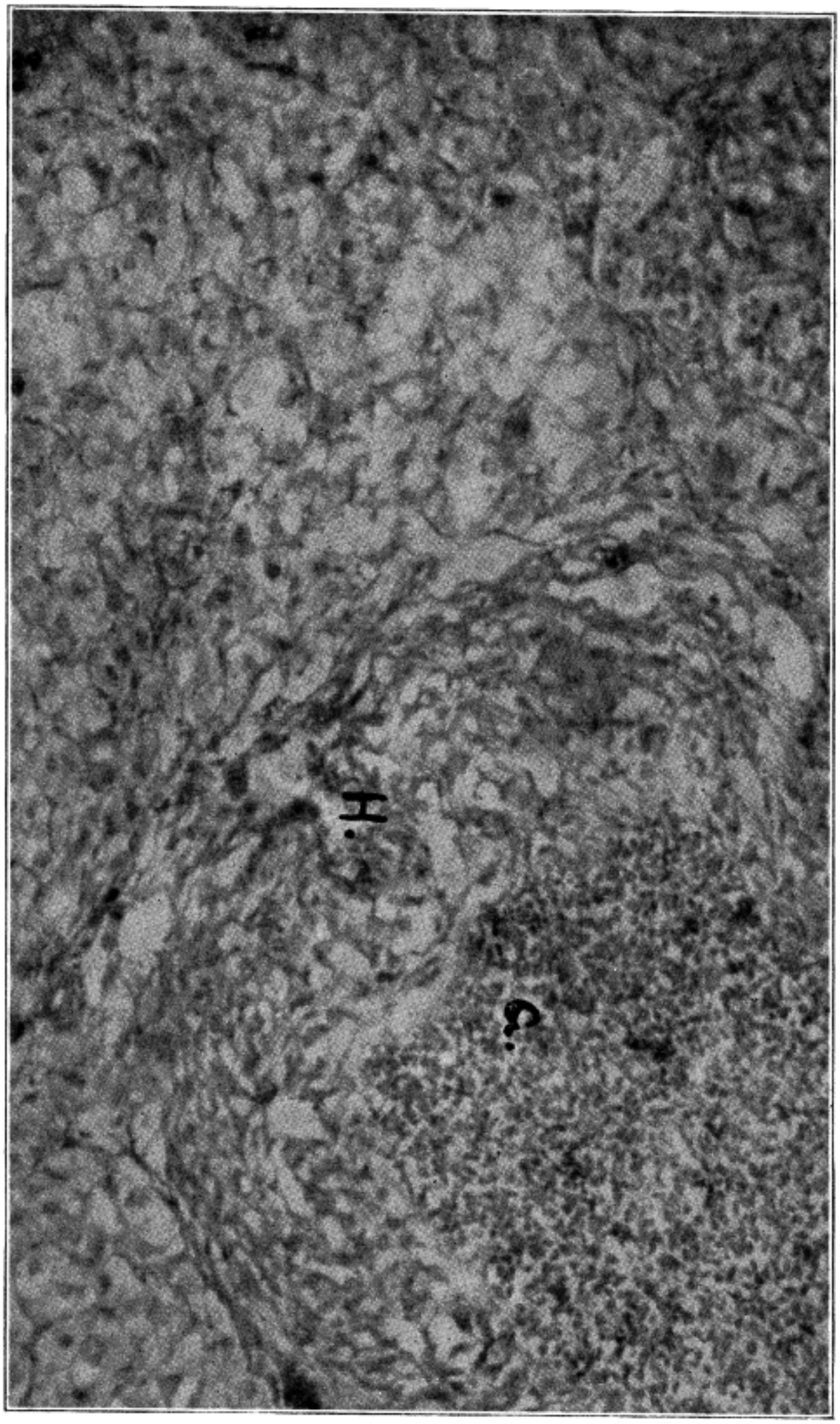

Fig. 23

Fig. 23. Microphotograph; ovary of rabbit with upper lumbar lesion.

G. Graafian follicle.

H. Hemorrhagic area.

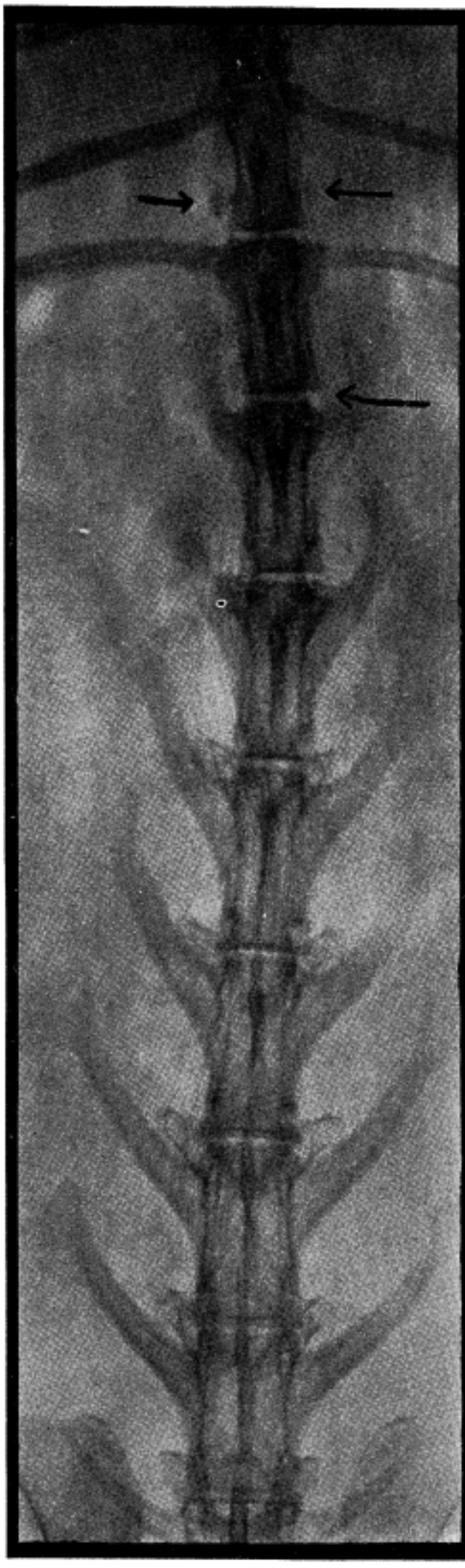

Fig. 24

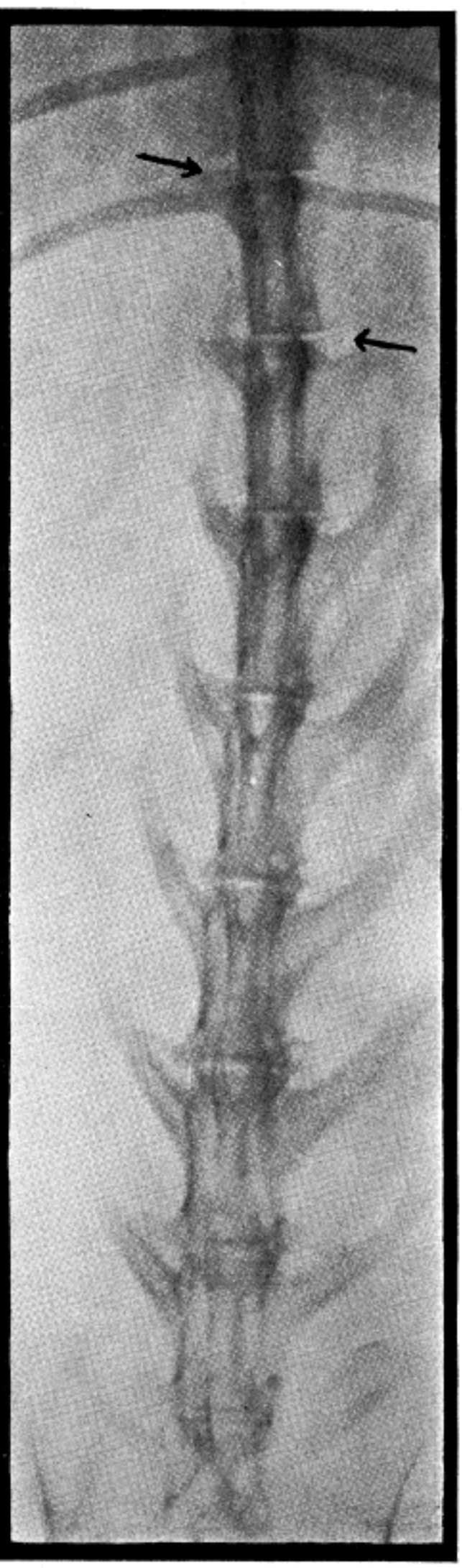

Fig. 25

Figs. 24, 25. X-ray plates of spinal columns of rabbits, showing lesions.

The arrows indicate the lesions, most of which are surrounded by edema and fibrosis.

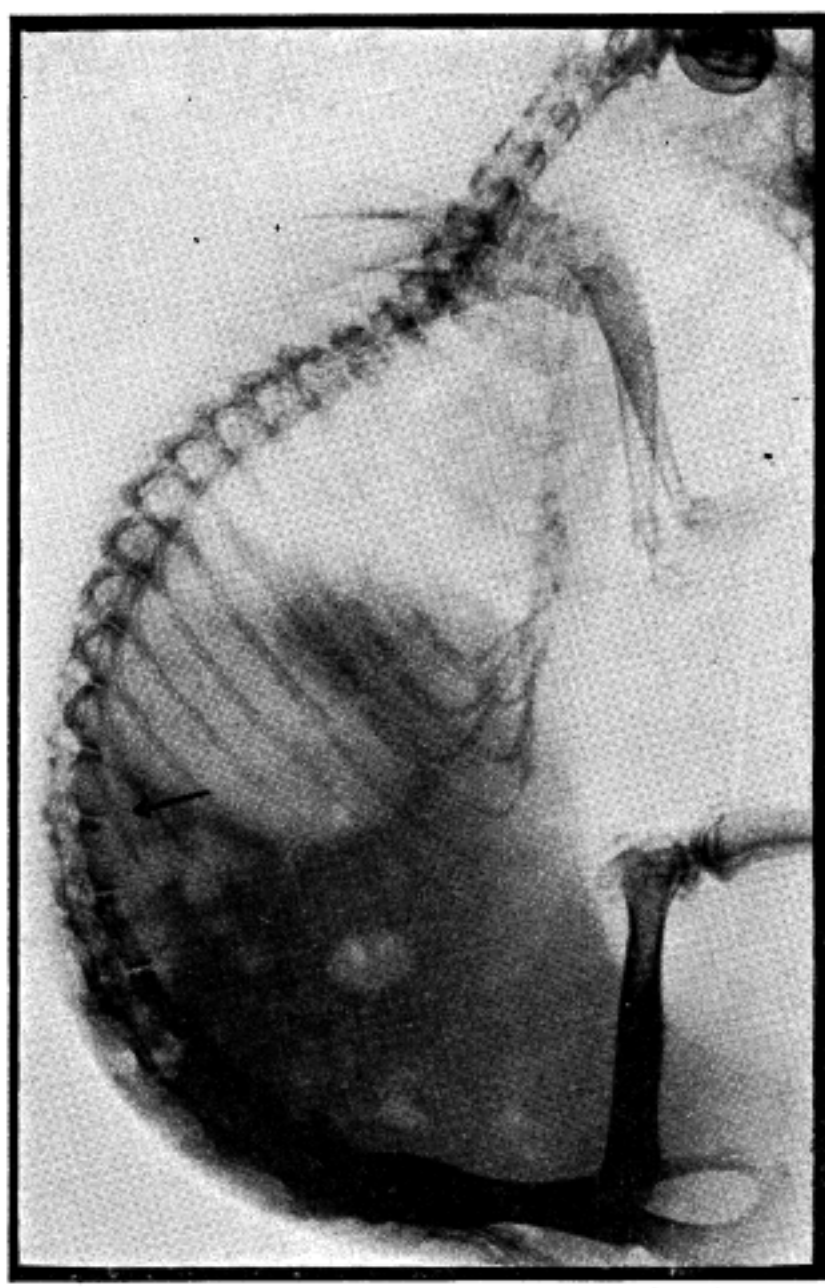

Fig. 26

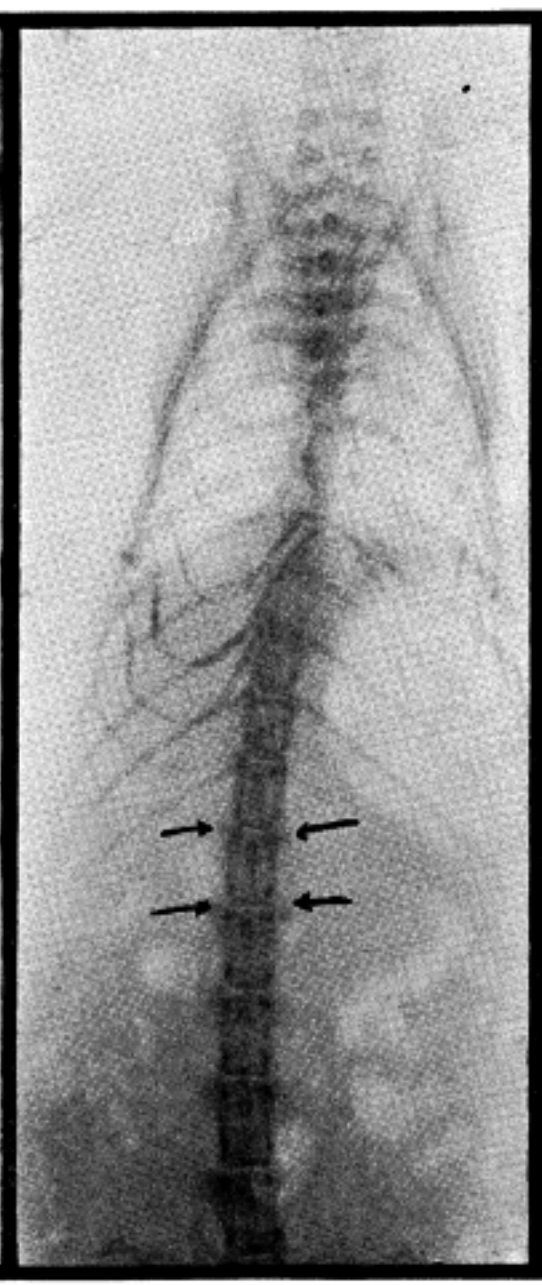

Fig. 27

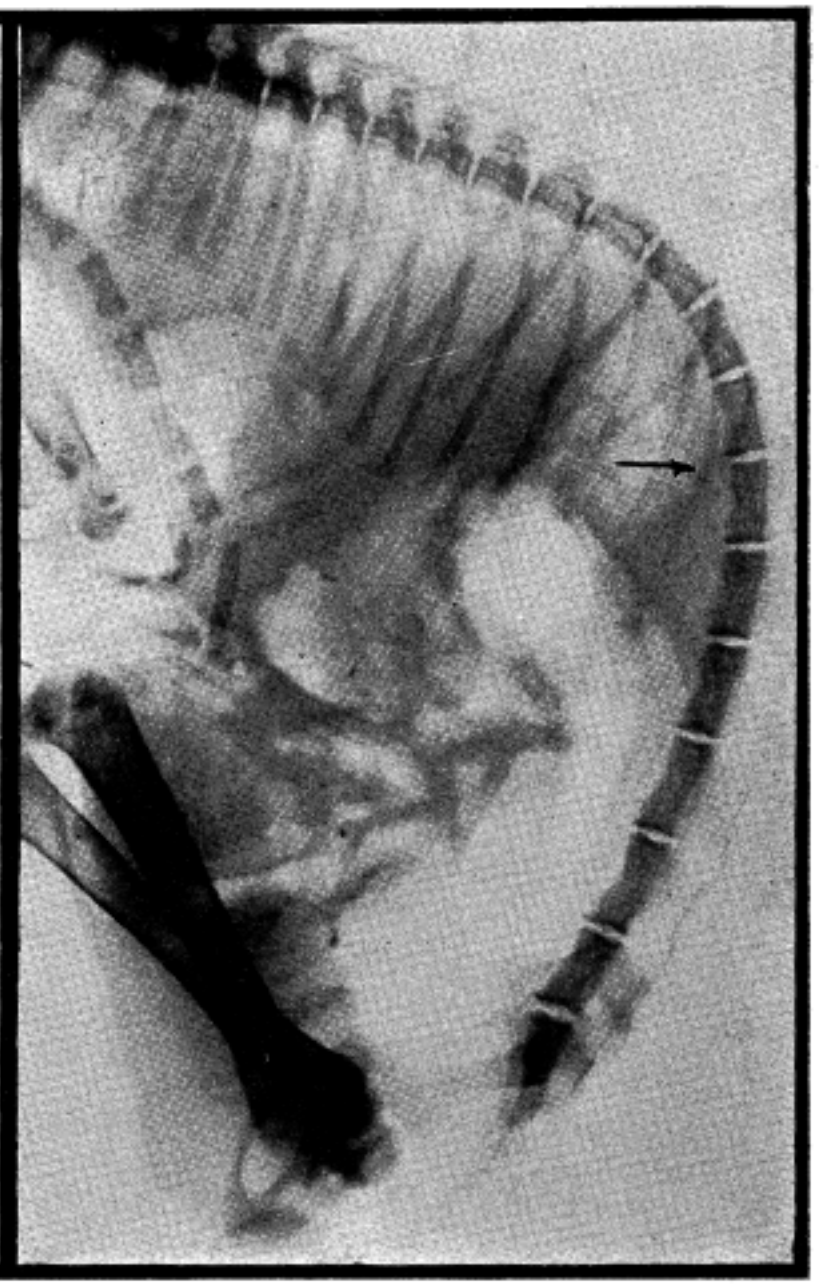

Fig. 28

Figs. 26, 27, 28. X-ray plates of guinea pigs, showing diminished flexibility and location of lesions.

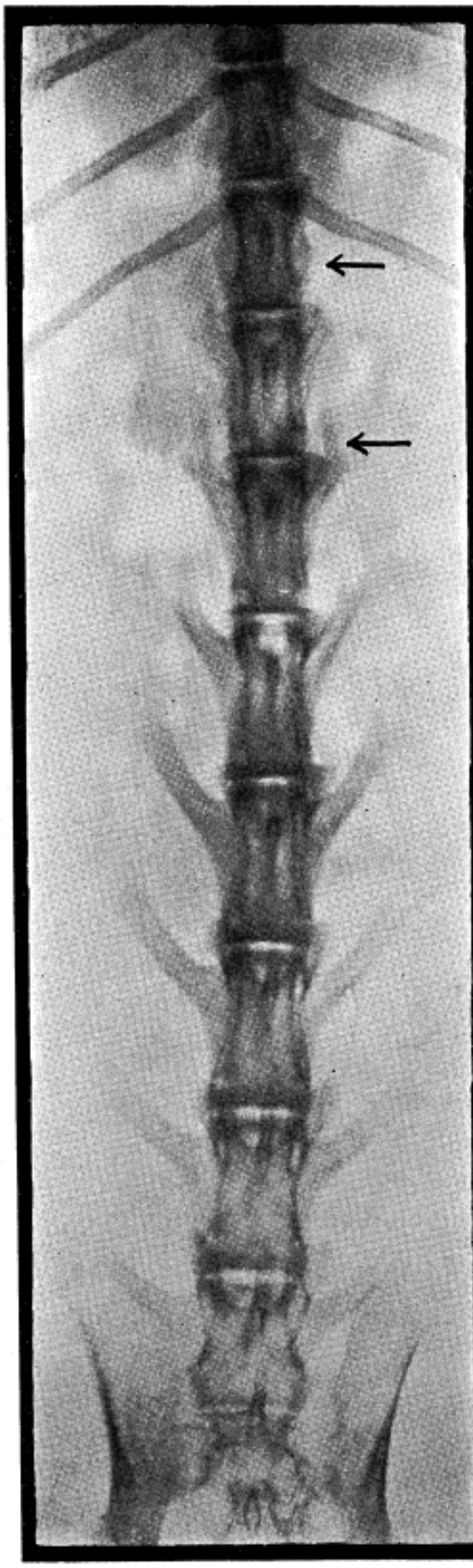

Fig. 30

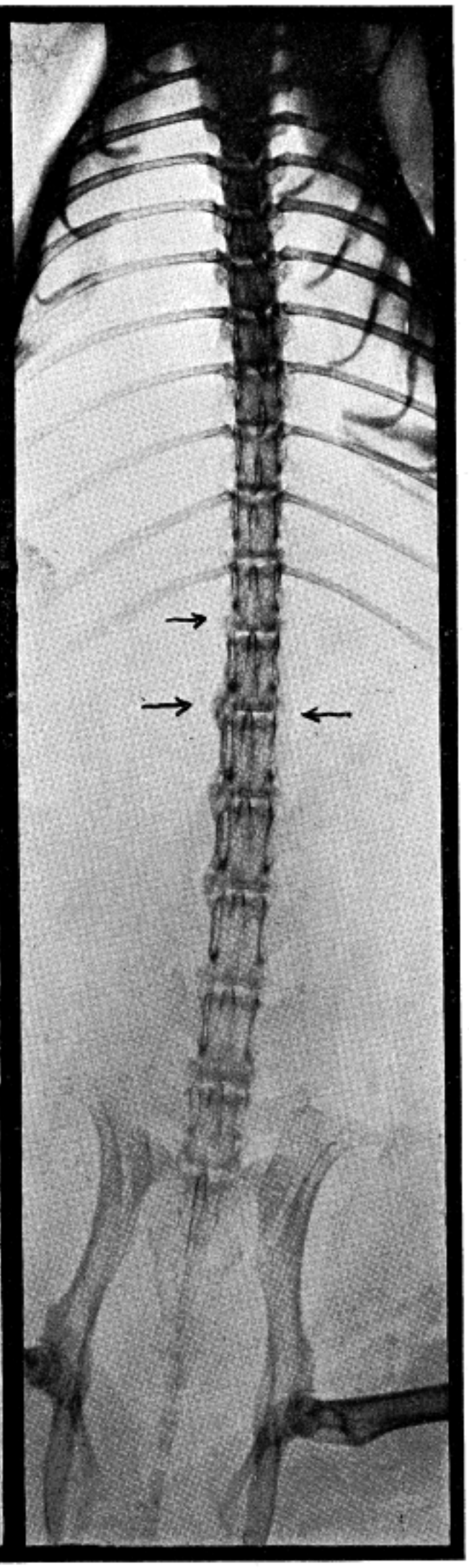

Fig. 29

Fig. 30. X-ray plate of rabbit, showing lesions with neighboring edema. The distinction between the edema, fibrosis and bony transverse processes is shown.

Fig. 29. X-ray plate of guinea pig, showing lesions in which the edema and fibrosis are marked, with slight bony mal-adjustment.

THE NERVOUS CONTROL OF THE REPRODUCTIVE ORGANS.

The vertebral lesion appears to affect the various tissues of the body partly by way of the nerve centers. This effect is probably secured in at least a two-fold manner; possibly there are also other pathways from the lesion to the affected tissues.

The reflex effect is unquestionable; sensory nerve impulses from articular surfaces concerned in the vertebral lesion, sensory nerve impulses from the muscles, ligaments, and other periarticular tissues affect the centers in the spinal cord of the same segments and of adjacent segments, and the activity of these centers may be adversely modified thereby. Efferent impulses to the structures innervated from those segments thus are more or less modified, and thus nervous disturbances become initiated in the affected organs or tissues.

The changes which occur in the position of the pelvic organs during embryonic development explain certain peculiarities of innervation, and thus the manner in which the reflex effects of bony lesions of the lower thoracic and the lumbar region upon these organs.

The reproductive glands, ovary and testis, are developed from the somites corresponding to the tenth, eleventh, twelfth and thirteenth thoracic segments of the adult and the first lumbar, perhaps also the second lumbar somite. The uterus and the prostate arise from lower somites, probably the fifth to the seventh lumbar and the sacral. The body of the uterus in the human corresponds to the united lower portion of the tubes in most mammals—perhaps it is better to say that the single human uterus arises from the union of the two horns of the bicornate uterus of the lower mammals. In the early human embryo also the uterus is bicornate. The prostate in the male corresponds fairly well to the uterus in the female. These points are of importance in appreciating the significance of the innervation of the pelvic organs in both sexes.

The innervation of the pelvic organs has been fairly well studied, from an anatomical standpoint, but some further study

of the spinal centers seemed advisable in connection with our study of the effects of lumbar lesions on the pelvic organs.

ANATOMICAL CONSIDERATIONS

The pelvic organs receive nerve fibers from the spinal cord. Sympathetic ganglia of the lower thoracic, lumbar and sacral regions are more or less directly concerned in the nervous control of the pelvic viscera. From the thoracic spinal segments the lesser splanchnic nerves arise and these enter into the formation of the various plexuses of the lower abdomen and pelvis.

The lumbar part of the gangliated cord of the sympathetic consists of four or five ganglia on each side, connected by nerve fibers. (Fig. 32.)

The sacral part of the gangliated cord consists of four or five ganglia upon each side of the sacrum; from the lower pair of these fibers pass to a single ganglion which lies in front of the coccyx—the ganglion impar.

The anterior divisions of the lumbar nerves receive sympathetic fibers (gray rami communicantes) from the sympathetic ganglia. White rami are not found in the lower lumbar. Through their sensory fibers and the association of these with the various visceral centers within the cord, the lumbar nerves are able to affect the activities of the abdominal and pelvic viscera.

The sacral spinal nerves carry fibers of the white rami type, and these enter into the formation of the various plexuses in the pelvis. They carry also very many sensory nerve fibers, and these, being intimately associated with the sacral and the lumbar spinal centers, may affect the pelvic viscera markedly under certain circumstances.

The solar (epigastric) plexus receives the splanchnic and the vagus nerves. Sensory fibers from these nerves pass through the solar plexus, to be distributed with the viscero-motor and vaso-motor nerves. The phrenic or diaphragmatic plexuses are a continuation of the solar plexus. The suprarenal plexus receives fibers from the solar, the diaphragmatic and the renal plexuses. (Fig. 33.)

The renal plexus receives fibers from the solar plexus and the aortic plexus, and also from the lesser and the smallest splanchnic nerves.

The aortic plexus is a continuation of the solar plexus downward around the aorta; its fibers follow the arteries to their

destination, forming other smaller plexuses around the arterial trunks.

The hypogastric plexus receives fibers from the aortic plexus, from the lumbar ganglia of the sympathetic and from the first two sacral sympathetic ganglia. It contains no ganglia, and is merely an anatomical relationship; no physiological change occurs in the nerve impulses passing through it. Its downward extension forms the inferior hypogastric or the pelvic plexus, one on each side. These receive fibers also from the second, third and fourth spinal nerves (sacral group of white rami) and also from the sacral sympathetic ganglia. Ganglia have been described for the pelvic plexuses, but have not always been found by histologists.

The hemorrhoidal plexus receives fibers from the pelvic plexus, and also from the aortic plexus by way of the inferior mesenteric plexus; it receives branches from the internal pudic nerve also.

The visceral plexus receives fibers from the pelvic plexus and also from the third and fourth sacral nerves. The latter carry fibers of the white rami group and also many sensory nerve fibers.

The prostatic plexus receives fibers from the pelvic and the vesical plexuses, which carry both medullated and non-medullated nerves. The prostatic plexus contains several rather large ganglia (prostatic ganglia).

The vaginal plexus receives fibers from the pelvic plexus; these include many medullated nerves, sensory and perhaps also motor.

The uterine plexus receives fibers from the lower part of the pelvic plexus, but above the part which receives the sacral nerves.

The innervation of the generative organs as usually given is as follows:

Ovary. The nerves come from the ovarian plexus, which is an extension of the aortic plexus and the renal plexus. The larger number of the fibers pass with the blood vessels and terminate upon their walls; these are probably vaso-motor nerves. The ovary is very plentifully supplied with these. A smaller number of nerves pass to the walls of the Graafian follicles, and make a fine network around each of these. The fibrils have not been found to enter the follicle. A few nerves enter the reticular substance of the ovary and terminate by branching around these cells. Sensory nerve endings are found in the ovarian peritoneum, but not within the ovarian substance. (Fig. 23.)

Fallopian Tubes. These are supplied with nerves from both the ovarian and the uterine plexuses and nerve bundles.

Uterus. The nerves are derived from the hypogastric plexus, by way of the utero-vaginal plexus. Fibers directly from the hypogastric and the vesical plexuses, and from the pudic and the third and fourth sacral nerves also are distributed to the uterus. These nerves include vaso-motor, viscero-motor, and sensory fibers. They are distributed to the walls of the blood vessels, the uterine muscle, the uterine mucous membranes, and the uterine glands. The sensory nerves are distributed chiefly to the uterine muscle.

Vagina. The vaginal nerves are derived from the utero-vaginal and vesical plexuses and from the coccygeal and third and fourth sacral nerves.

Scrotum. The nerves of the coverings of the testes are derived from the ilioinguinal and the genital branch of the genito-femoral, and from the vesical plexus, the two superficial branches of the internal pudic nerve and the inferior pudendal branch of the small sciatic nerve.

Testes. The spermatic cord carries the fibers from the spermatic plexus and fibers from the pelvic plexus which are carried with the artery of the vas deferens.

Seminal Vescicles. These are innervated from the pelvic plexus.

Penis. Sensory nerves with Pacinian bodies and other nerve endings are found on the glans and the bulb. These are probably from the internal pudic nerve. The pelvic plexus carries the vaso-motor supply for the most part.

Prostate. The prostatic nerves are from the pelvic plexus.

EXPERIMENTAL DEMONSTRATION OF THE SPINAL CENTERS

In this complexity of nerve plexuses it would be impossible to trace fibers from their origin in the cord to the viscera innervated by anatomical or by histological investigations. An attempt has been made to determine the essential factors in the innervation of the pelvic organs by means of the reflex contractions of the spinal muscles of the lumbar and sacral regions.

CONDITIONS OF THE EXPERIMENTS

1. *Animal Experiments.* The animals were examined as to the existence of vertebral lesions, then anesthetized and the abdomen opened and the chosen viscera exposed. In the first

group of animals the muscular contractions were recognized by palpation. Sometimes several people held their fingers steadily, thus touching all parts of the spinal column from the mid-thoracic to the sacrum, and sometimes one person palpated along the spinal column gently, as in making examinations of patients for diagnosis. The electrodes were placed upon the viscus selected, and the muscular contractions noted. Sometimes the skin was removed from the back and the reflex contractions were observed. Sometimes the large superficial muscles of the back were also removed, and the contractions of the deeper spinal muscles observed.

Animals intended for various purposes of dissection and those examined as controls for lesioned animals served as subjects, as well as those killed for this purpose alone.

2. *Human Experiments.* In a number of patients in whom the pelvic examination is more or less painful, it is customary to inhibit over certain spinal areas in order that the pain of the examination may be diminished. In such cases, the irritation produced by the examination causes reflex muscular contractions in the areas corresponding to the location of the viscus which is diseased or which is being subjected to irritating conditions.

A study of the areas of reflex muscular contractions which follow pelvic injuries due to gonorrheal and other infections, or to injury in patients in whom lesions did not exist previously has given corroborative evidence and has verified the localization of the centers as given in the tables.

Findings. The results of these tests are given in the following table. For each item in this table not less than twenty observations were made, and in most cases the number of tests was much greater.

TABLES SHOWING LOCATION OF THE PELVIC SPINAL CENTERS—NEWLY BORN WHITE RATS

Organ Stimulated	Muscular Contractions	Other Effects
Ovary, including parovarium and fimbria or testes with epididymis and spermatic cord.	Spinal and intercostal muscles of the 4th thoracic to the 4th lumbar segments. Anterior neck muscles, shoulder muscles, and diaphragm.	Accelerated heart beat and respiratory movements. Increased peristalsis.
Bladder and horns of uterus or prostate.	Spinal and intercostal muscles of the 10th thoracic segment to and including the tail, thigh and leg muscles and diaphragm.	Micturition and defecation. Increased peristalsis.

TABLES SHOWING LOCATION OF THE PELVIC SPINAL CENTERS— ADULT WHITE RATS AND GUINEA PIGS

Position of Electrodes	Deep Spinal Muscles	Superficial and Medial Spinal Muscles	Other Skeletal Muscles	Other Effects Produced	Location of Spinal Governing Centers
Ovary.	Multifidus spinae, spinalis dorsi on both sides of the 6th to the 13th thoracic segments, levatores costarum chiefly of the same side.	Serratus posticus inferior, chiefly of the same side.	Diaphragm, anterior cervical muscles.	Peristalsis increased in stomach. Sometimes in the intestines. Sometimes shoulder muscles, especially on the same side.	Seventh to 13th segments.
Parovarium.	Multifidus and spinalis, 9th to 13th thoracic segments.	None or very slight.	Anterior cervical muscles sometimes.	None, or a very slight increase in peristalsis.	Probably associated with ovarian centers.
Fimbria and upper half inch of tube (horn of bicornate uterus).	Multifidus and spinalis, 9th to 13th thoracic segments, levatores costarum, chiefly on side stimulated.	Serratus posticus inferior and quadratus lumborum, chiefly on side stimulated.	Diaphragm, anterior cervical muscles.	Peristalsis increased in the intestines and sometimes in the stomach.	Probably associated with ovarian center.
Horns of uterus below the upper half inch.	Multifidus, iliocostalis, thirteenth thoracic to fourth lumbar.	Erector spinae, quadratus lumborum, serratus posticus, chiefly on the side stimulated; latissimus dorsi, longissimus dorsi.	Diaphragm, adductors of the thigh and quadriceps. Abdominal muscles, both anterior and posterior.	Peristalsis increased in intestines; sometimes micturition and defecation.	First to 4th lumbar segments.
Cervix.	Multifidus, ilio-costalis, 1st to 5th lumbar.	Erector spinae, quadratus lumborum, serratus posticus inferior.	Adductor of the thigh; quadriceps. Abdominal muscles.	Micturition and defecation usually.	First to 4th lumbar segments.
Vaginal walls.	Contractions slight or lacking, lower lumbar and sacrum.	Latissimus dorsi, lumbar region, quadratus lumborum.	Gluteal muscles, flexors of thighs, perineal muscles.	Micturition usually.	Sixth lumbar to sacral segments.

TABLE SHOWING LOCATION OF THE PELVIC SPINAL CENTERS—ADULT WHITE RATS AND GUINEA PIGS—*Continued*

Vaginal orifice.	Muscles over sacrum.	Muscles over sacrum.	Gluteal muscles, flexors and adductors of thighs, perineal muscles.	Micturition usually.	Sixth lumbar and sacral segments.
Testes.	Multifidus and spinalis on both sides alike.	Serratus posticus inferior, most marked on side stimulated.	Neck muscles, abdominal muscles, pyramidalis, cremaster.	Retraction into abdomen.	Tenth thoracic to 1st lumbar.
Spermatic cord.	Slight or no contractions.	Slight or no contractions.	Abdominal muscles, pyramidalis, cremaster (by direct stimulation).	Retraction of testicle into abdomen. Increased peristalsis in jejunum.	Probably with center for the testes.
Seminal vesicles.	No reaction.	No reaction.	No reaction.	No reaction.	Not to be determined.
Cowper's glands.	Slight contractions of all abdominal muscles, muscles over the sacrum.			Micturition, erection, sometimes.	Fifth to 7th lumbar segments.
Prostate.	Multifidus and spinalis.	Serratus posticus inferior, quadratus lumborum.	Abdominal muscles, adductors of thigh, neck muscles.	Erection sometimes, retraction of testicles, increased peristalsis.	Third to 7th lumbar segments.
Glans.	Muscles over sacrum.		Abdominal and cervical muscles, adductors of thighs.	Micturition, defecation, increased peristalsis.	Third to 7th lumbar segments.
Top of bladder.	Multifidus and spinalis, throughout lumbar and sacral region.	Quadratus lumborum, latissimus and longissimus dorsi.	Abdominal muscles, thigh muscles generally.	Micturition (direct stimulation).	First to 6th lumbar segments.
Neck of bladder.	Muscles over sacrum.		Thigh and leg muscles, abdominal muscles.	Erection and sometimes defecation. No micturition.	Third to 7th lumbar segments.
Rectum.	All muscles of lumbar and sacral segments.		Cervical and abdominal muscles.	Defecation (direct stimulation).	Second to 5th lumbar segments.

ADULT DOGS AND CATS

Position of Electrodes	Deep Spinal Muscles	Median and Superficial Spinal Muscles	Other Muscles	Other Effects Produced	Location of Spinal Governing Centers
Ovary.	Multifidus and spinalis both sides, 8th to 12th thoracic segments; levatores costarum chiefly of same side.	Serratus posticus inferior, more marked on same side.	Anterior cervical muscles, diaphragm.	Peristalsis increased in stomach, sometimes in jejunum.	Tenth to 12th thoracic.
Parovarium and Fimbria.	Contractions less marked but otherwise the same as for the ovary.				
Horns of uterus.	Multifidus, ilio-costalis, levatores costarum, 1st to 4th segments.	Erector spinae, serratus posticus inferior, longissimus dorsi, 1st to 4th lumbar segments.	Quadratus lumborum, diaphragm, adductors and flexors of thighs.	Defecation, increased peristalsis in small intestines, sometimes micturition.	First to 5th lumbar segments.
Cervix of uterus.	Multifidus, ilio-costalis, intertransversales of 2d to 5th lumbar segments.	Erector spinae, quadratus lumborum, serratus posticus inferior.	Diaphragm, anterior neck muscles, adduction of thighs, sometimes all thigh and leg muscles.	Defecation and micturition sometimes.	Third to 5th lumbar segments.

ADULT DOGS AND CATS—*Continued*

Vaginal walls and orifice.	All muscles over the sacrum.		Gluteal and perineal muscles, leg and thigh muscles.	Micturition sometimes.	Fifth lumbar to sacrum.
Top of bladder.	Multifidus and spinalis, 3d to 6th lumbar.	Quadratus lumborum and latissimus dorsi.	Abdominal muscles.	Micturition (direct stimulation).	Second to 4th lumbar segments.
Neck of bladder.	All muscles of lower lumbar and sacral segments.		Muscles of thighs, legs and feet.	Erection in males.	Fourth to 7th lumbar segments.
Rectum.	All muscles of lower lumbar and sacral segments, thigh and leg muscles.			Defecation (direct stimulation), micturition, erection in males.	Fourth to 6th lumbar segments.
Testes.	Multifidus, ilio-costalis and spinalis.	Serratus posticus, most marked on side stimulated.	Neck muscles, diaphragm, pyramidalis, cremaster.	Retraction, sometimes erection.	Tenth to 12th thoracic.
Epididymis and spermatic cord.	Slight or no contractions.		Rectus abdominalis, cremaster, pyramidalis.	Retraction, rarely erection.	Probably with center for testes.
Seminal vesicles and Cowper's glands.	Muscles over sacrum.		Abdominal muscles, thigh and leg muscles.	Micturition, erection usually.	Third to 6th lumbar segments.
Prostate.	Multifidus, spinalis, ilio-costalis.	Serratus posticus inferior.	Quadratus lumborum, adductors of thigh.	Erection, retraction of testicles, increased peristalsis.	Third to 5th lumbar segments.
Glans.	All muscles over the sacrum.		Leg and thigh muscles.	Defecation.	Fourth to 7th lumbar segments.

PROBABLE LOCATION OF CENTERS IN THE HUMAN SUBJECT

Organ	Location of Hypersensitive Areas and Muscular Tension	Location of Spinal Governing Centers
Ovary, parovarium and fimbria.	Neck, iliac fossae, 9th to 12th thoracic segments.	Ninth to 11th thoracic segments.
Fallopian tubes and uterus.	Lower thoracic and upper lumbar segments, thighs, neck and upper arms, suboccipital muscles.	First to 3d lumbar segments.
Bladder and rectum.	Lumbar and sacral regions, lower abdominal region, thighs.	Second to 5th lumbar segments.
Testes, epididymus.	Lower thoracic and upper lumbar region, neck muscles.	Tenth to 12th thoracic segments.
Prostate.	Lumbar and lumbosacral region, thighs and gluteal muscles.	First to 3d lumbar segments.

A STUDY OF THE SYMPATHETIC GANGLIA

During the last few years special study has been made of the structure and development of the sympathetic ganglia, especially of the gangliated cord, and the semilunar ganglia of the solar plexus. The development has been studied in the rabbit and the chick during the process of differentiation and migration; in the cat and dog during the development from the fetus of less than an inch to the adult; in the human subject material has been secured from the fetus of ten weeks and at several stages to the full term fetus and newly-born child. This work, begun in the Pacific College laboratories, has been carried on since 1914 in the laboratories of the A. T. Still Research Institute. For the human material studied we are indebted to many osteopathic physicians of California and Illinois.

DEVELOPMENT

The nerve cells which make up the ganglia of the lateral chain are derived from the nerve cells of the column lying in the posterior part of what ultimately becomes the posterior horns of the spinal cord. These nerve cells migrate, passing forward toward the intervertebral foramen. Some of the cells stop in the foramen, and these become developed into the sensory ganglia. Other cells continue their journey into the body cavity and come to rest in the localities ultimately occupied by the adult sympathetic ganglia. This process can be recognized in the chick and the rabbit; the cells can be found in prepared slides in all stages of this development. Cells from the group which are to become the lateral chain ganglia are to be found placed anteriorly to these in the direction of the prevertebral ganglia; no doubt these do enter into the formation of the semilunar ganglia of the solar plexus and other sympathetic ganglia, though their later progress has not been certainly traced. Certain histologists have described the origin of the semilunar and other sympathetic ganglia from the connective tissue cells of the locality in which they are found. The pigmented cells of the medullary portion of the suprarenal capsules and certain other structures of the endocrine mechanism have been variously associated with

these sympathetic anlagen. Much investigation yet remains to be made before the definite relations of these structures are known.

ADULT STRUCTURE

The ganglia consist of nerve cells, nerve fibers, and supporting tissues. According to our studies, after the structure has become fairly well completed each nerve cell lies within a pericellular basket which is composed of nerve fibers. (Figs. 31, 32, 33.)

This basket is made up partly of very fine dendritic fibrillae from the cell itself, and these interlace in a very complicated manner with fibers which are probably always, and are certainly usually derived from the cerebro-spinal system. (Fig. 34.) The medullated fibers from the white rami communicantes lose their sheaths within the sympathetic ganglion and then give off collaterals which branch and interlace with the dendritic processes of the sympathetic cells. The number of white rami fibers which may affect any one cell is not known. (I have seen three different fibers entering into the formation of one pericellular basket.) It is not known also how many sympathetic cells may be governed by one white rami fiber; five branches from one such fiber have been counted within one sympathetic ganglion. The tremendous complexity of these rami fibers and of their relationships may be appreciated when it is remembered that the white rami fibers and also the cells of the sympathetic ganglia may be counted by dozens for each spinal segment. The matter is still further complicated by the fact that the white rami fibers often give off a collateral to one sympathetic ganglion, and then pass either upward or downward in the cord of the lateral chain, or toward the prevertebral plexuses.

The ganglion is enclosed in a connective tissue capsule, from which delicate trabeculae pass into the nervous material, supporting and separating the blood vessels and to a slight extent the various bundles of fibers entering the ganglion. The blood vessels are very small and have nerve endings upon their walls; these are probably vaso-motor nerves.

The axons of the sympathetic cells are not medullated. They pass directly to their destination and end in motor plates upon the walls of the viscera or the blood vessels, or in fine branching fibrillae among secretory or other active cells of the viscera, the bone marrow, or the skin. According to Howells, a sympathetic

axon or nerve very rarely forms a synapsis with another sympathetic cell, if indeed such a relation ever occurs.

Cells which appear to be sympathetic nerve cells are found within the tissues of certain organs. The cervix of the uterus, the heart muscle and the intestinal wall are some of these localities. These cells may be solitary or may lie in groups of two or three or more, or may be arranged in the form of very small ganglia.

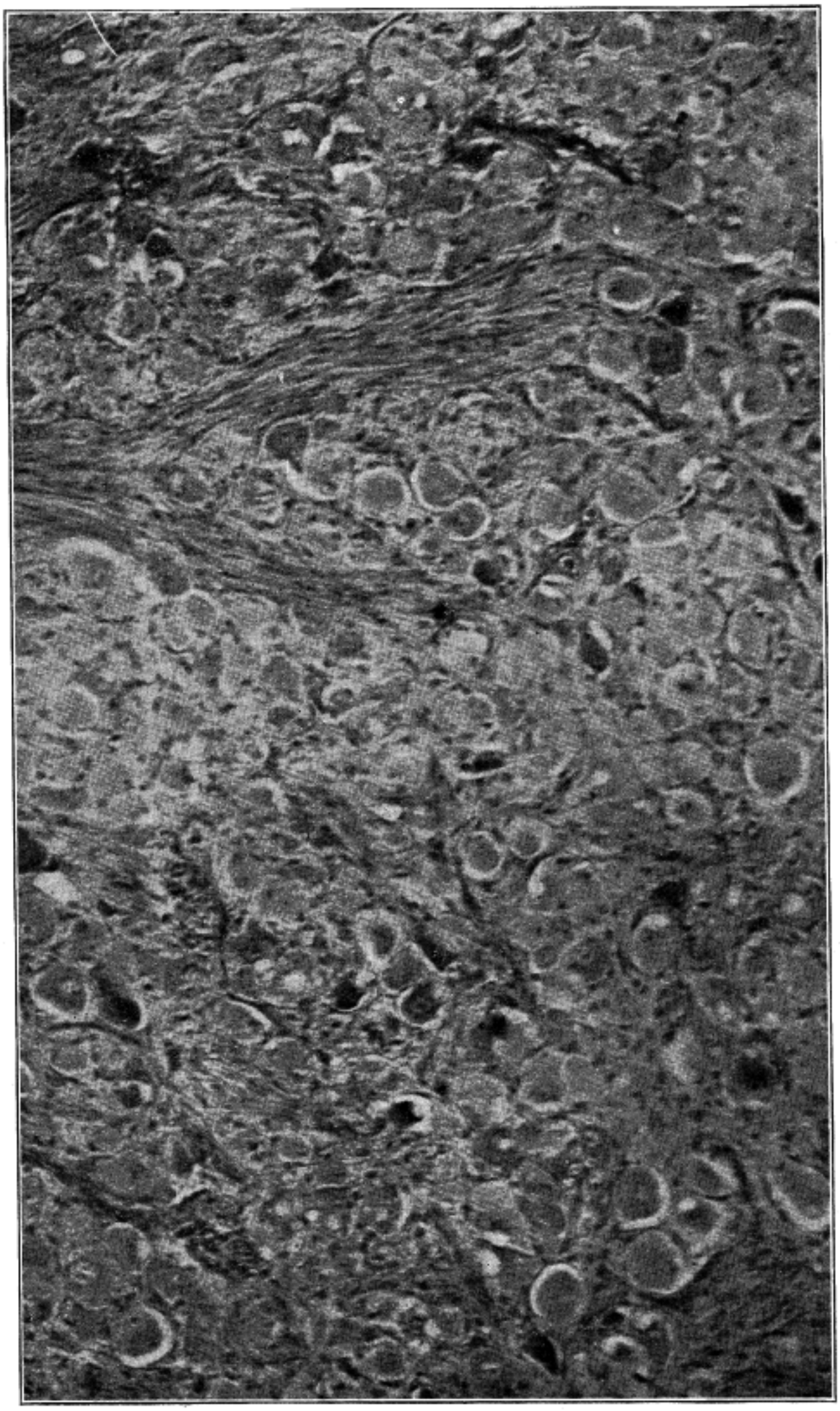

Fig. 31

Fig. 31. Microphotograph, low power, section cut obliquely to the direction of the entering fibers. Note the branching of the bundles of nerve fibers, with the formation of the pericellular baskets. Ganglion stellatum of dog.

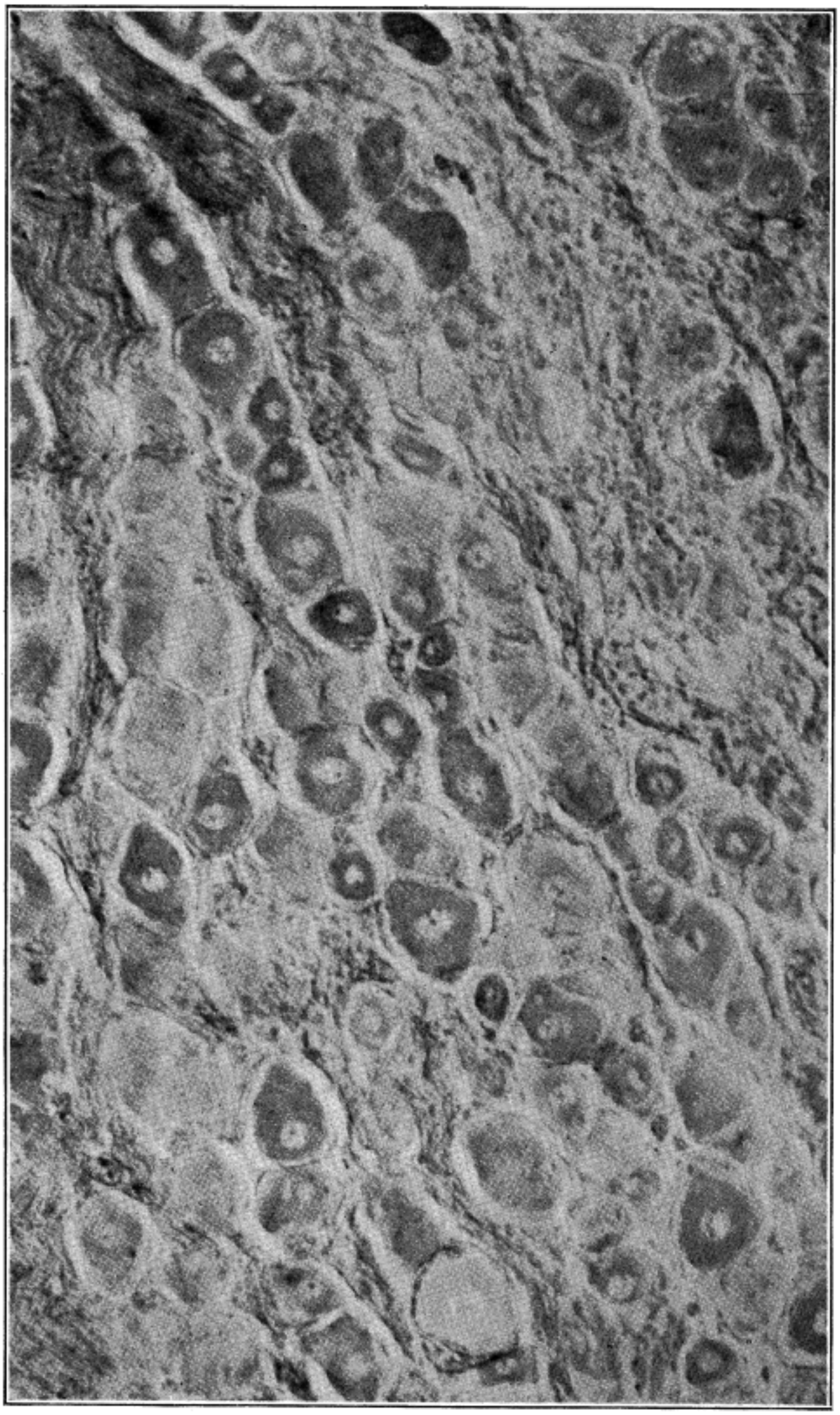

Fig. 32

Fig. 32. Microphotograph, higher power, section cut parallel with direction of nerve fibers. Ganglion of sympathetic chain, from dorso-lumbar region of dog.

Note the formation of the pericellular baskets, and the variations in size and in staining reactions of the different cells.

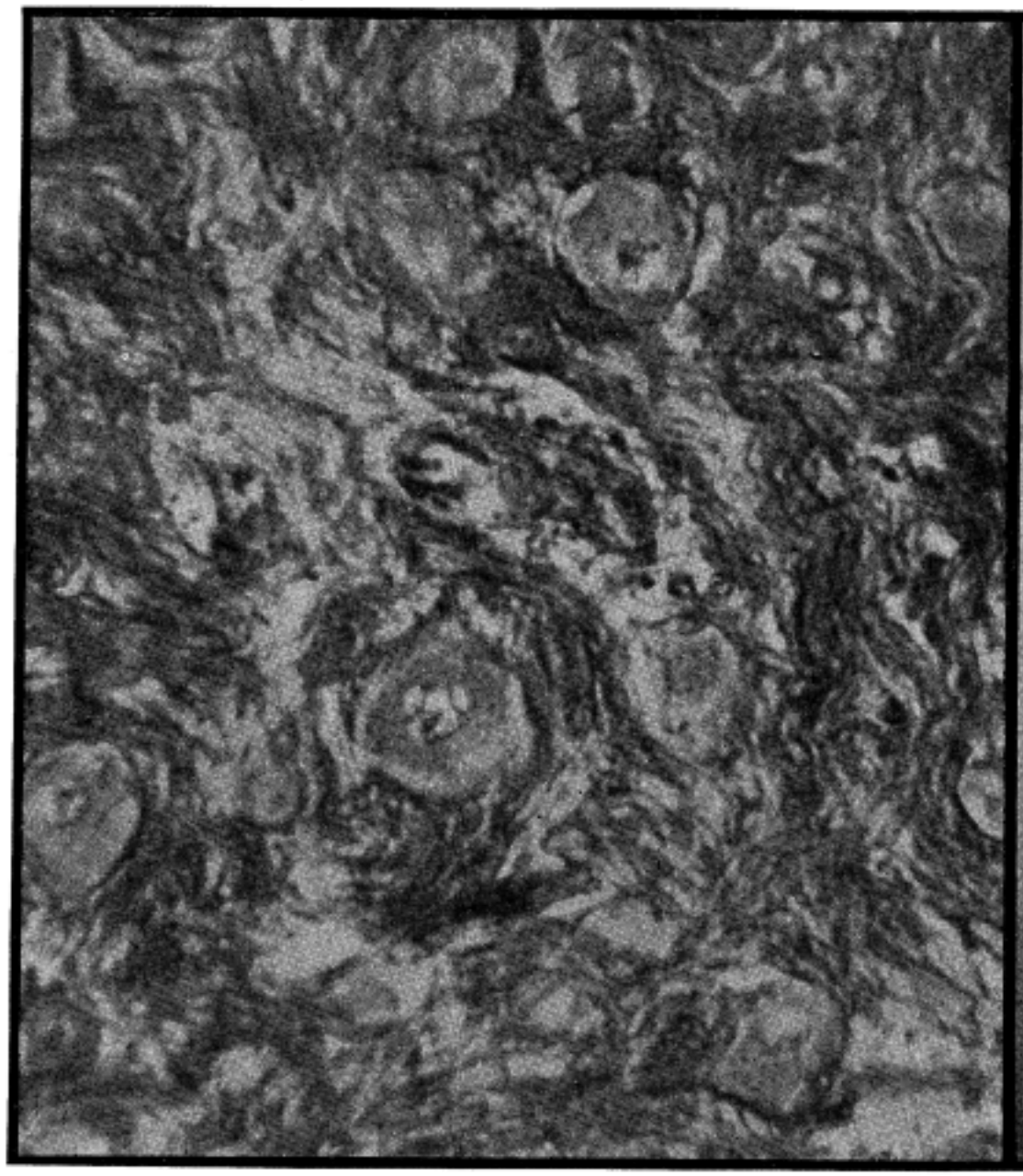

Fig. 33

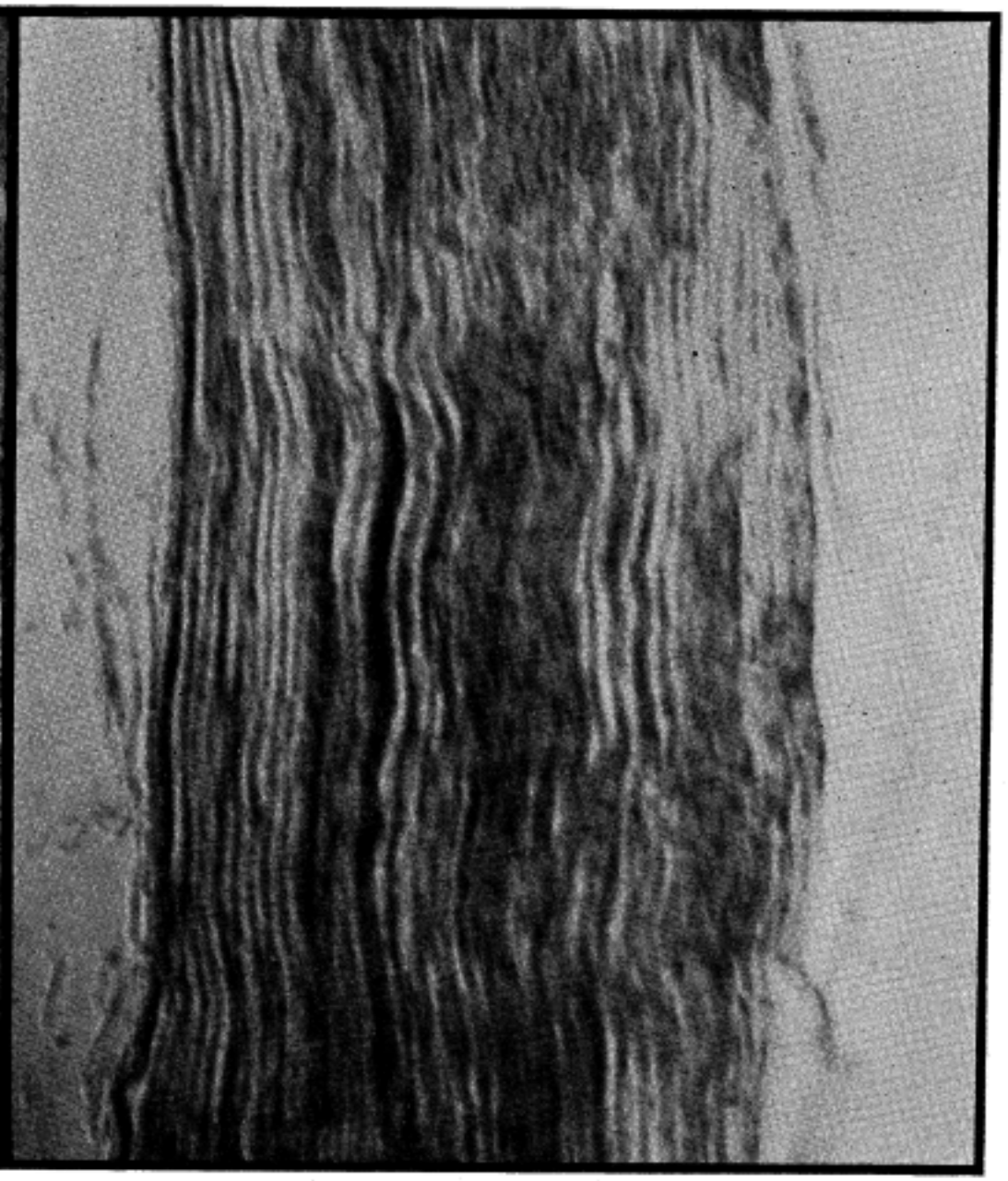

Fig. 34

Fig. 33. Microphotograph, still higher power; section cut almost at right angles to direction of fibers. Semilunar ganglion of solar plexus of dog. Note the pericellular baskets and the branching of the individual fibers. (These show distinctly in the original slides.)

Fig. 34. Microphotograph, same magnification as in Fig. 33. Longitudinal section of nerve bundles connecting the ganglia of the sympathetic chain. The fibers which appear dark are non-medullated; the fibers which appear light have their medullary sheaths, and are therefore derived from the central nervous system. Since this was adjacent to the ganglion shown in Fig. 32, the medullated fibers must have originated in the lateral horns of the lower thoracic spinal cord.

MATERNAL VERTEBRAL LESIONS A POSSIBLE CAUSE OF FETAL ABNORMALITIES

The possibility that vertebral lesions in the mother might act adversely upon the circulation of the blood through the uterus, and thus interfere with the nutrition of the embryo and fetus is suggested by the fact, already known, that such lesions do interfere with the circulation of the ovaries and the uterus itself. It seems also to be true, from clinic evidence, that a tendency to abortion may be due in some cases to lower thoracic or to lumbar lesions. Whenever it has been possible to do so, the products of conception in lesioned female animals have been examined. In certain instances this has not been possible, for obvious reasons. The number of young animals examined is too small to permit any conclusions to be drawn, yet the observations are, to some extent, significant. Further study should be made of the clinic history of mothers of whom deformed or monstrous children are born, with especial reference to the existence of vertebral lesions, or of slight accidents which might have exerted a similar influence upon the innervation of the uterus itself, or of the blood vessels of the uterus and ovaries. It is hoped that this report of the few animals examined may increase the more careful study and discussion of these cases by osteopathic obstetricians.

PREVIOUS INVESTIGATIONS

The occurrence of deformed children has aroused a lively interest among mankind probably from the beginning of the race. Many fanciful and naive explanations have been given for these unfortunate occurrences. Even so late as the time of Martin Luther monstrosities were supposed to result from intercourse with animals, or with Satan. The adverse action of malignant "spirits" which gained entrance into the maternal organism through fright or anger of the mother was supposed, later, to account for the monstrous or deformed child. The mother is thus held to be at fault. An interesting feature of this idea is that the mother might localize evil, by putting her own hand upon her own body in some place whose injury is supposed to be less injurious. For example, having seen some unpleasant sight, which

would be supposed to act adversely upon the unborn, the prospective mother would very quickly rub her own foot, in order that the foot of the child, rather than its face, might be deformed. A great mass of therapy based upon this idea of the influence of the mother's imagination upon the fetus is described in various old books—and in some not so old. To this day these ideas are passed from one generation to another, by whispered words of mouth, and much harm is constantly being done by the frightfulness of the doctrine, as untaught young wives and mothers are deceived by ignorant older women.

One of the earliest attempts at a saner explanation of the occurrence of monsters was made by James Augustus Blandell in 1729, in a book called "The Power of the Mother's Imagination Over the Fœtus Examined." He shows the anatomical and embryological impossibility of any direct influence of the imagination, apart from physiological states, in a number of cases which he describes in considerable detail. Without attempting any exact explanation of the production of monsters, he lays down a foundation for the later study of teratology which is exactly in harmony with recent ideas. (The little volume is most quaintly worded, and includes a reference to the "ring-striped and streaked" cattle of Jacob, in which he imposes the dream of Jacob in a subsequent chapter upon the events as usually quoted, and by this means he makes the occurrence of the "ring-streaked and striped" in the flocks a matter of heredity from males miraculously provided.)

During the eighteenth, nineteenth and beginning of the twentieth centuries the problems of teratology have been attacked from the standpoint of animal and vegetable experimentation. In 1759 Caspar F. Wolf published "Theoria Generationis," in which the development of the embryo and some discussion of the possible causes of disturbances in development are to be found. The elder St. Hilaire incubated eggs in unusual positions and produced chick monsters. Pare, Darester, Panin, Warynski, Foe, Lombardini, and a number of other men of the eighteenth and the nineteenth centuries produced monsters among chicks and certain other fowls by exposing the eggs to adverse influences—changes of temperature, injection of substances into the shells, varnishing the shells, exposure of the eggs to volatile drugs, such as alcohol, ether, chloroform, mercury, and by producing many other abnormal environmental influences. McClendon exposed eggs to an excess

of carbonic acid gas and produced monsters. Stockard experimented with guinea pigs, giving them alcohol fumes to breathe, and found that in forty matings of alcoholic guinea pigs with normal or alcoholic guinea pigs, thirty-five resulted in no pregnancy, while of the fifteen pregnancies, nine gave still-born young, one pig was grossly deformed, and all the living young, five in number, were smaller and weaker than normal. Nice repeated this experiment upon mice, with variable results. A long list of men and women have experimented with fishes, reptiles and invertebrate animals; it seems to be without exception the case that no change from the normal environment can be supposed to be harmless to the developing young. Experiments with drugs upon mammals indicate that no drug capable of acting upon the maternal organism can be held harmless to the developing child. It also seems to be shown that any adverse influence acting in the very earlier days of pregnancy produces the greater deformity. These are biological principles, and hold true for all living organisms, in their broader application.

In the human pregnancy, considerable study has been made of the relationship between disease of the pelvic organs and the occurrence of monsters. Franklyn P. Mall has examined some hundreds of embryos and fetuses from ectopic and uterine gestations. He found 96% of tubal embryos abnormal—deformed—while of uterine embryos, though from abortions, only about 7% are abnormal. He attributed this to the fact that tubal embryos are, almost from the beginning, placed under an abnormal environment.

It is known that women who work in lead have frequent abortions and that monsters and deformed and weakly children are more frequent among female lead workers than among other groups of female workers. Women whose husbands work in lead, but who do not themselves do such work, have a larger number of abortions, still-births and deformed and weakly children than do women whose husbands are not engaged in occupations with poisonous conditions. This shows the poisoning of the sperm cells.

Even more important, though less striking, is the occurrence of slight evidences of defective development. The structural perversions included in the term "stigmata of degeneracy" illustrate some of these peculiarities. At least a few of the various

malignant tumors appear to be due to such defective developmental conditions. Children who are mentally or morally defective have always physical and cerebral developmental defects. Hemorrhages into the central nervous system, while hardly to be included among the developmental defects, are yet important factors in modifying the life of the unfortunate subject of this accident. Weaklings are sometimes born, who owe their feebleness to deficient nutrition before birth. Probably all of these conditions, and many others, require investigation from the standpoint of the maternal circulatory disturbances and the variations in the quality of the maternal blood.

Many gynecological diseases are supposed to be either directly or indirectly the result of malformation. Certain disturbances of refraction in the eyes are due to malformation. Visceral disease is often associated with malformation. Malformation of the brain is present in all idiots and imbeciles in recognizable degree. It is often recognizable, and probably always present, in the feeble-minded, morons, and essential criminals. Deformities of the body lie at the basis of moral obliquity unquestionably. These deformities may not be sufficiently pronounced to interfere with a moderate amount of health, or even with a fairly attractive face and form. Yet careful autopsies show that in nearly all of the individuals whose lives are especially unfortunate or especially wicked a variable number of stigmata of degeneracy are present. These represent slight deformities, and are known to be associated in most cases with deformities of the brain.

Examination of Lesioned Animals.

Structural perversions of the fetus must be classified into two chief groups: 1. Abnormal states which are due to deficient development; in such cases the tissue concerned has ceased its development at a certain time, or it has followed an aberrant line of differentiation. 2. Abnormal states which are due to the influence of some abnormal agency acting upon tissues which have, at an earlier time, been engaged in normal development. The first class may be hereditary or may be due to any one of several pathological conditions acting upon the germ cells, or upon the fertilized ovum. The second class may be due to nutritional, toxic, infectious or mechanical agencies acting on the embryo or fetus.

There seems no doubt that any factor which modifies the circulation or the nutrition of the ovarian or the uterine tissues is potentially a cause of fetal deformity. That the vertebral or innominate lesion is such a factor is amply demonstrated by experimental and clinical evidence. That deformities do follow such lesions to some extent is indicated by these few observations made upon a few animals. To what extent these lesions may be responsible not only for monstrosities, but also for slight perversions of structure is a matter for future investigation.

The few lesioned animals which have been kept under observation during pregnancy have given several instances of congenital defect. When the sterility of these animals is remembered, the proportion of defective young becomes even more striking.

Only one lesioned cat became pregnant. A missed pregnancy, with retention of an inspissated fetus, changing in form to the cylinder usual in the human fetus under such circumstances, was the result of that pregnancy. (Fig. 49, 52.)

In one group of white rats only one became pregnant after the lesions were produced. Extra-uterine gestation with death of the mother was the result of that pregnancy. The embryo was not to be found.

No guinea pig became pregnant for several months after the lesions were produced. When pregnancy did occur, a normal course was the exception.

One guinea pig died, and two fetuses, apparently long dead, were found.

Another gave birth to two pigs, one dead at birth, the other dead very soon after birth. The still-born pig had two placentas and two umbilical cords, passing to a single umbilicus. This pig was much larger than normal pigs at full term. In the placenta of the other pig a tumor was found, hard and apparently fibroid. Cerebral hemorrhages were found in both pigs.

A malignant neoplasm, apparently associated with the death of a third embryo of this same pregnancy, was found within the uterus of the mother at autopsy ten days after the birth of these abnormal babies.

A third guinea pig died a few days after the birth of her two still-born babies. The cause of death could not be found.

A fourth pregnant pig was anesthetized and killed. One abnormally large pig was found. Two distinctly discoid tumors were found in the uterus above the placental site which were composed of tissue resembling placenta of an early stage of development. An irregular mass below the placental site of the living fetus appeared to be composed of thin-walled cells undergoing rapid multiplication. Many white blood cells were present in the mass.

Previous Notes

The following earlier observations may be of interest in this connection:

1. A human fetus was sent for examination. It appeared to be about five months gestation, though the mother's history gave three months as its age. A professional abortionist had given the mother electrical "treatments" four times within a week, expressing surprise at the number required to produce expulsion. The mother then decided to avoid the abortion, and placed herself in the care of a reputable osteopathic physician. The process of labor could not then be stopped, and the fetus was born within a few hours after she applied for treatment.

The nervous system was put up in paraffine and stained with iron hematoxylin. The interesting factor is the presence of numerous hemorrhages in the lumbar part of the spinal cord. (Fig. 35, 36.) It is conceivable that an amount of electrical irritation insufficient to produce death might have produced slight hemorrhages in the nervous system and that various paralyses might result from such a condition. (This fetus was made the subject of a report by C. A. Whiting, in "Public Sanitation and Other Papers.")

2. A cat was sent in to be killed, on account of long and painful labor. Examination showed shoulder presentation. A lesion was found in the upper lumbar area; muscular contraction, vertebral rigidity and hypersensitiveness were all present. The origin of the lesion was not known. After anesthesia the abdomen was opened, and a second fetus, also with the shoulder presenting, was found. A third fetus was in normal position.

3. The presence of whitish crystals of calcium carbonate in human placentas is supposed to be due to deficient oxidation of the placental tissues; this, in turn, is probably due either to deficient hemoglobin in the maternal blood, or to inefficient

circulatory conditions, including cardiac difficulties, especially valvular lesions. The presence of the crystals is not incompatible with the development of a fairly normal child, unless they are so plentiful as to interfere with the placental functions; in this case the fetus is poorly nourished and poorly supplied with oxygen, and the waste products of fetal metabolism are not efficiently carried away.

These granules were found present rather conspicuously in the placenta of a primapara in whom an innominate lesion, with secondary lumbar curve, had been present for some years. The child was poorly nourished and thin, but lived and became healthy and strong after two or three months.

4. Anencephalus or Pseudocephalus Monster. A full term fetus was brought for examination. The body was the shape and size of a normal child at term, male, well developed except for the head. No external evidence of spina bifida was present. The limbs, hands and feet were all perfect. Only one testicle was descended; not an abnormal condition.

The face was normal up to the eyes, which also were apparently without fault. There was no forehead, and the supraorbital arches marked the upper limit of the face. Eyebrows were indicated by faint lines. The horizontal portion of the frontal bone was present and appeared to be perfect. The vertical portion was entirely absent, except for a thin and narrow ridge which united it with the greater wing of the sphenoid.

The squamous portions of the temporal bones were absent, except for a thin and narrow wing which arched over toward the central portion. This appeared to mark about the region of the petro-squamous suture.

The occipital bone was deformed. The basilar process seemed normal, as did also the foramen magnum. The crucial ridge, or about that region, marked the upper extent of the normal part of the occipital bone; above this the flat thin plate of bone turned forward, making a wing-like projection continuous with that of the temporal bone.

The base of the skull appeared normal. The petrous portion of the temporal bone, the sella turcica and the fossae appeared normal for the full term fetus.

A mass of membranes filled with blood, within which some delicate tissues could be recognized with difficulty, occupied the

shallow cup formed by the ridges of bone around the base of the skull. The portions of this which were set aside for microscopic examination were, unfortunately, lost.

The palate was cleft; there was no hair lip. The lung floated in water. The heart protruded through the diaphragm into the thoracic cavity, protected by the pericardium; possibly also there may have been some of the central tendon of the diaphragm included in the sac which carried the heart. The diaphragm was deficient, having scanty muscle and either an absent or an extremely thin central tendon.

The stomach was of the hourglass type. The intestines and kidneys seemed normal; the lobular appearance of the fetal kidney was not more marked than is usual in the newly born infant. The laminae of the third, fourth and fifth lumbar vertebrae and of the sacrum were lacking; this was not associated with tumor formation.

The history of the pregnancy was normal, except that during the first or second month a nervous shock occurred, the nature of which bore a striking relation to the character of the deformity. No indication of heredity. uterine inflammation, syphilis, or any other maternal cause of fetal malnutrition could be secured. The effects of the shock passed away, after some symptoms of impending miscarriage, and pregnancy followed an uneventful course thereafter. Birth was normal, and the monster was born alive. It cried vociferously and died a little later. The cause of death was not apparent at the autopsy; monsters of this type rarely live longer than a few minutes.

The shock which may be supposed to be responsible for the condition evidently occurred at the time of the development of the bones of the skull, and of the beginning enlargement of the cerebrum. The manner in which the shock to the maternal nervous system acted upon the embryo is not clearly understood. It is known, however, that intense emotional disturbances affect the circulation and the pressure of the blood, the secretions of the glands of the entire body, and the activity of the non-striated muscles of the digestive and the reproductive tracts. Either one of these three factors might be responsible for the injury to the embryo, or there may be other factors concerned which are not included in our present knowledge of the subject.

No conclusions can be drawn from this scanty evidence. It appears, however, that some relationship between maternal vertebral lesions, pelvic congestion, and fetal abnormalities is sufficiently probable to warrant further study.

The investigation of the place of vertebral lesions in the cause of monsters and of the birth of young which have less easily recognizable deformities is only just begun. Further investigations need to be made. The study of a larger number of women who give birth to deformed young is urgently necessary, in order that animal experimentation may be co-ordinated with the results of clinical investigation. In medical literature monsters are very often reported as the result of pregnancies which are, from the maternal standpoint, perfectly normal; but this literature takes no account of vertebral lesions. It thus becomes imperative that the clinical study of this subject be made by the osteopathic profession.

Fig. 36

Fig. 35

Fig. 35. Microphotograph, low power; longitudinal section of fetal spinal cord.

H. Hemorrhage.

Fig. 36. Microphotograph, higher power; same section.

H. Hemorrhage.

B.V. Blood vessel.

A STUDY OF A UTERINE NEOPLASM IN A GUINEA PIG

The guinea pig from whom this tumor was taken is that called "Guinea Pig No. 3," in the series described by Dr. Avis G. Hoskins in this number.

Briefly, this pig gave birth to normal young early in February, 1917. The birth was normal, the babies lived, and no signs of anything abnormal were at any time noted until the pregnancy terminating July 2, 1917.

After weaning the babies born in February, the pig was added to the lesioned series. After having a lesion produced involving the first and second lumbar vertebra, the pig was placed in a cage with other females, normal and lesioned, and with a normal male. She did not become pregnant until in May, 1917. Two babies were born, July 2, 1917; one deformed and dead, and one which died very soon after its first breath. The mother did not recover well; seemed very ill, and was barely alive on July 12, when chloroform anesthesia terminated her life. The autopsy findings included a tumor involving the uterine wall, and supplied with very large and tortuous blood vessels.

This tumor was removed and prepared for microscopic examination.

Examination of teased specimens show that the center of the mass is occupied by a vessel containing blood, which is disintegrated and appears to be undergoing organization. Around this lies a tissue which appears to include solid cords of large cells, with deeply staining nuclei. There are masses of coagulated blood among these cords of cells, and some thin fibrils of what appears to be connective tissue.

The slides were cut of varying thickness and were stained with hemotoxylin and eosin. The center of the mass is composed of a blood clot, partly organized and undergoing invasion by the tumor cells. (Fig. 37.) Around this central mass lie small masses of partly disintegrated and organized blood. Cords of cells arranged in solid masses appear to originate from among these masses of blood, and to be growing centrally into the blood clot

and peripherally into the uterine wall. (Fig. 38.) These cords of cells appear to be of embryonic type and include nuclei showing karyokinetic figures. The supporting fibrils are of connective tissue, mostly of an adult type. The red blood cells within the vessels have no nuclei.

The general appearance of this tumor, the gross appearance of the uterus and its blood vessels, the illness of the guinea pig for the ten days after birth of the two abnormal pigs and before she was killed all indicated malignancy. Two possibilities are to be considered. (1) That the tumor is homologous with human carcinoma, and has arisen from the uterine glands as the result of the presence of the irritation of the remnants of a dead embryo or its membranes, or (2), that it is homologous with human chorio-epithelioma, and has arisen from chorionic remnants of a dead embryo.

Microphotographs have been made of this tumor and of human carcinomas and epitheliomas. (Fig. 37 to 48.) The history of each of these is given upon the page opposite the photograph illustrating the section.

This tumor is especially interesting in that it illustrates a borderland between two classes of neoplasm. One class of neoplasm originates from the tissues of the individual whose body it invades. This has been called autochthonous. If this tumor be a carcinoma, which is one of the autochthonous neoplasms, then it has originated from the uterine glandular tissue, at the seat of an irritative lesion of some sort, probably the remnant of a dead embryo.

The other class of neoplasm originates from the tissues of another individual than that whose body it invades; this has been called heterochthonous. If this tumor be a chorio-epithelioma, then it has originated from the chorion and decidua of a dead embryo; it thus should be considered heterochthonous.

In either case, the tumor is of an intensely malignant nature; it has been the result of the death of an embryo, and it bears marked resemblance to human cancer found invading the uterus.

For the material illustrated, and for other material which has been valuable for study, we are deeply indebted to Drs. A. C. Condon and C. R. Atzen, of Omaha; Drs. J. B. Littlejohn and A. E. Linander, of Chicago; Dr. Lura B. Nelson, of Hollywood and Dr. T. B. Gotham, of Elsinore, California; Dr. Clara Wer-

nicke, of Cincinnati; Dr. F. I. Furry, of Cheyenne, and Drs. A. E. Perry, Fannie Shaffer, M. L. Burns, R. D. Emery, W. J. Vollbrecht, F. E. Clark, A. S. Clark, C. A. Whiting, C. H. Phinney and Olive Clarke, of Los Angeles; Dr. Mary S. Croswell, of Maine.

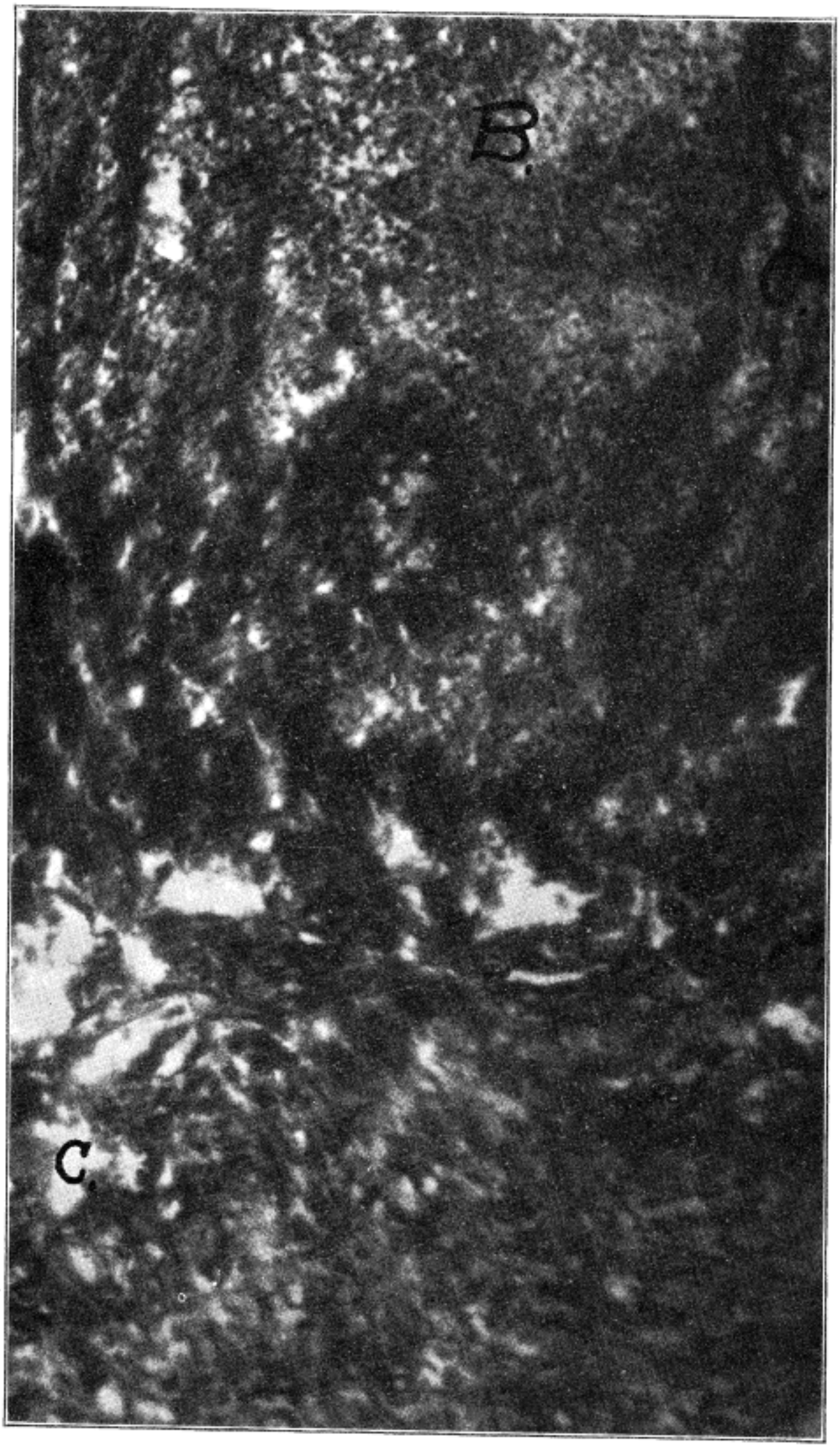

Fig. 37

Fig. 37. Microphotograph, low power, of tumor from uterus of guinea pig No. 3.

B. Blood clot, undergoing organization.

C. Cords of cells of embryonic type, which are invading the blood clot, the blood vessel, and the uterine wall.

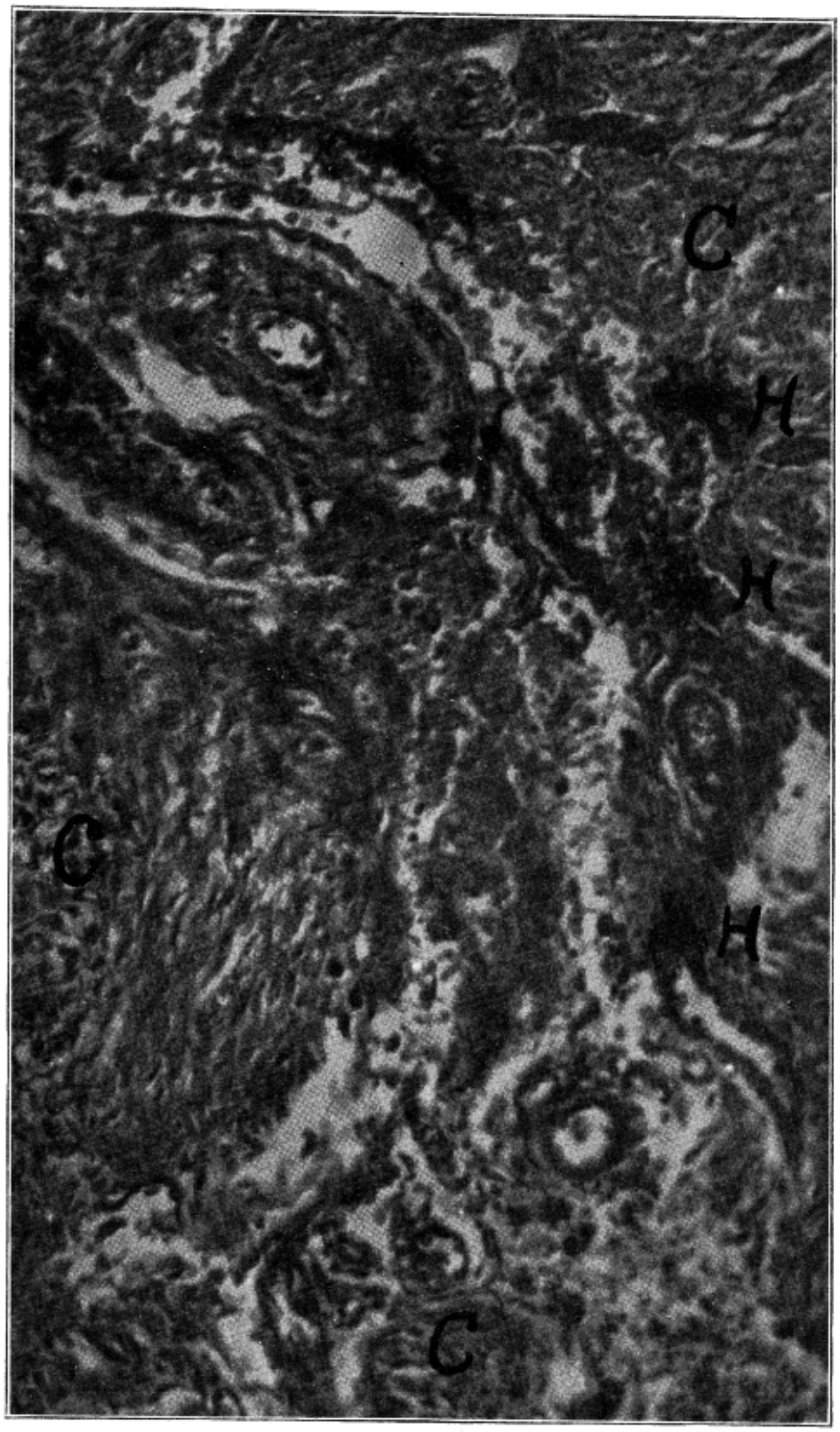

Fig. 38

Fig. 38. Microphotograph, higher power, different section of same tumor as that shown in Fig. 37.

H. Hemorrhagic areas.

C. Cords of cells of embryonic type which are invading the uterine wall and are surrounding the blood vessels. Lymphoid infiltration is shown between the cords of cells.

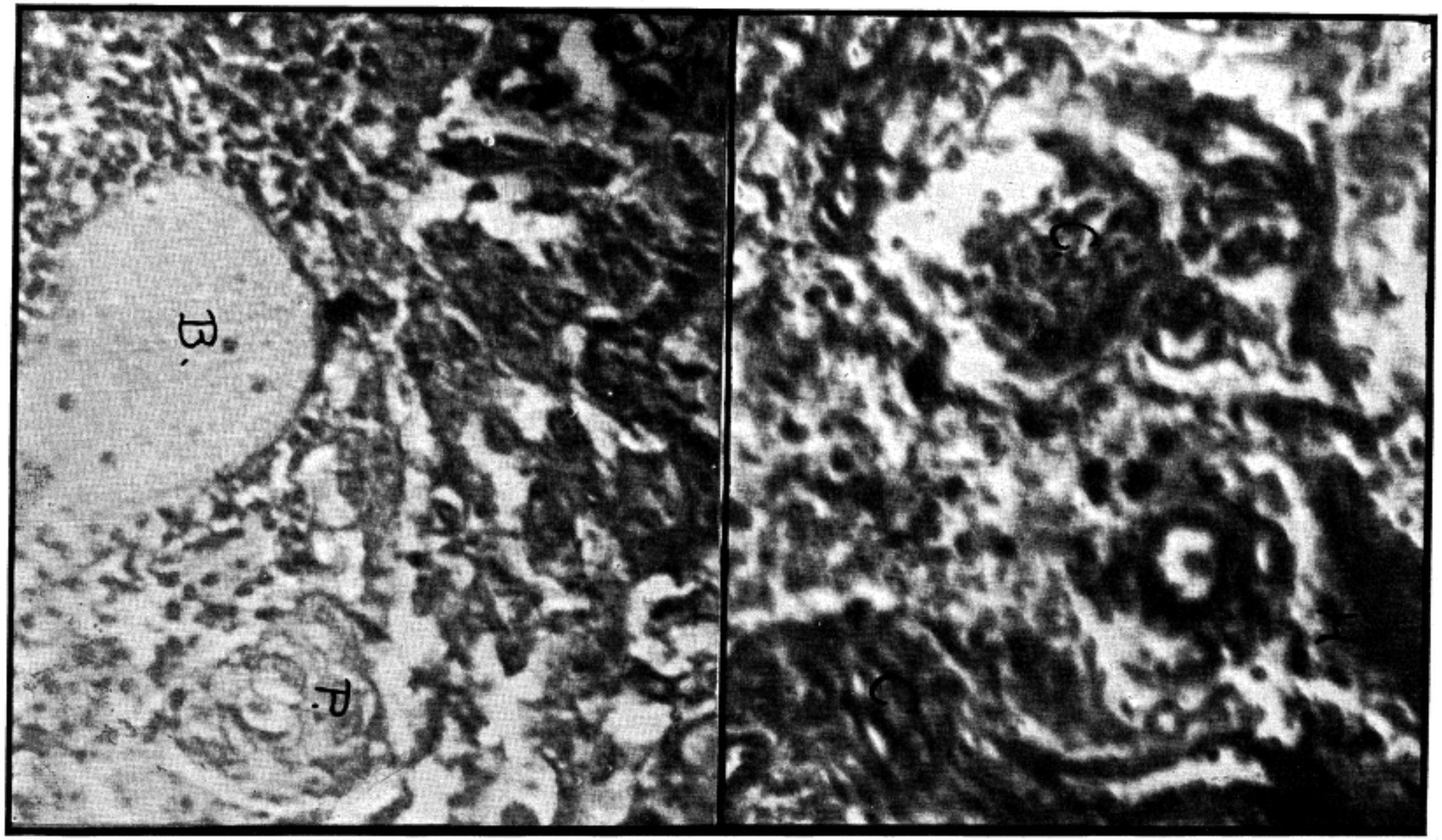

Fig. 40

Fig. 39

Fig. 39. Microphotograph, higher power; part of section shown in Fig. 38.

C. Cords of embryonic cells invading the uterine wall.

Fig. 40. Microphotograph; epithelioma of human cervix uteri.

B. Blood vessel.

P. Epithelial pearl.

Note the thin wall of the blood vessel, the lymphoid infiltration and the extravasation of blood.

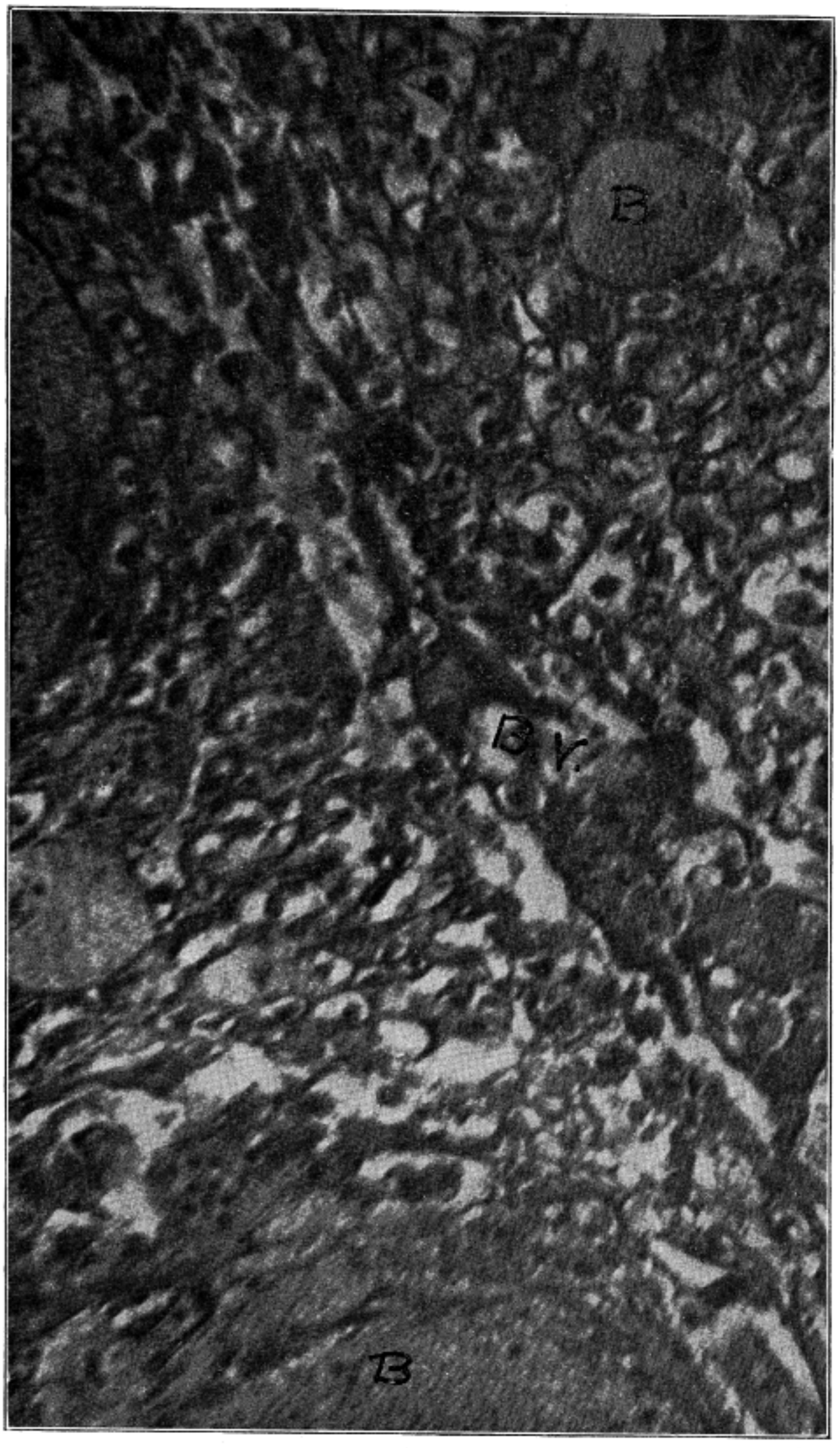

Fig. 41

Fig. 41. Microphotograph; carcinoma, human cervix uteri.

B. Blood within thin-walled vessels.

B.V. Blood vessel being invaded by the cancer cells.

Note the arrangement of the cells in cords, resembling embryonic gland tissue.

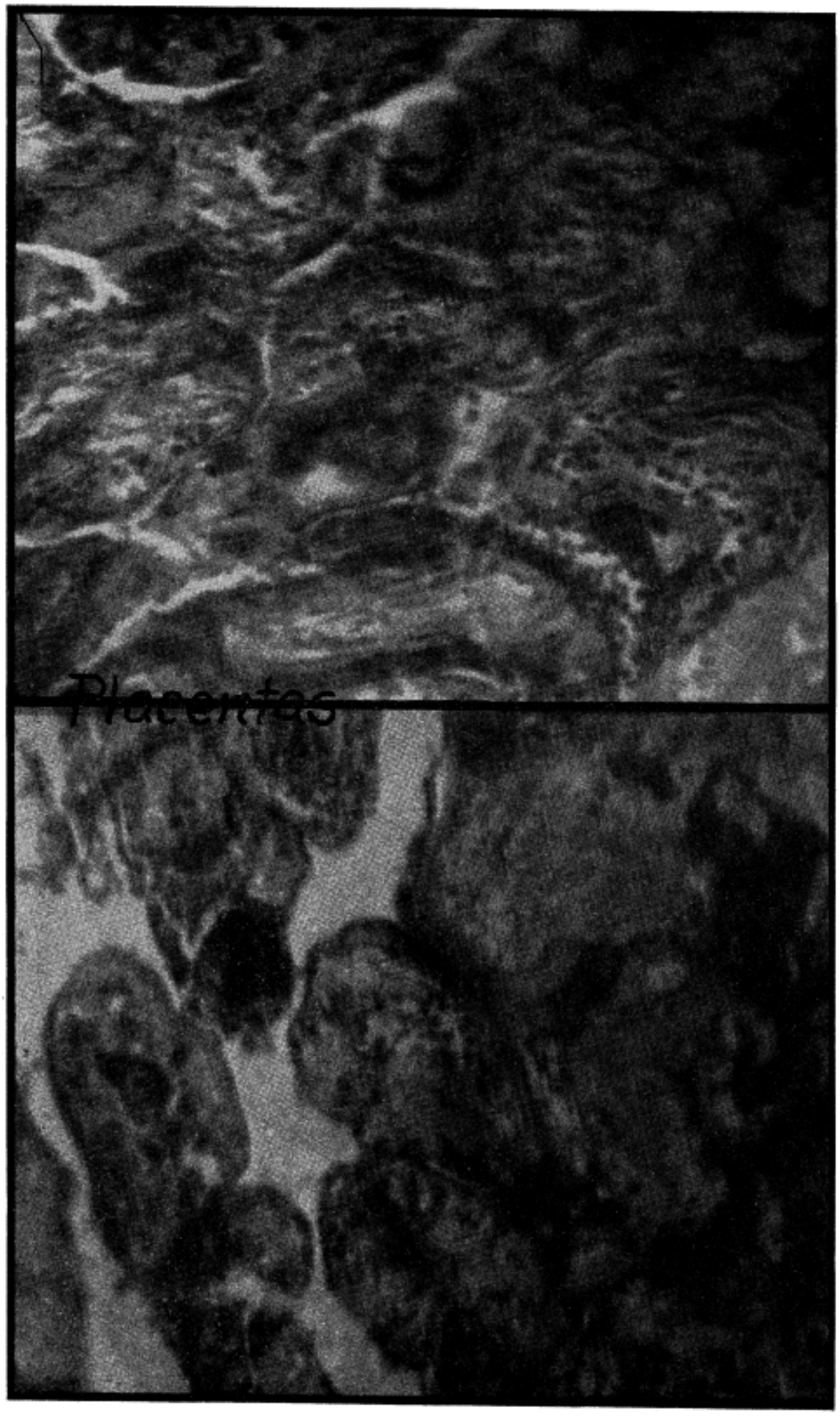

Fig. 42

Fig. 42. Microphotographs. Two sections of placenta of cat.

Placental tissue presents certain resemblances to malignant neoplasms of several types. This marked variability in type, the presence of open sinuses, the finding of fetal blood cells on the fetal side of the circulation and of adult blood cells upon the maternal side of the circulation, all help to give the diagnosis even if the history of the cases gave no hint of the real condition.

(In the malignant tumor of the guinea pig, the history gives no help so far as distinguishing between autochthonous and heterochthonous tumors is concerned.)

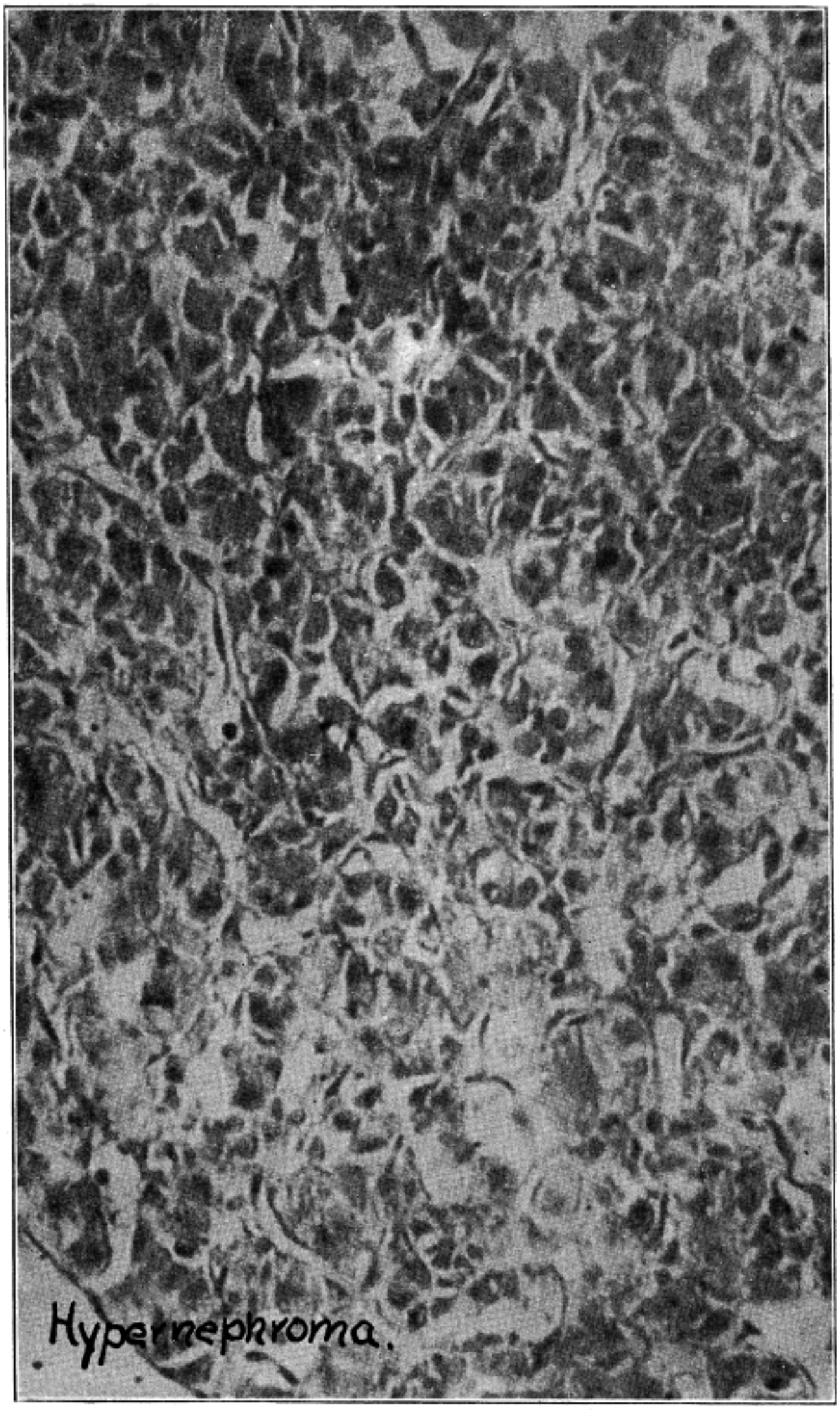

Fig. 43

Fig. 43. Microphotograph. Hypernephroma. This tumor is included for the sake of comparison with the others shown. It is derived from aberrant masses of tissue built upon the type of the supra-renals, and usually invades the kidney. It illustrates a type of malignant neoplasm which is almost certainly due to aberrant developmental conditions.

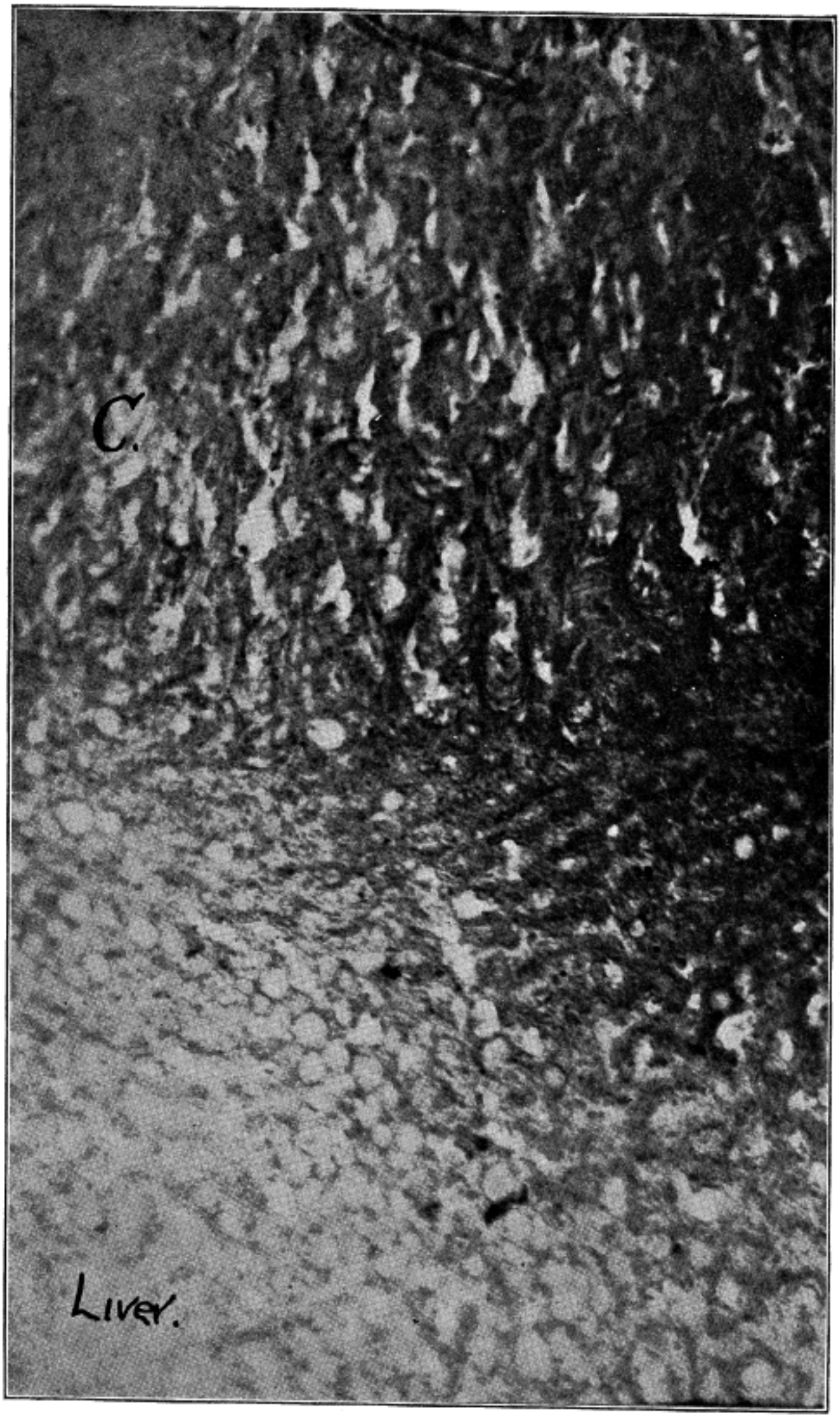

Fig. 44

Fig. 44. Microphotograph; cancer cells, metastases from mammary carcinoma, invading the liver.

C. Cords of cancer cells.

Note the arrangement of cancer cells, and the resemblance to embryonic mammary gland.

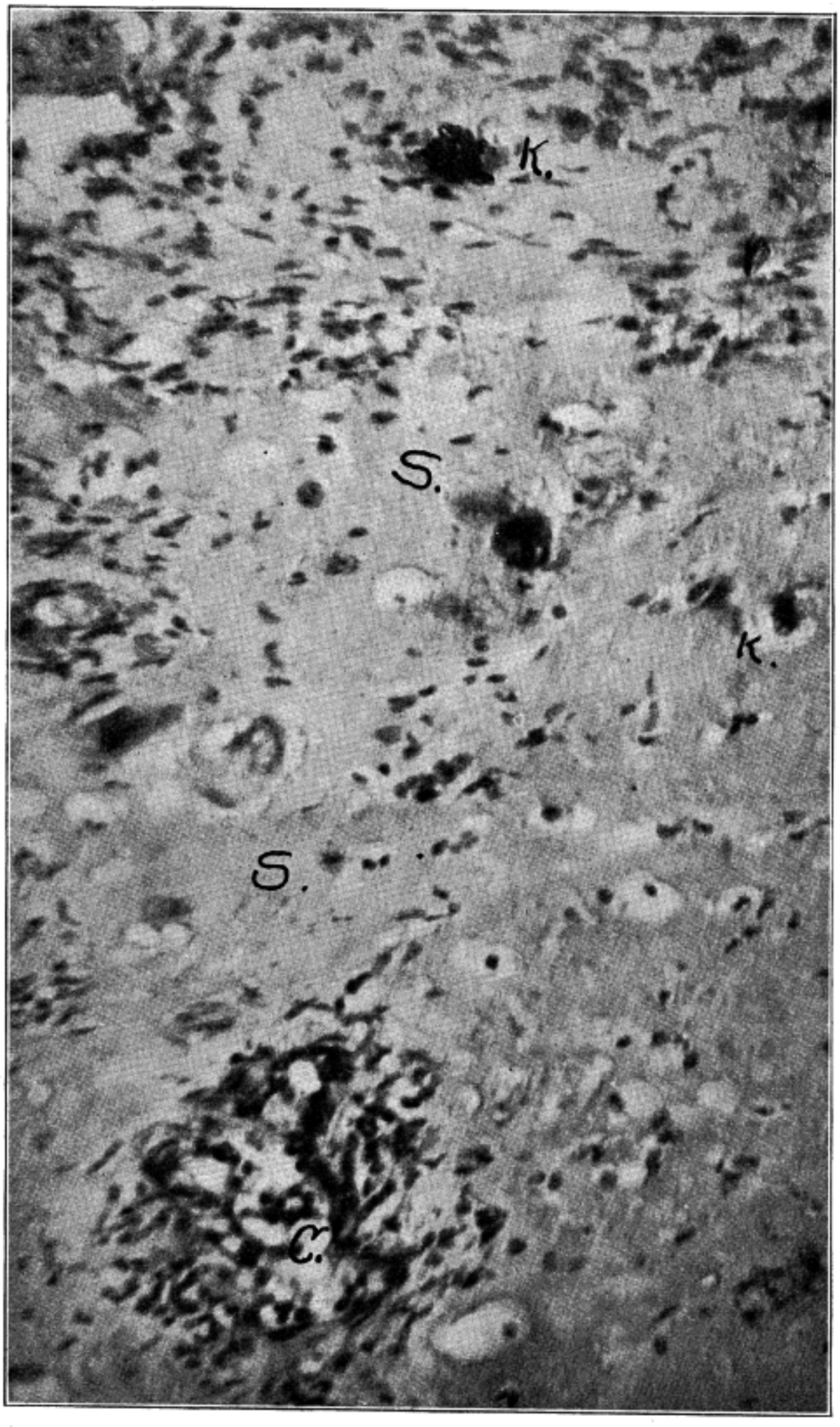

Fig. 45

Fig. 45. Microphotograph; chorio-epithelioma from human uterus.

C. Cords of cells resembling embryonic epithelial tissue.

S. Syncytium; a mass of protoplasm in which the nuclei are dividing, but in which the cells do not become divided.

K. Cells undergoing karyokinetic changes; these indicate rapid growth and marked malignancy.

The presence of the syncytium is characteristic of the chorio-epithelioma, but it is not always found in this neoplasm.

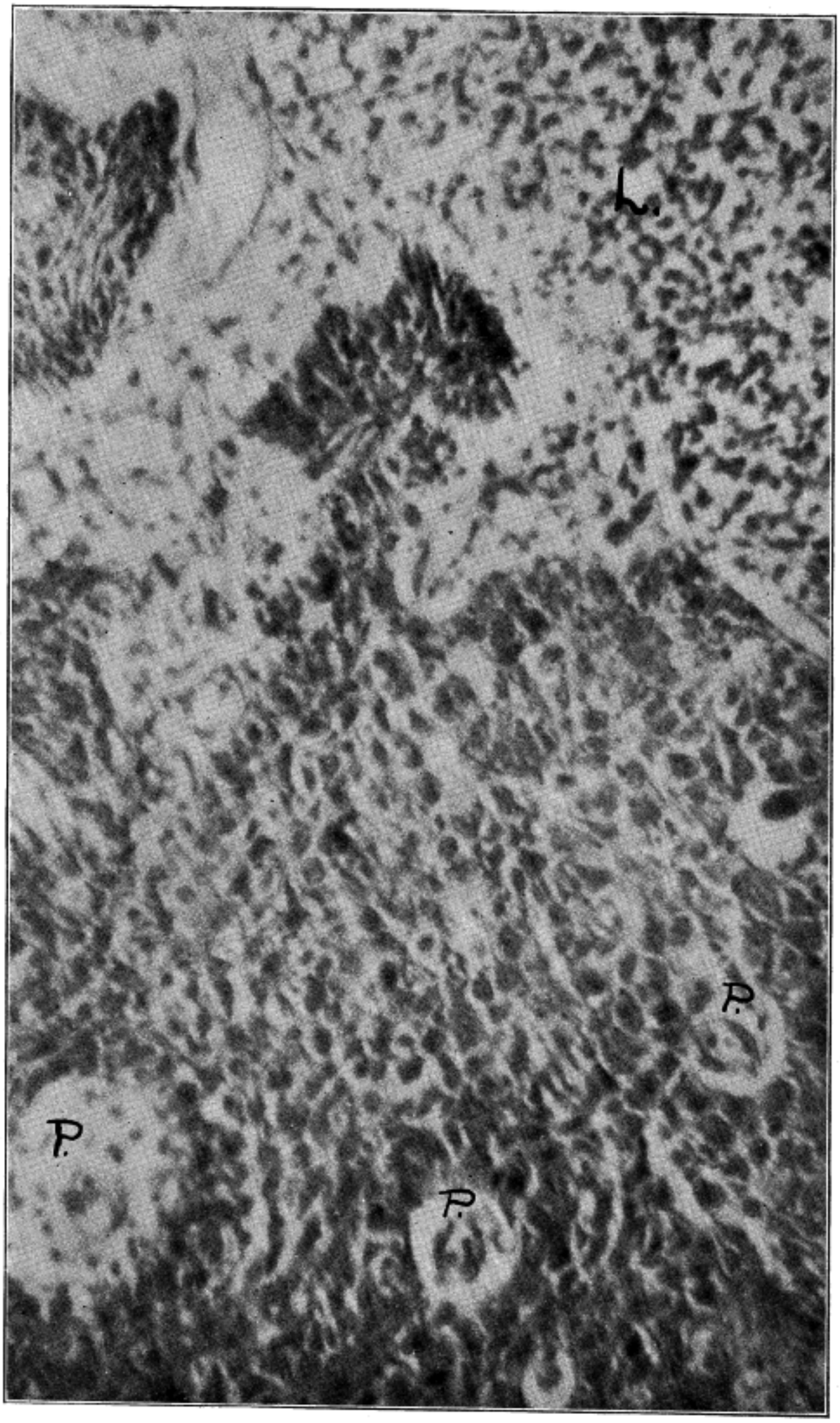

Fig. 46

Fig. 46. Microphotograph; epithelioma of human cervix uteri.

L. Lymphoid infiltration.

P. Epithelial pearls.

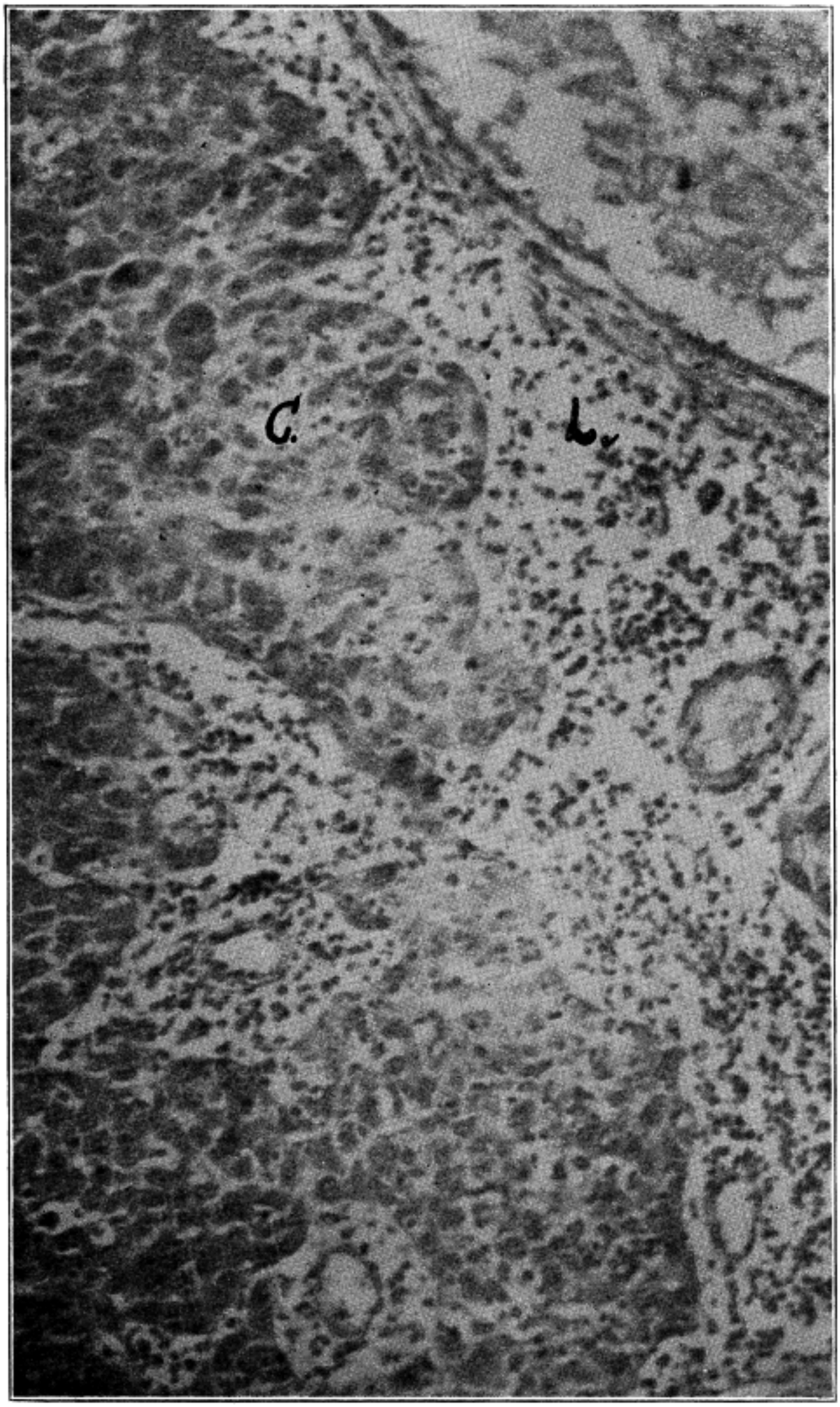

Fig. 47

Fig. 47. Microphotograph; carcinoma invading old scar.

L. Lymphoid infiltration.

C. Cords of cancer cells pushing into the connective tissue of the scar.

Fig. 48

Fig. 48. Microphotograph; uterine carcinoma, human.

L. Lymphoid infiltration.

B. Blood vessel; note that the blood is escaping, and that the cancer cells are invading the blood vessel.

Note also the arrangement of the cords of cancer cells.

A CASE OF MISSED LABOR IN A LESIONED CAT

During August, 1916, about twenty-five cats were purchased for use in the study of bony lesions, and of the effects of lumbar lesions. Among these was one female which appeared to be in a late stage of pregnancy. On examination she was found to have very well marked bony lesions, including the upper lumbar vertebrae, with the lower thoracic probably somewhat involved. After discussing the matter, it was decided to employ very gentle corrective manipulations, and then, if the lesion was not thus corrected, to allow pregnancy to take its course with the lesion still present. (This seems to be in harmony with the views of osteopathic physicians as nearly as we could determine.)

About August 1, supposing labor to be imminent, the cat was placed in a separate cage. After about two weeks, no signs of labor occurring, an X-ray plate was taken in order to secure a permanent record of the lesion, and also in order to determine whether pregnancy was present or not. A fetus at about full term was present, lying in normal position and apparently the end of pregnancy was near. The cat was returned to the separate cage to await labor.

September 1 the cat still showed no signs of labor. An X-ray plate taken at this time shows the fetus in a different position, and the bones are very much less distinctly shown on the X-ray plate. Early in October, since no signs of labor appeared, another X-ray plate was taken. This shows the fetus lying transversely across the abdomen, with its bones showing only very dimly. Evidently maceration is occurring. An epidemic of pneumonia occurred among the cats in December, and all the animals were killed, in order to eliminate the infection. This cat was radiographed (Fig. 49, 50, 51) and then chloroformed.

Autopsy. The abdomen presented a protuberance, as if from a hernia. On opening the abdomen it was found that the large uterus and the omentum were bound firmly to the abdominal muscles by masses of adhesions. The uterus lay across the abdomen in the position indicated in the X-ray plate by the location of the fetus. The appearance of hernia was due to this mass.

The omentum was congested and very soft. The uterine wall was hard, thick and tough, presenting a marked difference from the soft, elastic uterine wall of normal pregnancy. The uterus contained a very long, slender fetus, apparently dead about three months. It appeared to have been about full term at the time of its death. It was placed in a formalin solution for further examination.

The maternal kidneys were very large and full of blood. The liver was deeply engorged. Other viscera appeared normal.

The fetus was broken in preparing it for an X-ray examination. The bones show no abnormalities of structure. The soft parts have evidently become macerated. (Fig. 52.) The causes of the death of the fetus and of the missed labor are not known.

The existence of the bony lesion is interesting. By the accumulation of similar cases, both of those in which the bony lesion is present and of those in which no bony lesions can be found, we may be able to explain the cause of these unfortunate obstetrical accidents.

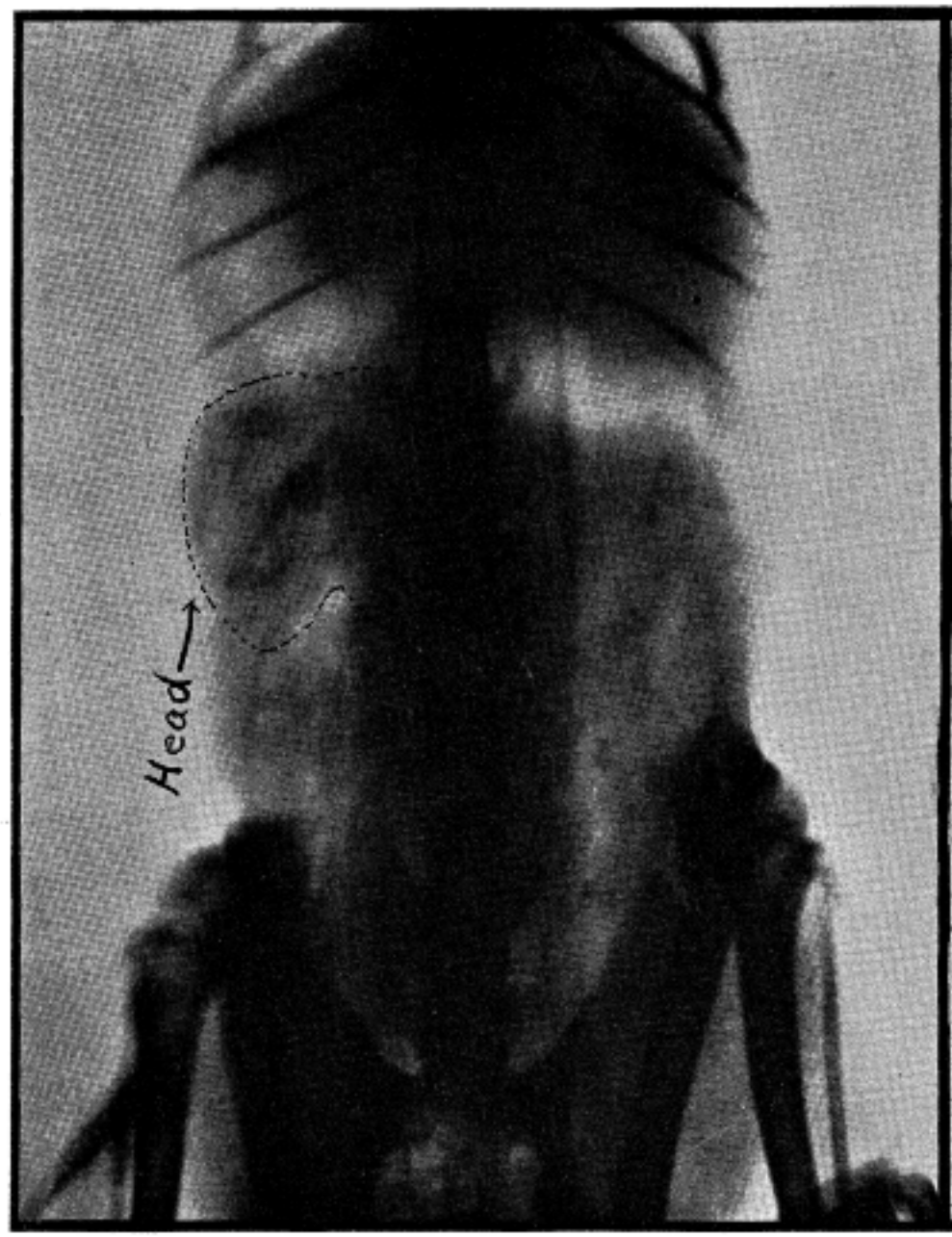

Fig. 49

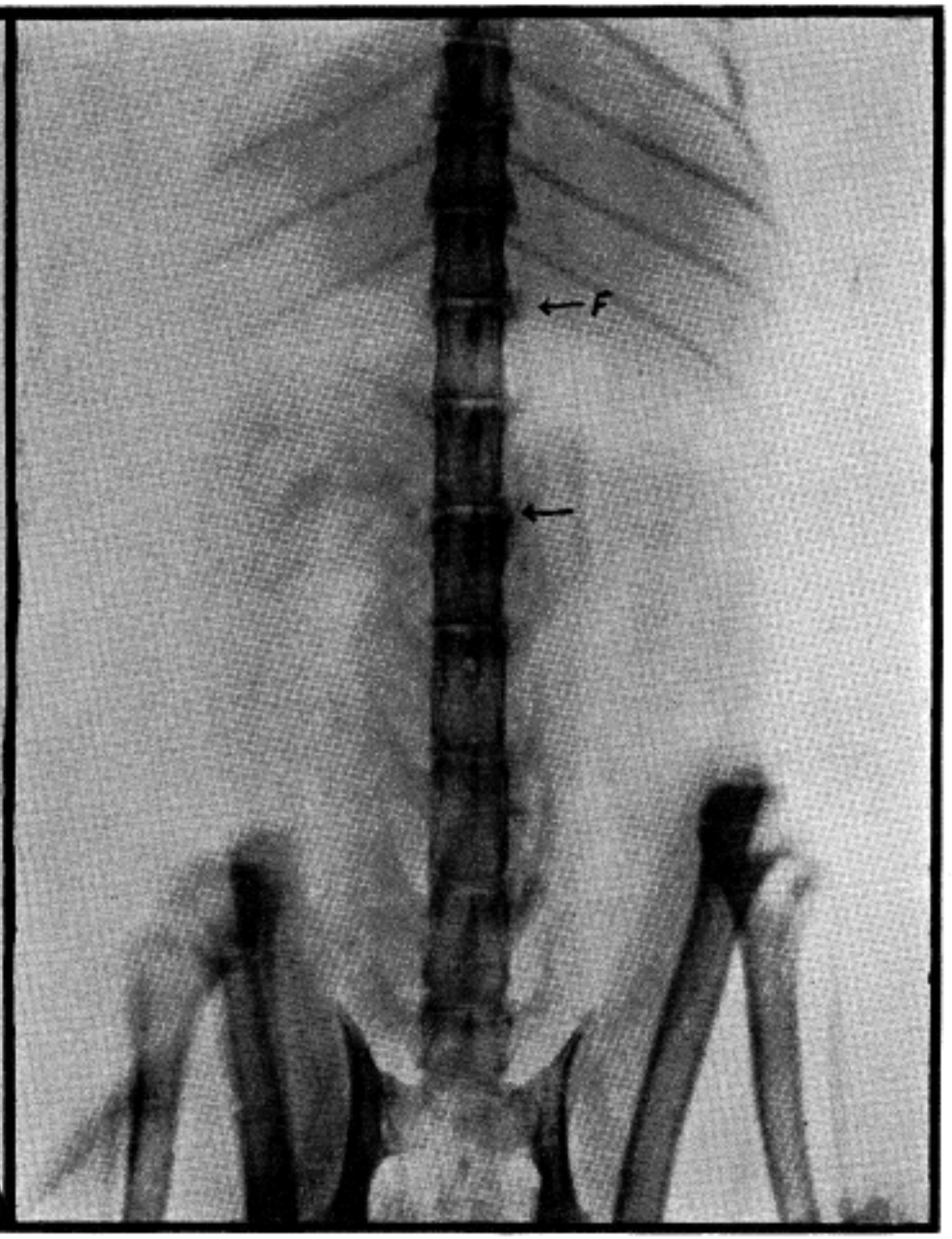

Fig. 50

Fig. 49. X-ray plate of cat with missed pregnancy. This is printed deeply in order to show the head of the fetus.

Fig. 50. Same X-ray plate, but printed lightly, so as to show the maternal bony lesions.

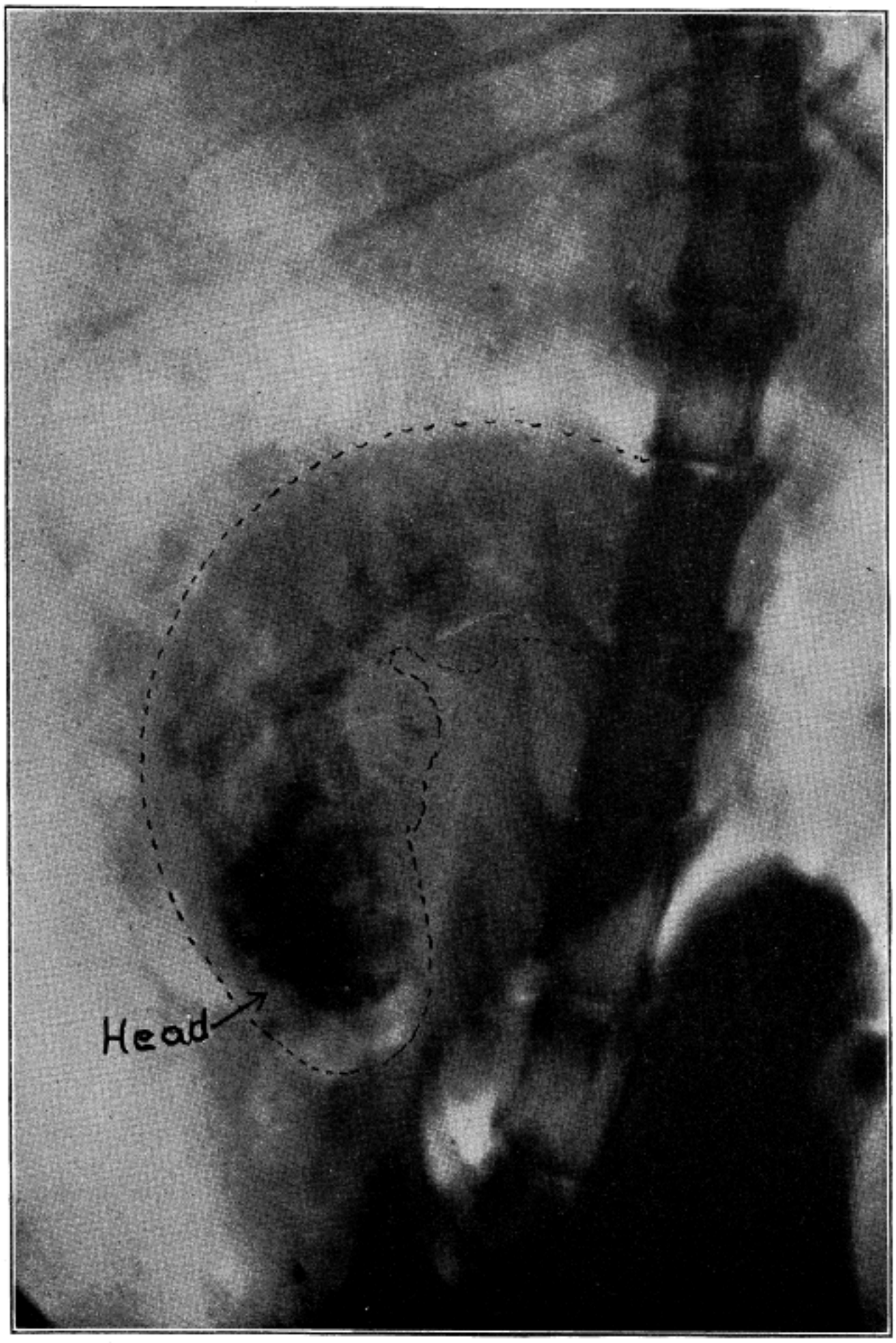

Fig. 51

Fig. 51. X-ray plate of same cat as that shown in Figs. 49, 50. Note the maceration of fetus. Life size.

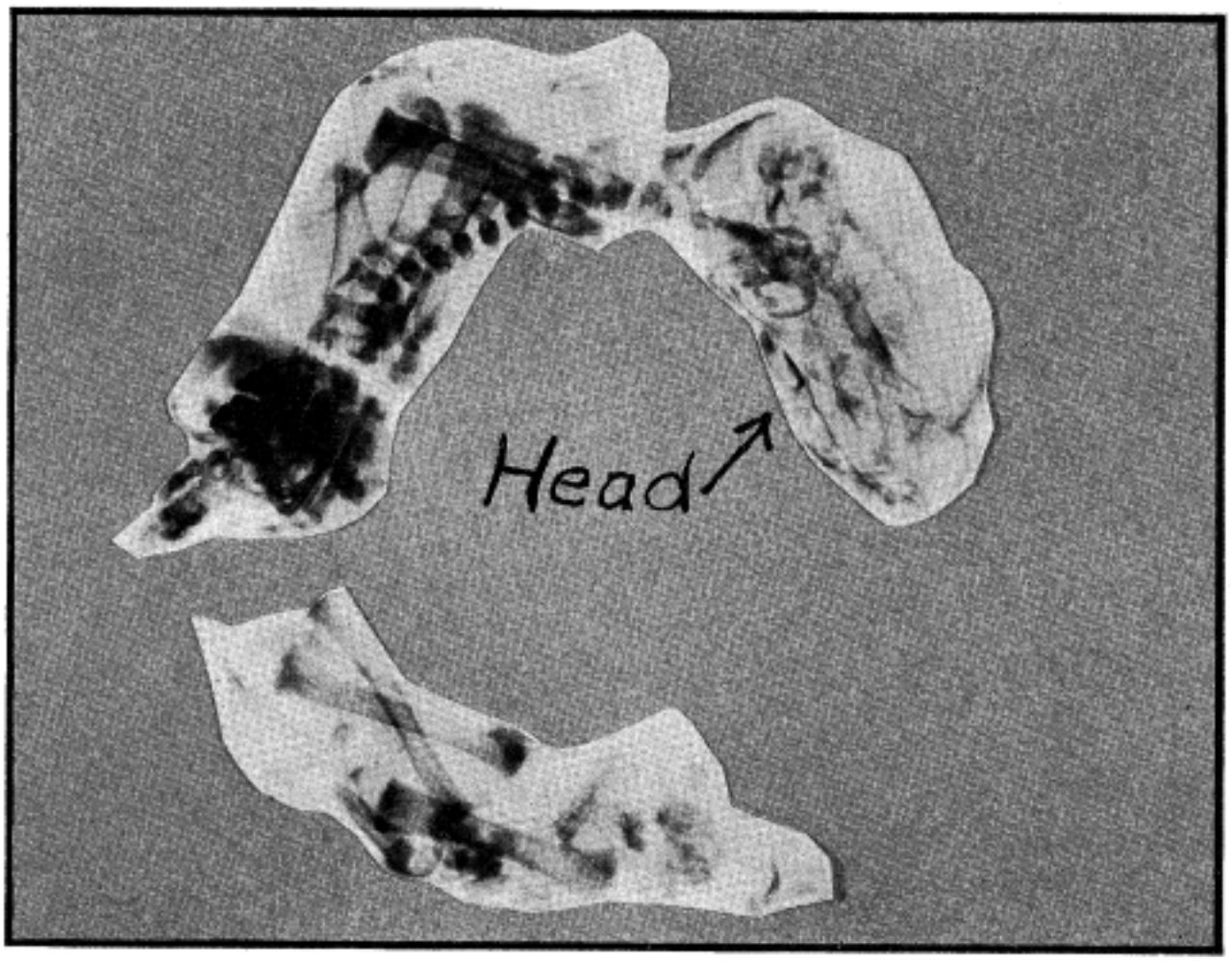

Fig. 52

Fig. 52. X-ray plate of fetus after removal from cat shown in Figs. 49, 50, 51. Note maceration, disturbed bony relations, and cylindrical outline. Slightly magnified.

CLINIC INTERPRETATIONS

Very great caution is necessary in applying the results of animal experimentation to the treatment of human subjects. This is true of all diseases, but it is especially true of those diseases which affect the digestive and the reproductive systems. In order to give some help in this application, a study has been made of such clinic material as is available.

The centers controlling the pelvic viscera have been located within fairly well established limits. In the human subject, especially, there seems to be considerable variation in the location of the various reflexes which affect the activities of the pelvic organs, or by means of which the pelvic organs are able to affect the activities of other and sometimes widely distant organs. There seems no doubt that vertebral lesions affecting the centers in the lower thoracic region are more apt to affect the ovaries or the testes; that lesions in the upper and mid-lumbar region are more apt to affect the uterine and prostatic tissues; that lesions of the sacrum are more apt to affect the rectal, vesical and vaginal tissues.

The examination of clinic patients does not give such exact and clear-cut pictures of lesion and visceral disease as does the examination and care of laboratory animals, for many reasons.

1. Human subjects walk with the spinal column erect, and compensatory curves and lesions increase or become modified according to a great number of factors—the time spent in standing; habits of standing or sitting; the height of the heels of shoes; the disturbing influences of dress; volitional muscular contractions of various types; occupation and the many more or less disturbing circumstances of life inseparable from civilized existence, all modify the spinal curves and the strength of the spinal muscles in a most complicated manner.

2. Human subjects are still developing the human normal in physiology. By this is meant that the human race has not yet learned exactly the best way for humankind to live so as to get the best out of life. It is very evident that humanity cannot live out of doors, without clothes, upon uncooked food, as animals do,

and still have those modern conveniences and the esthetic and altruistic and spiritual advantages which make human life worthy of being called human. Just how physical well-being is to be adapted to the demands of human psychology is yet to be determined. The result of this unsolved problem is found in the fact that nowadays the members of the human race are living in such a way as to encourage diseases of the abdominal and pelvic organs. Taking the human race as a whole, it may be said that our food habits, dress habits, sex habits, our physical habits in general, are more or less unsatisfactory. These factors all predispose to diseases of the pelvic organs especially, and to digestive diseases to a considerable extent.

3. Human subjects are not often examined in the early stages of disease. Thus, the secondary and complicating factors are often found at the first examination, and these may be so much more serious than the original disease as to overshadow the first completely. In many cases it is practically impossible to decide which of several abnormal conditions occurred first; or whether all may have been due to some pre-existing common cause.

4. Animal experiments are not subject to these disadvantages. On the other hand, the value of animal work lies chiefly in the fact that the knowledge thus gained can be applied to human disease processes. This means that all findings must be checked up by human experience, or must be repeated upon so many animals of so many classes that the findings appear to be general among mammals, or general among living organisms, as the specific aim of the research may demand.

The fact that animals are not generally subject to the same quality of diseases as are the human subjects increases the ease of experimental procedures, but increases also the difficulty of making the translation into the language of human pathology and etiology.

5. In all mammals, and especially in the human subject, the relationship between these various organs is so intricate that anything which acts adversely upon one must ultimately affect all. The heavy, congested uterus must affect the ovaries—here, again, the effects are rendered more serious in the human subject, walking erectly, on account of gravity. Examples of the intimacy of the physiological and the anatomical relations of the pelvic

organs of both sexes are known to every physician who handles gynecological and genito-urinary cases with attentive care.

It must be recognized, also, that a very intimate relationship exists between the various organs of internal secretion in the body. In many of these lesioned animals, both male and female, there appeared to be some congestion of the suprarenals when the ovary was diseased. In a few instances only one suprarenal was congested, the other remaining normal in appearance. The thyroid is often found diseased in ovarian disease, and also in disease of the testicles. While very little is known of the relationships of these, and of the other endocrine glands, it is fairly evident that no study of lumbar and pelvic relations is complete which does not take into account the variations in the endocrine functions due to the ovarian disturbances.

6. Certain infections, among which gonorrhoea is most important, affect the human subject in a serious and persistent manner. The reproductive organs of animals are usually free from infections of any kind. The progress of inflammations due to the presence of infectious agents follows the laws of gravity, the direction of the flow of the lymph, secretions, blood and the motion of cilia; and several other less frequent factors may be concerned in determining the ultimate location of the most serious infectious foci. The nature of the infectious agents, whether parasitic or saprophytic, whether bacterial or protozoan, are important factors in determining the manner in which the body reacts to their presence. Bony lesions, with their disturbing influences upon circulation, innervation, secretion and nutrition must be considered important factors, but not only the factors, in controlling the localization of infections and the power of the tissues to overcome various infectious agents. Local circulatory disturbances may be due to the bony lesion exclusively.

7. The real cause of neoplasms is, so far, altogether unknown.

The occurrence of a malignant neoplasm in a guinea pig, known to have borne normal young before having been lesioned, and the occurrence of other tumors in guinea pigs of the same group, similarly lesioned, suggests an attractive field for further investigation. In this study the distinction between autochthonous and heterochthonous tumors must be kept clearly in mind. The great-

est conservatism is necessary in dealing with the many problems associated with the development of neoplasms of all kinds.

Literature

The place of lumbar lesions in producing certain types of pelvic disease in women, and in perpetuating the effects of other diseases, has been discussed rather widely, especially by Dr. Ella D. Still, Dr. M. E. Clark, Dr. Percy H. Woodall, and others.

The importance of maintaining correct lumbar spinal conditions in pregnancy has received the attention of the osteopaths who practice obstetrics, among whom Dr. Lillian M. Whiting, Dr. Blanche Élfrink, Dr. M. E. Clark, Dr. E. R. Proctor have written upon the subject, and have given illustrative case reports in support of this relationship.

The relationship between lumbar lesions and diseases of the appendix and the colon have received much attention. The place of lumbar lesions in producing or in perpetuating diseases of the bladder and the kidneys has been much less freely discussed.

Lumbar lesions as causes of diseases of the male reproductive organs have scarcely been mentioned, though a few authors have referred to this matter.

The place of lumbar lesions in causing or perpetuating diseases of the digestive tract, the kidneys, bladder and pelvic organs in general, is discussed in "Clinical Osteopathy" edited by Dr. C. P. McConnell.

"Diseases of Women," by Dr. M. E. Clark, and "Osteopathic Gynecology" by Percy H. Woodall, give the lesions responsible for certain diseases of the pelvic organs in women.

"Manhood," by Dr. O. E. Smith, mentions the place of lumbar lesions in disturbing the functions of the male reproductive organs.

One of the earliest papers upon this subject was published by Dr. C. E. Henry, in the "Journal of the Science of Osteopathy," April 15, 1900.

Articles published in the Journal of the American Osteopathic Association by the following osteopathic physicians:

Adsit, Marie N., May, 1908, May, 1909; Banning, J. W., May, 1905; Barry, Joanna, May, 1905; Chase, C. M., May, 1905; Clark, M. E., July, 1903, Oct., 1904; Crenshaw, J. H., June, July, 1915; Crow, Louise P., Sept., 1911; Dufur, J. I., June, 1909; Edwards, Eliza, April, 1908; Elfrink, Blanche M., March, 1913; Fisher,

Albert, April, 1911; Foster, Julia E., Dec., 1907; Gerrish, Clara D., March, 1904; Heist, Mary L., June, 1909; Locke, Orella, Dec., 1908, April, 1913; Mackinnon, Barbara, Feb., 1912; Millard, F. P., Nov., 1907; Proctor, E. R., March, 1913, Dec., 1916; Roger, Effie L., May, 1910; Semple, Wm., July, 1917; Smith, Frank H., July, 1917; Smith, O. E., Oct., 1910; Still, C. E., June, 1903; Still, Ella D., April, 1904, July, 1907, Aug., 1906, Feb., 1912; Wernicke, Clara F., April, 1907; Wheeler, G. D., Nov., 1910; Whiting, Lillian M., Feb., 1912; Woodall, Percy H., Aug., 1908, June, 1910.

Clinic Records

Accurate records of the lesions present in patients suffering from diseases of abdominal and pelvic organs are not easily found. A study has been made of published literature, of such clinic records as were available, and of a few patients examined at the Institute on various occasions.

In addition to the books already mentioned, the following material has been consulted:

A. O. A. Case Reports, edited by Dr. Edythe Ashmore.

P. C. O. Clinic Reports, 1900-1914. These include fairly complete records of the entire spinal condition in about two thousand patients in the general clinics and about six hundred patients in the gynecological, obstetrical and genito-urinary clinics.

Several clinics, lectures and case reports have been considered. These have been given by many osteopathic physicians, many of whom have already been mentioned. Others whose lectures or cases reports have been especially helpful are:

Drs. Pearl Bliss, Asa Bliss, Etta Champlin, Chas. Champlin, Jennette H. Bolles, Olive Clarke, E. S. Comstock, Eliza Edwards, R. D. Emery, J. O. Hunt, Barbara Mackinnon, Rebecca B. Mayers, Zuie A. McCorkle, Lura B. Nelson, Martha Petree, C. H. Phinney, Grace L. Smith, Ella D. Still, Geo. A. Still, D. L. Tasker, Margaret Thompson, Clara F. Wernicke, C. A. Whiting.

Index

(The black-faced numerals refer to sections or sub-headings)