HYDRODYNAMICS OF THE RENAL PELVIS

- AN EXPERIMENTAL STUDY -

BY

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INTRODUCTION AND DEFINITION OF TERMS

"ABOUT NATURE, CONSULT NATURE HERSELF" - FRANCIS BACON

Considerable differences exist in the findings of workers who have studied the hydrodynamics of the renal pelvis. By some (Woodside 1944), the pelvis is regarded as a pump, whilst others (Kiil 1957) have suggested that it functions as a relatively inert reservoir. There is, therefore, no agreement about the part played by the renal pelvis in the transport of urine.

In strict anatomical terms, the "renal pelvis" of the clinician, is described as the "pelvis of the ureter" (Cunningham 1943, Gray 1962), as morphological and histological evidence indicate that the pelvis is a part of the ureter. Urological usage, however, has not followed this precept and as this term, "renal pelvis" has gained general acceptance, it has been used throughout the text.

There has been a paucity of studies on the hydrodynamics of the renal pelvis, possibly because the pelvis is not easily accessible for studies of

function, and because, until the 1950's only water manometers were available for measuring intrapelvic pressures. Water manometers record fluid displacement of relatively large volume and so cannot accurately measure the pressure changes associated with the transport of small volumes of urine in the upper urinary tract. The more refined manometers, developed for cardiovascular research, were first used to measure pressures in the renal pelvis in man by Kiil (1953). As pressures were measured through small ureteric catheters connected to Statham physiological pressure transducers the problems of fluid displacement were overcome, but the question of whether the indwelling ureteric catheter seriously distorted ureteric dynamics has not been satisfactorily answered. Kiil believed that, provided the ureteric catheters were small, there was no significant obstruction to urine flow, as he had found that pressures in the renal pelvis were low and did not increase with prolonged periods of recording. However, other workers (Rattner, Fink and Murphy 1957) using similar methods have recorded very much higher pressures in the renal pelvis.

It is important that accurate pressures are

obtained from the renal pelvis as these will provide information about the emptying mechanism of the renal pelvis and possibly contribute to an understanding of the aetiology of some types of hydronephrosis.

It therefore seemed desirable that an attempt should be made to measure intrapelvic pressures by a method which could not interfere with ureteric dynamics. An examination of the anatomy of the pelvis suggested that this difficulty could be overcome by inserting a manometric tube through the renal parenchyma into the pelvis. This method would not, of course, be suitable for measuring pelvic pressures in man, but could be used in the dog.

In this thesis, studies are described on pelvic pressures recorded through "permanent" manometric nephrostomy tubes in the dog. To obtain the fullest possible knowledge, pelvic pressures were measured systematically throughout a range of urine flow rates up to maximum diuresis, and other possible factors affecting pelvic pressures were also examined. In this way, it seemed possible that more information would be gained about the pressures in the renal pelvis and hence about its role in the transport of urine.

GENERAL STATEMENT OF THE PROBLEM TO BE INVESTIGATED

What part does the renal pelvis play in the transport of urine?

REVIEW OF THE LITERATURE

A perusal of the literature on the "Hydrodynamics of the Renal Pelvis" shows that the contribution which the renal pelvis makes to urine transport is closely bound with the function of the ureter. In consequence a review falls naturally into the following sequence of grouping:

a) The Renal Pelvis

b) The Ureter

c) Physio-pathology of the Upper Urinary Tract

d) Mechanisms of Urine Transport.

For clarity a similar order of presentation has been adopted.

a) The Renal Pelvis: In mammals there are two anatomical types of pelvis, the unipapillary and the multipapillary. The former type is found in common laboratory animals such as the rat, guineapig, rabbit, cat and dog, and the latter in the pig, cow, monkey and man (Hinman 1934). The unipapillary pelvis encloses a long papilla and multiple eccentric fornices are formed by the reflections of the pelvic mucosa around large arteries and veins. These fornices give a false appearance of calyces on pyelograms.

In the more complex multipapillary system, minor and major calyces connect the papillae to the pelvis. In the walls of the calyces in man there are circular sphincter muscles (Muschat 1939) and longitudinal levator muscles (Narath 1940). Also in man, the muscle fibres in the wall of the pelvis are collected into loosely arranged bundles, but there is no agreement as to the distribution of these bundles. Schneider (1938) had concluded from a detailed study that the bundles were arranged in a spiral manner continuous with the ureteral musculature. Narath (1951), on the other hand, found that the muscle fibres were interwoven in a seemingly irregular manner, whereas a recent edition of an anatomy textbook (Gray 1962) describes an inner longitudinal and outer circular group of fibres.

Contractions of the renal pelvis of a rhythmic and fleeting nature have been observed in the dog, and in man Muschat (1939) has described very powerful contractions in a large human extrarenal pelvis. Hjort (1954) during operations on man was able to produce contractions of the pelvis by the direct infusion of fluid through a needle, and he noted that the pressure increased during the contractions. Pelvic contractions have, however, been more commonly observed at pyeloscopy. This technique

was first described by Manges (1918) and later improved methods permitted the making of serial films (Legueu, Fey and Truchot 1925) and cinex-camera records (Jarre and Cumming 1930) of pelvic movements.

Contractions of the calyces followed by contractions of the pelvis propelling the contents into the ureter were reported by Jona (1936). He also recorded phasic pressure waves with a modification of "Trattner's hydroporograph" and noted small waves associated with calyceal contractions and large waves with contractions of the pelvis. Woodside (1944) thought that these changes in appearance implied a pumping action by the calyces and pelvis. Moore (1950) and Oldham (1950) also considered that there was a regular sequence of movements of the calyces and pelvis with systole and diastole. In a review of the literature on serial pyleography Narrath (1951) was of the opinion that the viscus and irritating solutions used by earlier workers detracted from the value of their experiments. He suggested that the calyces emptied the tubules of urine by a milking action and that during the pelvic emptying phase the calyceal sphincters prevented a back flow of urine. The development of the X-ray image intensifier has made possible more careful study of the normal and abnormal upper urinary tract under physiological conditions.

Observations by Hanley (1955) have not confirmed a set rhythm of conduction in the calyces and pelvis, the pattern changing from one individual to another and from minute to minute in the same kidney. Hanley (1959) also noted that urine regurgitated freely into the calyces during pelvic contractions.

The literature contains only a few records of attempts to measure the pressures in the renal pelvis in either animals or man. In animals, Lucas (1906-7, 1908) found that the intrapelvic pressure was low and Morales, Crowder, Fishman and Maxwell (1952) reported that it increased to 10-15 cm water during diuresis. During lumbar sympathectomy in man Underwood (1937) recorded a range of 8-16 cm water in the pelvis.

Kapandje (1949) described a technique which permitted the recording of intrapelvic pressures from the undisturbed human kidney. Under fluoroscopic control he was able to insert a needle into the pelvis of normal kidneys but unfortunately he did not report the values of the pressures measured. As recently as 1954, Davis considered that basic physiological data, such as the normal intrapelvic pressure, had not been determined. He stated "there is no suitable method for measuring the intrapelvic pressure, either experimentally or clinically."

The refined methods of pressure measurement being used in cardiovascular research (Hansen 1949) were applied to the human upper urinary tract by Kiil (1953) who made a preliminary report of his technique of "urometry". A preliminary communication was also made by Sorensen and Andreasson (1954). These authors, employing less stable manometers than Kiil, noted that contractions in the renal pelvis were less frequent and more diffuse than in the ureter. Oscillations due to the movements of the patient, respirations, and transmitted pulsation from adjacent arteries were also recorded. Absolute pelvic pressures were not quoted and the illustrations were not calibrated. In 1957, Kiil published the results of an investigation in which intrapelvic pressures were recorded in nearly 200 examinations in man. Normal pressures were low and were uninfluenced by changes in the flow of urine, or by increases in bladder pressure. Respiratory waves, reflecting intra-abdominal pressure fluctuations during respiration, were recorded from the renal pelvis. Low contraction complexes were occasionally seen and were present both in the recumbent and the upright position. These complexes were intermingled with respiratory waves so that it was impossible to ascertain when the pelvis

was in a resting phase and when it was contracting. Only in exceptional cases were fluctuations as high as 3-4 mm Hg. recorded and in these pelves some slight distension was noted. Kiil considered that his failure to demonstrate high intrapelvic pressures excluded a pump action and suggested that the pelvis functioned only as a reservoir and conduit. He thought that low pressures, ensured continuous excretion by the kidney tubules thus making it unnecessary to attribute an ingenous protective mechanism to the calyceal system.

Rattner, Fink and Murphy (1957) using similar methods and recording equipment determined the pressures in the pelves of 17 normal females. Pressures ranged from 6-24 cm H_2O, average 14.7 cm H_2O and waves of maximum amplitude of 11 cm H_2O were recorded. There was no apparent relation between urine flow rate and pelvic pressures.

Davis (1962) considered that "urometry" would soon take its place as a routine diagnostic method in urology. He reported the results of a few intrapelvic pressures measured by this technique to illustrate "how revolutionary the precise knowledge of urodynamics will probably be". He found a range of 7-9 mm Hg.

Pressures in the pig pelvis measured by Melick,

Naryka and Schmidt (1961) ranged from 11-14 mm.Hg.

As in the dog so in man, the anatomical transition from the pelvis to the ureter may be ill-defined and in Hanley's series (1960) a closed pelvi-ureteral junction sharply separating pelvis from ureter was present in less than 10% of normal individuals. Narath (1940) considered that there was a functional sphincter at the pelvi-ureteral junction and Hatz (1950) described a concentration of ring muscle in this region. Murnaghan (1959) on the other hand found a uniform distribution of mixed muscle bundles which, he believed, were necessary for co-ordinated contraction of pelvis and ureter.

The "urometric" studies of Kiil (1957) lend no support for a functional sphincter and although there was a level of sudden transition from the low amplitude pelvic complexes to high uretal waves, this point varied with distention of the tract.

b) The Ureter. Investigation of the properties of the ureter has dominated research on the upper urinary tract. Unfortunately, much of the data recorded is contradictory and may reflect the unphysiological methods used in many of the experiments.

A clear knowledge of the muscle pattern ought to

throw light on the dynamic action of the ureter, but possibly because of technical difficulties, there is no agreement on the direction or grouping of the muscle fibres. A standard anatomy textbook (Gray 1962) describes only longitudinal and circular muscle fibres but the presence of oblique fibres was recorded by Engelmann (1869) and Satani (1919). Schneider (1938) in a detailed study of the musculature of the upper urinary passages, found that, in the main, the muscle fibres formed a spiral, running downwards from the outside to form a middle circular layer and end as an inner longitudinal layer next the mucosa. It was, however, impossible to distinguish separate layers though an isolated section could give this appearance. Murnaghan (1957) observed that the tightness of the spirals changed between the upper, middle and lower thirds of the ureter, and the movement patterns on perfusion were, in his opinion, explained by the different directions of the muscle fibres.

The calibre of the ureter is not uniform and even in foetal life the lumbar ureter is flattened whereas the pelvic ureter is circular (Williams 1951). The suggestion that the ureter could be divided into spindles was made by Fuchs (1933) who had found in his post-mortem studies, narrowings of the ureter in which the sub-mucosa

was dense with few elastic fibres. Between these narrowings were spindles in which the sub-mucosa was loose and the elastic fibres profuse. On perfusion the spindle areas were more distensible than the narrowings. Begg (1946) confirmed these observations in specimens removed at operation from man.

The movements of the ureter were carefully described by Engelmann (1869). In the anaesthetised dog, cat and rabbit he noted that when the peristaltic wave passed down from the pelvis to any part in the ureter, that part dilated, became cylindrical and darker, and moved towards the kidney. The ureter then contracted, the walls became thicker and whiter and moved downwards as the contraction reached its maximum. Thereafter, the walls relaxed, the flat shape reappeared and the ureter returned to its original position, to pause there until the next peristaltic wave passed downwards from the kidney. Normally, the contraction wave always started in the pelvis and there was no anti-peristalsis, but mechanical stimulation resulted in a contraction wave proceeding in both directions from the point of injury. Satani (1919) used this longitudinal movement of the pig ureter to record the contraction of the circular muscle, which he considered, occurred simultaneously. Kiil (1957) has confirmed that the

longitudinal ureteric movement also occurs in man.

Urine stained by the excretion of intravenous indigo carmine enabled Fuchs (1933) to observe the passage of fluid down the ureter. He found that normally urine passed by continuous peristaltic wave from the pelvis to the bladder, but if the bladder was distended the through peristaltic wave ceased and the ureter divided itself into long columns, separated by short empty segments. Pelvic contraction forced a spindle of urine into the upper column and simultaneously a similar spindle detached itself from the lower end of the same column without any apparent intervening peristalsis. This process was repeated through all the columns down to the bladder. Fuchs associated these segments with the morphological segmentation which he had seen at dissection, and each of these units he called a cystoid.

No species difference was noted in the ureteric movements observed by Gould Hsieh & Tinckler (1955) and these authors described a run back into the lower cystoid, distinct from anti-peristalsis, when the contraction wave relaxed at high urine flow rates. In dogs, artificially exteriorised trigones permitted Morales and others (1952) to study the flow of urine from the ureteric orifices.

At low flow rates urine spurted weakly, but, as diuresis increased, the spurt was more powerful and lasted several seconds, until, at extreme diuresis, the peristaltic nature disappeared and urine poured continuously from the orifices at low pressures.

The isolated ureter has been a favourite preparation for the study of ureteric function. Preparations consisted of lengths of ureter (Lucas 1908) whole ureters (Mingers 1936), or longitudinal strips and rings of ureter (Macht 1917) which were immersed in oxygenated physiological solutions and the effects of drugs added to the medium were observed. Adrenaline was reported to increase the amplitude and rate of contractions (Roth 1917, Satani 1919) but this was not confirmed by Agar (1948) who found the ureter insensitive to this drug. An increase in the rate of contraction, and in some instances the force of contraction when acetylcholine was added to the bath was reported by Gruber (1930). Lapides (1948) considered that isolated preparations were "highly unphysiological and in many instances the results were not applicable to the intact human structure." This view was not held by Murnaghan (1957) who thought that the study of movement patterns of the isolated normal and abnormal ureter under controlled conditions of pressure and flow, would permit

correlation of dynamic function with muscle patterns.

In the analysis of the action of drugs on the intact ureter, it has proved necessary to dissociate the effects of urine flow, (Greene and Essex 1942). When urine volume was kept constant, Lapides (1948) recording peristalsis with the "hydroporograph", concluded that drugs were without effect.

Merenyi and Kovasci (1952) were able only in the case of pituitrin to decide that it had a stimulating effect, and the only active drug in Hanley's (1953) series was methantheline bromide which produced inhibition, a finding also noted by Draper and Zorgniotti (1954)

In dogs in which the ureter had an intact blood supply, Abrahams and Pickfury (1956) found that at constant perfusion pressures, intravenous 5-hydroxytryptamine caused occlusive contraction of the ureteric muscle and that adrenaline and noradrenaline produced only transitory constrictions.

Even with massive doses of d-tubocurarine, Scott and de Luca (1960) failed to abolish peristalsis and further evidence for the inactivity of drugs comes from Weinberg and Maletta (1961) who reported that the definite ureteric pattern of frequency of contraction in

31 patients was un-influenced to any extent by drugs.

The relationship of peristaltic movements to intra-ureteral pressure was determined by Lucas (1904-5). The peristaltic wave was seen to raise a column of water a considerable height and a suction effect normally followed each wave. By means of a trocar in the pelvis and a T- or L-tube in the ureter, Lucas (1906-7) found that the wave curves from the ureter were large and infrequent compared with the small rapid oscillations in the pelvis, and he was of the opinion that the ureter protected the pelvis from high bladder pressures (Lucas 1908).

An instrument, "the hydroporograph", was devised by Trattner (1924). This provided a graphic method of recording phasic contraction waves in the ureter. The ureter under investigation was blocked with a large catheter and urine flowed through the catheter past a tambour from which pressure waves were recorded. By varying the size of a needle at the outflow, the pressure in the recording system was regulated to lie within the range of 3-10 cm H_2O. Because the ureter was obstructed and the recording mechanism insensitive Sorensen and Andreasson (1954) doubted if results obtained with the "hydroporograph" could be applied to normal ureteric function. None the less many conclusions about

ureteric physiology have been based on evidence recorded with the "hydroporograph" or a modification of this instrument (Jona 1936, Traut 1937, Petersen 1946, Toth 1948, Lapides 1948, Morales and others 1952 and Hanley 1953).

Lapides (1948) confirmed Trattner's observations that ureteral contractions might vary markedly in rate, rhythm and frequency in the same subject as well as in different patients. He found that intraureteral pressure was altered by the volume of urine excreted, by the peripheral resistence, and by the intra-abdominal pressure. Excretion of a large volume of urine was accompanied by a high intra-ureteral pressure with no visible peristaltic waves.

In anaesthetised dogs, at laparotomy, Gould, Hsieh & Tinckler (1955) inserted hypodermic needles into the ureter. Basal pressures of 0-30 cm H_2O with waves of 4-70 mm Hg were recorded.

In Kiils extensive study (1957) ureteric pressure was recorded at different rates of urine flow. There appeared to be no obvious correlation between rate of urine flow and ureteral contraction, nor between pressure amplitude and travel rate of the contraction wave. The standing pressure in the ureter was low and the amplitude

of the contraction waves varied from 25-60 mm Hg.

In a more limited study of the ureteric pressures in females, Rattner, Fink and Murphy (1957) were also unable to determine any connection between the volume of urine excreted per minute, standing pressures, and the amplitude or frequency of peristaltic waves. However, in children, Melick & Maryka (1960) although agreeing generally with Kiil, found that a rise in intravesical pressure produced a marked rise in the pressure in the lower end of the ureter. In acute experiments in dogs, Scott & deLuca (1960) observed that a rising bladder pressure resulted in an increased amplitude and frequency of the peristaltic wave in the lower ureter, and a very high bladder pressure caused ureteric distension with a raised standing ureteric pressure.

The observations by Orbelli and Brücke (1910) of changes of electro-potentials in ureteric muscle during contraction suggested a further means of study of ureteric dynamics. Mingers (1936) applied this method of "electroureterography" to man, but even the more recent studies in animals (Baker and Huffer 1953, Butcher, Sleater and Schmidt 1957) and man (Hanley 1953) have proved of limited value. A modification, combining "electroureterography" with records of ureteric wave

form, measured by intraluminal transducers, was claimed by Bors and Blinn (1955) to simplify the interpretation of the "electroureterograph". These workers confirmed Hanley's (1953) observations that ureteral stumps may remain active after nephrectomy. Murnaghan (1961) because of technical difficulties and the varying results recorded, considered that "electroureterography" was of little more than empirical value.

c) Physio-pathology of the Upper Urinary Tract. Although the upper urinary tract is considered to exhibit tone, the actual nature of this quality and its method of regulation remain obscure.

Evans (1926) has defined tone as the resistance of smooth muscle substance to extension, but this implication of contractile elements has been disputed in studies on the bladder by Remington & Alexander (1955) who attributed tone to passive structures present in dead tissue. The functional significance of elasticity in ureteral physiology is also uncertain and although ureteral elasticity diminishes with age, this cannot be correlated with quantitive changes in elastic fibres (Boone and Smith 1955).

The isolated ureter does not exhibit tone and behaves as an open tube with equal pressure throughout

the length of its lumen (Tinckler 1956) but the addition of acetylcholine restores the normal intraluminal pressure differentials.

Narath (1951) considered that tone was an important quality of the upper urinary tract and that a change in tone was not associated with a change in internal pressure. In his view, increase of pressure was a dynamic action which could occur in the hypotonic, normotonic, or hypertonic ureter. He illustrated the effect of tone with an excretion urogram, in which, when the bladder was empty the pelvis was of normal size, and when the bladder was full, the pelvis was dilated. Narath considered that a full bladder, by an unspecified reflex, lowered tone in the pelvic muscle with consequent pelvic dilatation. On the other hand, Tinckler (1956) believed that ureteric tone was so regulated that it protected the pelvis from bladder distension and pressure.

A decrease in amplitude of recorded peristaltic waves was held by Greene and Essex (1942) to be evidence for a decrease of ureteral tone, whereas Kiil (1957) considered that, in the absence of obstruction or other dynamic disturbance, the resting pressure did not give any information about the tone of the ureter.

Ureteric tone was thought by Narath (1951) to be regulated by the autonomic system and Gould, Hsieh and Tinckler (1955) were of the same view, finding both sympathetic and parasympathetic stimulating drugs increased tone. Acetylcholine, however, was considered by Durand and Descotes (1952) to lower tone, but the evidence for their claim was poor. It has also been claimed that the autonomic system controlled ureteric motility. Both sympathetic and parasympathetic (vagal) nerves join in the renal plexus which encircles the renal artery, and the ureteric nerves are arranged in superior, middle and inferior groups (Mitchell 1953). Having reviewed the innervation of the urinary system Gruber (1923) concluded that until evidence to the contrary was produced, one must assume that the sympathetic innervation contained both motor and inhibitory fibres while the parasympathetic contained motor fibres. Hatz (1950) held that the vagus was the motor nerve to the calyces and the pelvis, and that a sphincter system was innervated by the sympathetic. These claims were, in the main, based on the observations of Harris (1935) and Jona (1936) that an intramuscular injection of eserine produced vigorous pelvic and calcyeal contractions.

The animal experiments of Durand and Descotes (1952)

failed to demonstrate an alteration in pyeloureteral activity with section or excitation of the vagus, or of the lumbar sympathetic chain. The hypogastric nerve, however, appeared to be excito-motor, an observation first recorded by Fagge (1902). The apparently normal reaction of the upper urinary tract in the auto-transplanted kidney led Quinby (1916) to conclude that denervation does not interfere with function, but Mitchell (1953) has express the opinion that transplantation may not produce absolute denervation.

Connell (1961) put forward the view that in the bowel, the sympathetic and parasympathetic nerves excited a continuous and complimentary regulating action rather than mediating opposing and antagonistic impulses and this suggestion may be relevant to ureteric innervation.

Little light has been thrown on the normal function of the pelvis and ureter by the study of the abnormal.

The effects of acute obstruction have been investigated in experiments in animals. Henderson (1905-6) found that when the ureter was acutely obstructed, peristalsis ceased at 26-32 mm Hg. The pelvis was apparently protected from ureteric pressures up to 25-50 cm H_2O, but, when this was exceeded, back pressure was transmitted to the kidney, Lucas (1908). Pilcher, Bollman

& Mann (1937) confirmed that the pelvis was practically empty up to ureteric pressures of 20-30 cm H_2O. Above this pressure hydronephrosis was present associated with impairment of kidney function.

It was not, however, until recently that the pelvic pressure was recorded directly in acute obstruction experiments and not via the ureter. Risholm, Ulfandahl and Öbrink (1959) inserted a plastic tube through the renal parenchyma into the pelvis, connecting the tube to an electro-manometer. Their observations, however, did not differ significantly from earlier workers. Pelvic pressure remained low until the ureteric pressure exceeded 30 mm Hg. at which level the character of the ureteric contractions changed and pelvic and ureteric pressures gradually reached the same level with a maximum pressure in the system of 45 mm Hg. Tubular secretion would seem to be responsible for some of this pressure head as during mannitol diuresis Kiil and Auckland (1961) obtained maximum pressures of 150 - 160 cm H_2O in the acutely obstructed pelvis, whereas the pressure was 10-15 cm lower during water diuresis and during saline diuresis the pressure rarely exceeded 100 cm water.

The pelvis at first responds to chronic obstruction by hypertrophy of its musculature but later

it becomes a thin walled dilated sac (Jewett 1940). In sections of the chronically obstructed ureter Maluf and Halpert (1956) observed hypertrophy of the "urothelium", the tunica muscularis, and the tunica propria. Lengthening as well as dilatation of the ureter was recorded by Vermooten and Wheeler (1930), a failure of the blood vessels to lengthen proportionately being thought responsible for the severe kinking of the ureter which sometimes occurred.

The experimental methods employed to produce chronic hydronephrosis in animals have mainly depended on some form of narrowing of the ureter, either by an elastic band (Scott 1912) or a lossely tied ligature (Vermooten and Wheeler 1930).

Re-establishing the lumen of a sectioned ureter over a short glass cannula (Watson Beech 1931) resulted in dilatation of the proximal ureter and pelvis with slight dilatation in the distal ureter. Obstruction occurred even when the widest possible lumen was used. Repeated dilatation of the ureter with a large ureteric catheter also produced hydronephrosis (Greene 1944) as did cellophane which was loosely wrapped round the ureter (Mosowsky 1961). Finckle, Karg and Smith (1962) thought that this latter method produced an atonic obstruction as the lumen of the ureter remained of normal calibre.

Crushing the rabbit ureter with a small clamp was found by Sheehan and Davis (1960) to produce a hydronephrosis in about 50% of solitary kidneys. The short crushed segment appeared to interfere with the passage of the peristaltic wave, but there was no gross mechanical obstruction as urine could be easily squeezed through the crushed segment.

In the human, in the few instances in which pressures have been recorded, the intrapelvic pressure and contraction complexes have not apparently been increased (Underwood 1937, Kiil 1957, Melick and others 1961). Leger, Caillet and Libaude (1948) on two occasions inserted a T-tube into the hydronephrotic kidneys, so that one end of the short limb lay in the pelvis and the other in the ureter. Through the T tubes these authors made pyelograms with radio-opaque fluid at different heads of pressure and found that the pressure required to fill the pelvis varied from 4-7 cm H_2O. The term "radio-manometry" was applied to this technique. In an animal preparation in which chronic ureteric obstruction was produced by enclosing the ureter in an aluminium band, pressure from the proximal ureter was measured through a polythene T-tube (deLuca, Swanson and Smyth 1961). The initial ureteral pressure of 0 cm H_2O increased to 10-30 cm H_2O and these authors considered

that this increase in pressure was responsible for the hydronephrosis. In chronic obstruction Hinman (1954) believed that an initial pelvic pressure of 50-70 mm Hg would gradually decrease as the obstruction continued and atrophy of the kidney occurred.

d) Mechanisms of Urine Transport: The propulsive wave observed in the ureter of animals (Engelmann 1869) and of man (Anderson 1951) consists of a wave of relaxation preceeding a wave of contraction. The pattern of this wave appears to conform with that of the peristaltic wave described by Bayliss and Starling (1899) in the intestine, but Hanley (1956) from his observations with the X-ray image intensifier, doubted if such true peristalsis occurred in the upper urinary tract. The contraction or peristaltic wave is a simple stretch response elicited by distention of the ureter with fluid (Bozler 1947) A similar wave could also be produced by pinching the ureter (Engelmann 1869) or by the insertion of intraluminal beads (Wislocki and O'Connor 1921). Peristalsis has also been observed in the ureter after kidney transplantation (Dempster 1957) apparently before secretion of urine had commenced. De Klerk (1954) has suggested that there may be a biochemical factor in the

urine influencing the pattern of ureteral peristalsis.

It is believed that the normal peristaltic wave commences high in the ureter or the pelvis (Engelmann 1869, Penfield 1920) and a pacemaker has been postulated in the upper ureter (Bozler 1942). Dempster (1957), however, reports that Sir Arthur Keith searched in vain for a node similar to that in the heart. If the ureter is slightly traumatised a contraction ring develops and, from this ring, contraction waves pass in either direction with equal facility (Penfield 1920).

The present theory of myogenic conduction of the impulse was first suggested by Engelmann (1869) who, failing to find motor nerves, concluded that the excitation impulse was conducted directly from fibre to fibre across protoplasmic bridges. The evidence for a protoplasmic syncitium was strengthened by the monophasic character of the action potential and conduction by the "all or none" response (Bozler 1947), and Agar (1948) has also drawn attention to the similar qualities of ureteric and cardiac muscle.

Nerve plexuses enclosing the muscle layer were identified by Satani (1919) and Henderson (1923) suggested that peristaltic failure was in part due to fatigue of the nerve network. Underwood (1937) considered that

extrinsic nerves modified peristalsis probably by inhibitory rather than motor impulses, but Bulbring, Lin and Schofield (1958) found that degenerative section of the extrinsic nerves had no effect on the peristaltic response. However, these workers were able to abolish the peristaltic reflex by the application of local anaesthetic to the mucous membrane, or by removing the mucous membrane. Further support for an intrinsic reflex arc conducting peristalsis comes from the work of Connell (1961) on the gut, who found a sensory nerve ending on the mucosa and a motor nerve innervating the muscle cell.

As usual where the basic facts are at variance, there are conflicting theories to explain the actions of the organ under discussion. That the pelvis and ureter convey urine from the kidney to the bladder is practically the only generally accepted observation in the literature.

It remains undecided whether the pelvis acts as a pump (Woodside 1944) is relatively inert (Kiil 1957) or perhaps is drained by milking action of the ureter (Lucas 1904-05)

In the ureter there are three possible mechanisms of urine transport.

1. <u>The straight through peristaltic wave.</u>

In this, urine is conveyed by active muscle

relaxation from pelvis to bladder (Engelmann 1869, Anderson 1951)

2. The ureter as a suction pump

This is based on the observation of Lucas (1904-5) that a suction phase normally followed a peristaltic wave. This was supposed to milk the pelvis which was prevented from collapsing by its anatomical structure. More refined methods of pressure recording (Kiil 1957) throw considerable doubt on a suction theory as the only negative pressures which have been recorded by recent authors have been artefacts caused by technical faults.

3. The Cystoid theory of transport

This was based on Fuch's observations (1933) although he himself regarded the through peristaltic wave as the more normal mode of transport. It was postulated that the division of the ureter into cystoids safeguarded tubular secretion by maintaining a low pressure in the pelvis, while higher pressures, necessary to force urine into the bladder could be developed in the lower ureter. This cystoid theory was held by Begg (1946) to explain most satisfactorily, radiographic observations.

A combined mechanism, incorporating the through peristaltic wave and the cystoid division of the ureter,

received support from the experiments of Gould, Hsieh and Tinckler (1955). These workers considered that the actual method of conveying the urine was primarily dependent on mechanical factors, with accompanying reflex modification of ureteric tone being due to stimulation of organs in the bladder or perhaps the ureter.

The literature on the dynamics of the upper urinary tract during the past 100 years is full of contradictions and the relative importance and possibly complimentary actions of the pelvis and ureter have not been clearly established. The present situation would seem to be comparable to that of the physiology of the gut at the end of the 19th century when Bayliss and Starling wrote "on no subject in physiology do we meet with so many discrepancies of fact and opinion".

DETAILED STATEMENT OF PROBLEM TO BE INVESTIGATED

DETAILED STATEMENT OF PROBLEM TO BE INVESTIGATED

It will be seen from the above review of the literature that the contribution which the renal pelvis makes to the system of urine transport is not clear as pelvic pressure measurements made by different workers suggest functionally opposite roles for the pelvis. Difficulties arise in comparing and evaluating previous results as workers have tended not to define the physiological conditions obtaining in the urinary tract during their experiments, or have simulated varying rates of urine flow by the direct infusion of fluid into the renal pelvis.

It was the aim of this study to determine if the basal pelvic pressure, which was arbitrarily defined as the pressure in the renal pelvis when the rate of urine flow was less than one ml per kidney per minute, was modified by such physiological factors as urine flow rate and bladder pressure, and to ascertain what further knowledge of pelvic pressure and function might be learned from the production of certain physio-pathological states. In addition, pelvic pressures were measured

when fluid was directly infused into the pelvis and attempts were made to correlate pelvis pressures with pelvic movements and determine if there was an interconnection between pelvic pressures and pressures in the zone of transition, between the pelvis and ureter, and in the ureter itself.

The following plan of study was adopted for the investigation.

I To establish basal pelvic pressures and determine if these pressures were modified by urine flow rate and bladder pressure.

II To determine if pelvic pressures would be modified by autotransplanting the kidney, hence depriving the upper urinary tract of its extrinsic nerve supply.

III To record the changes in pelvic pressure consequent on:

a) Ureteric obstruction

b) Uretero-colic anastamosis

IV In addition, studies were made to correlate pelvic pressures with:

a) Direct infusion of fluid into the pelvis

b) Pelvic movements observed on pyeloscopy

c) Pressures in the zone of transition between the pelvis and ureter

d) Ureteric pressures.

MATERIALS AND METHODS

I

The basic experimental preparation was made by inserting a "permanent" nephrostomy tube into the dog kidney so that subsequently intrapelvic pressures could be recorded with electronic manometric equipment. This preparation was then used to determine the effects of various factors and procedures on pelvic pressures.

THE MANOMETRIC TUBE: The permanent nephrostomy tube (Fig.I) was composed of an inner polytetrafluorethylene (Teflon) tube, inside diameter 0.035 ins., wall thickness 0.012 ins., ensheathed in a No.8 Ch. Neoplex (Porges) tube 33 cms in length. Two cuffs of polyvinyl alcohol (Ivalon) sponge each 1½ cm. in length were bonded to the Neoplex with polyurethane (Estane 5740X), so that the tube would be anchored to the renal parenchyma and the muscle of the abdominal wall.

GENERAL ANIMAL MANAGEMENT: Healthy female greyhounds weighing between 22 and 27 kilos were used in this investigation. These dogs were kept in modern animal quarters on normal diets and without special restrictions of their activities. Operations were

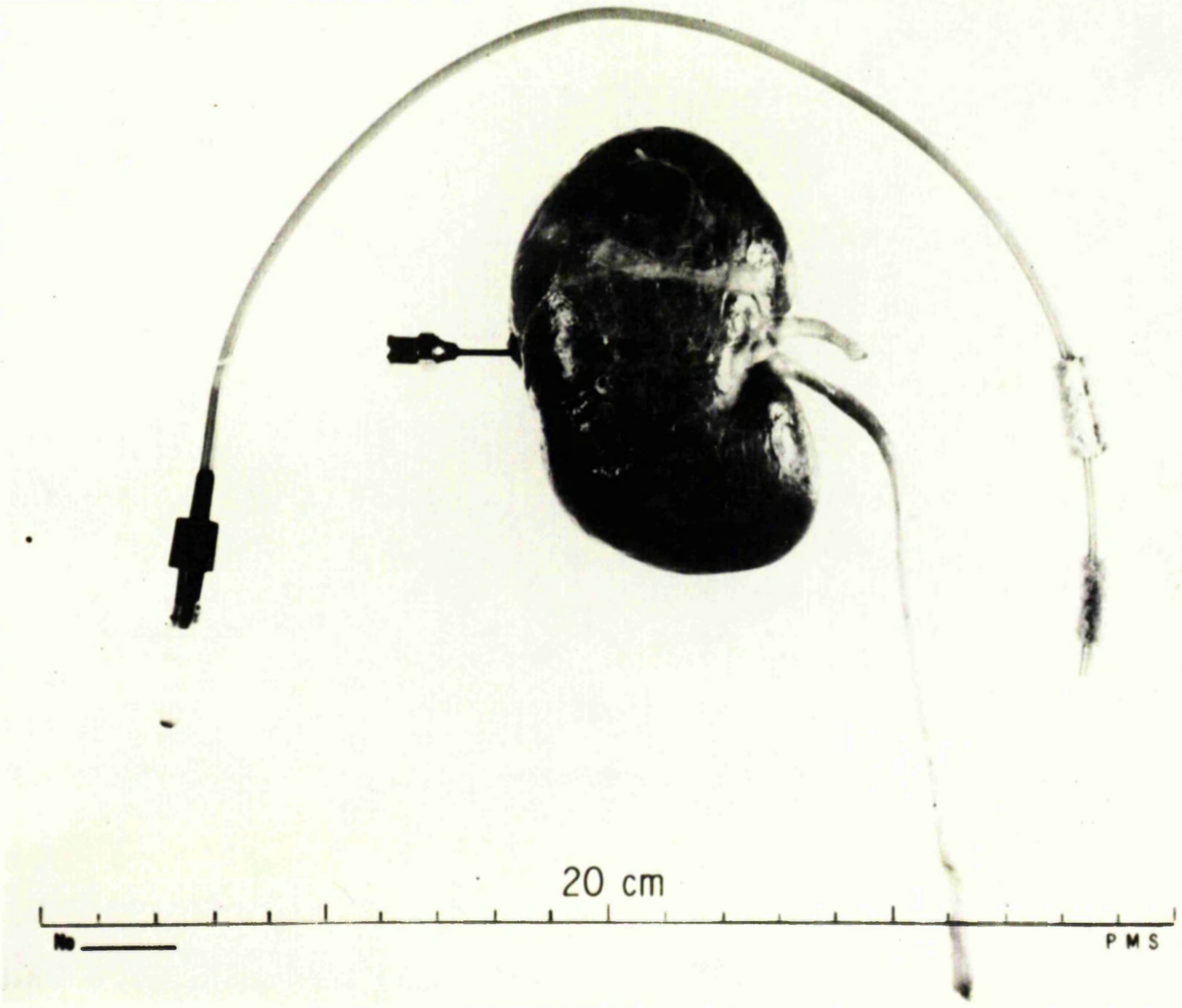

FIG. 1 PERMANENT NEPHROSTOMY TUBE

carried out under general anaesthesia and premedication with 0.5 mg atropine and 25 mgm promazine hydrochloride (Sparine) was favoured. After induction of anaesthesia with intravenous thiopentone sodium (Pentothal), the animals were intubated with endotracheal tubes and anaesthesia maintained with nitrous oxide, oxygen and ether, administered from an Oxford Vapouriser. The operations were carried out in a fully equipped theatre with aseptic technique. Tetracycline 50 mg/kg. body weight was administered in two doses per day for five days after operative procedures.

TECHNIQUE FOR INSERTING THE MANOMETRIC TUBE: The abdomen was opened through a midline incision and the gut was packed aside to expose the kidneys lying behind the peritoneum of the posterior abdonimal wall. The lower left kidney was freely mobile and easily accessible. On the right side exposure was poor and in a few animals it was necessary to incise the lateral peritoneal reflection to permit the convex lateral border of the kidney to be rotated anteriorly.

In dogs 1 - 7, tubes composed of No.8 Neoplex without the Teflon inner tube were inserted by a standard nephrostomy technique (Badenoch 1953), the small incision in the pelvis being repaired.

In the remainder of the series the tubes were introduced by a modification of a technique described by Seldinger (1953). A no.18 gauge 1½ inch needle was inserted through the renal cortex at a point opposite the hilum. The object was to locate the pelvis at the first puncture at a point opposite the pelvi-ureteral outlet so that the nylon stilette would pass down the ureter, thus ensuring that the tip of the nephrostomy tube was correctly positioned. As pelvic dilatation facilitated this procedure, a diuresis was induced with intravenous saline. If the pelvis was not satisfactorily located the needle was withdrawn and the wound compressed for a few minutes to obtain haemostosis. The needle was then re-inserted at another site. When urine escaped from the needle the nylon stilette was advanced into the ureter and the needle withdrawn. The depth at which the pelvis was entered was noted. The Teflon tube was then cut to the appropriate length and the manometric tube inserted over the guide stilette until the tip of the tube lay in the renal pelvis, the Ivalon sponge being forced into the parenchyma (Fig.2)

The nephrostomy tube was then drawn through a long oblique track in the abdominal wall to a puncture wound

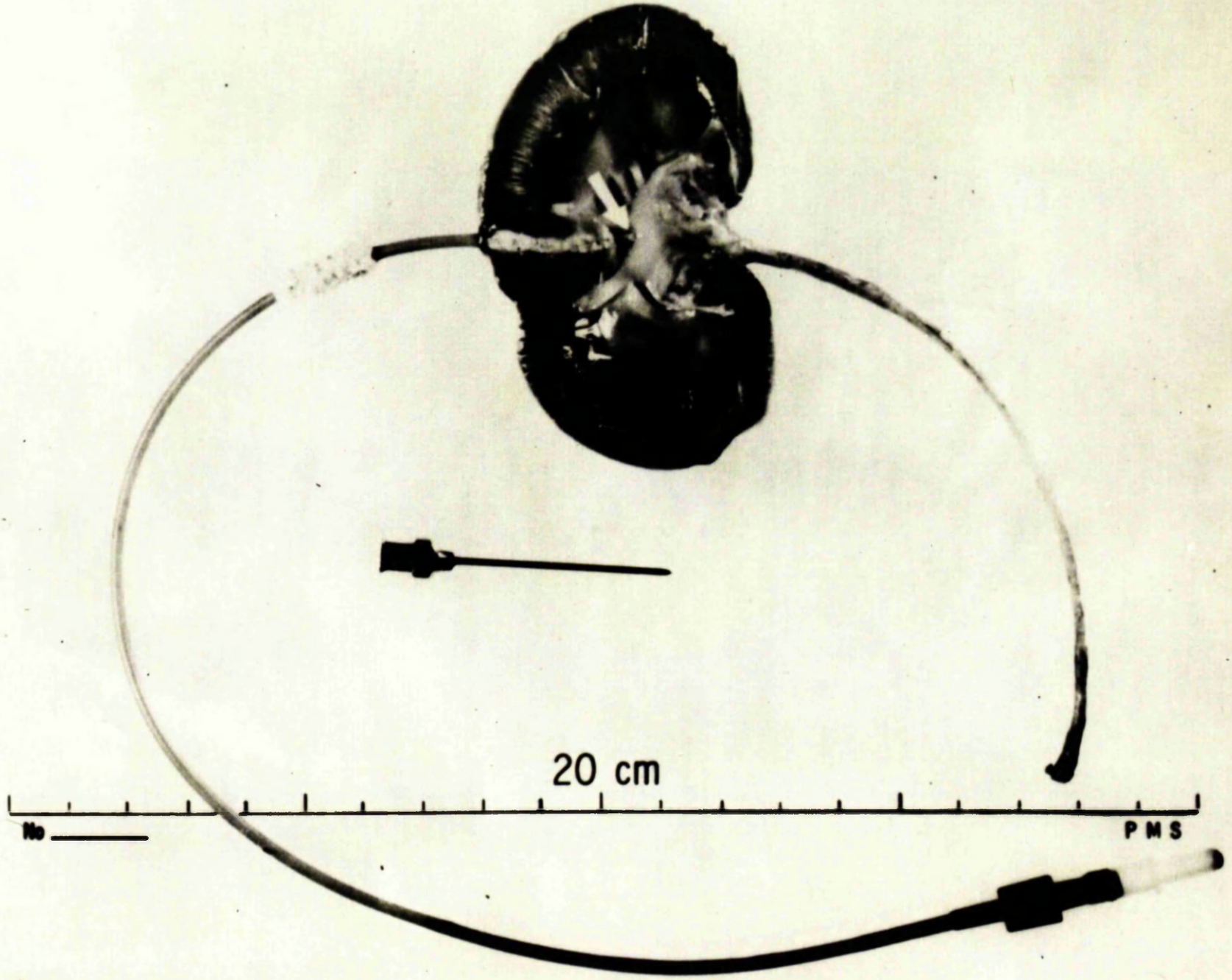

FIG. 2 SECTION OF KIDNEY SHOWING TIP OF MANOMETRIC TUBE IN RENAL PELVIS.

in the skin over the lower ribs.

A needle of the same internal diameter as the Teflon tube was inserted into the Neoplex tube to abut on the Teflon and a plastic cap sealed the tube.

In dogs 21, 45 and 46, two nephrostomy tubes were implanted in the same kidney.

In No.35, the Teflon inner tube was 2 cms longer so that the tip of the tube lay in the pelvi-ureteral region and a second manometric tube was inserted into the pelvis. The pelvi-ureteral Teflon inner tube was later removed so that the Neoplex tube was also available for measuring pressures in the renal pelvis.

PRESSURE RECORDING APPARATUS: The manometric equipment used consisted of inductance pressure transducers, carrier amplifiers, and a multi-channel recording camera*. The inductance transducer had a total range of 0 - 1,000 mm.Hg. and an accuracy within the frequency range of ½% The frequency response of the pressure head is 12 cycles per second, which is adequate for recording both pelvic and ureteric phasic

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* Manometric equipment provided by New Electronic Products, 360, Kennington Road, London S.E.11.

waves. The volume displacement for total deflection of the diaphragm is 0.0005 ml. The carrier amplifier has an output on the full pressure range of ½ volt and the oscillator is amplitude stabilised. Pressures were recorded on the 40 mm.Hg. range. Each recording was calibrated with a zero baseline and 50 mm.Hg., using a saline column equivalent. The light spots on the multi-channel recording camera were adjusted to a range of 5 cm for 50 mm.Hg. The recording camera could be adjusted for various paper speeds and 2.5 mm. per second was found to be the most satisfactory speed, although occasionally tracings were recorded at 8 mm. per second. Second intervals were marked with a time signal along the upper margin of the photographic paper.

The apparatus was sterilised with alcoholic chlorhexidine (Hibitane) solution, and for recording was filled with sterile physiological saline.

STANDARD PROCEDURE FOR RECORDING PELVIC PRESSURES: In the early part of the series the dogs stood in a Pavlov stand (Fig.3) but it was subsequently found preferable to have the animal recumbent in a metal trough (Fig.4)

The caps and nephrostomy tubes were carefully cleaned with alcoholic chlorhexidine solution. Sterile

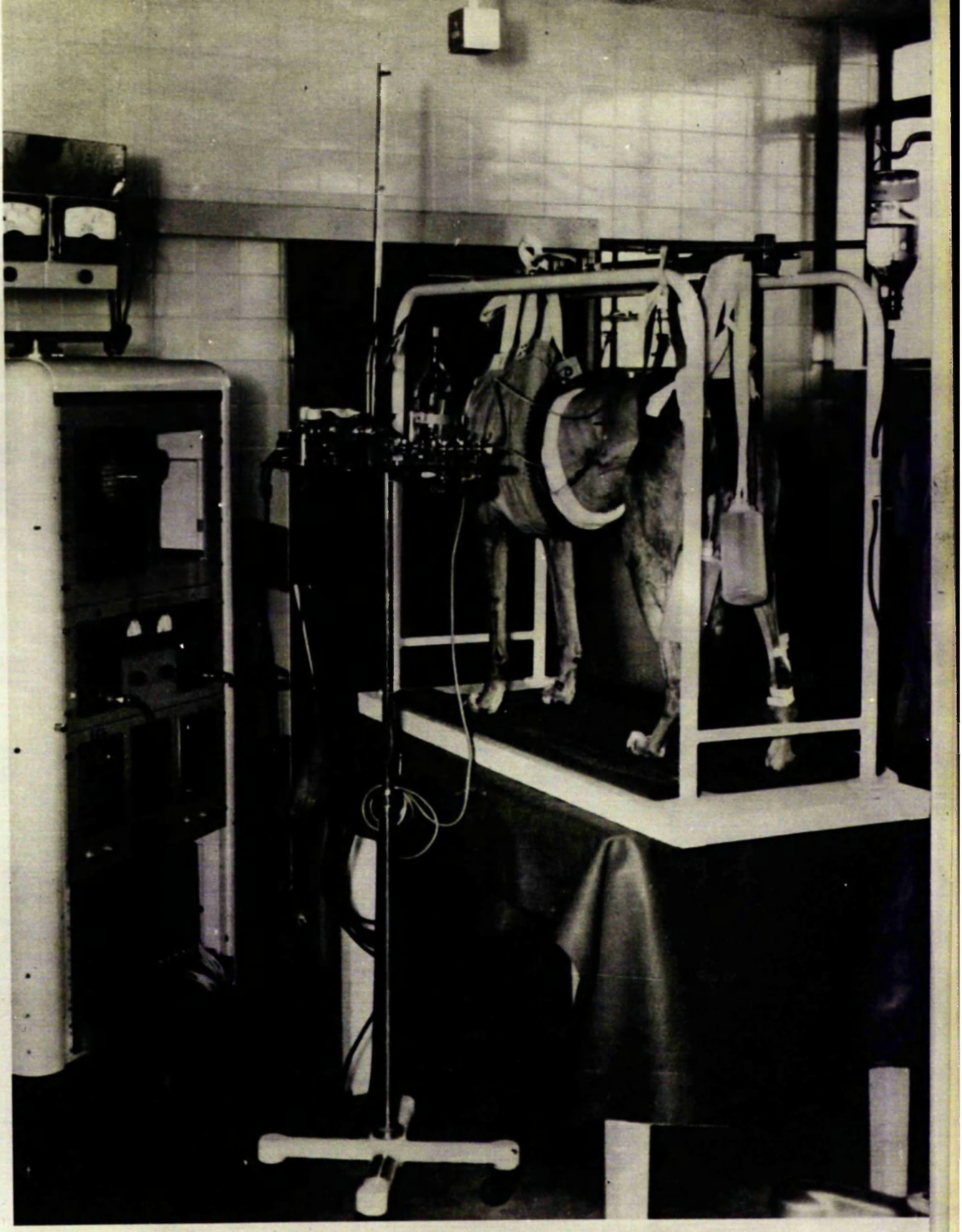

FIG. 3 DOG IN PAVLOV STAND DURING PRESSURE RECORDING EXPERIMENT.

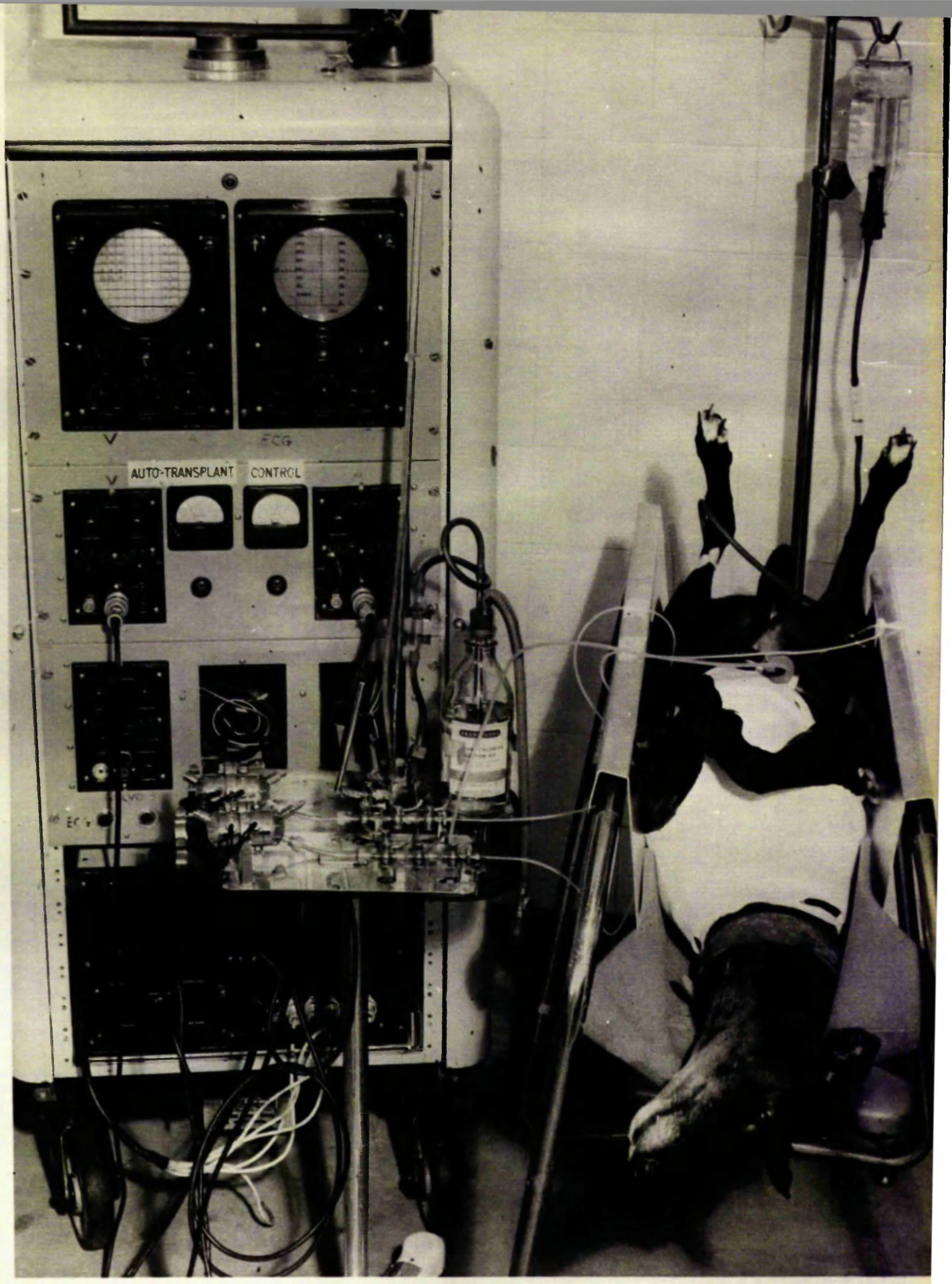

FIG. 4 DOG LYING IN TROUGH DURING PRESSURE RECORDING EXPERIMENT.

polythene tubing was connected to the manometers, flushed with saline to exclude air bubbles, and connected to the manometric tubes. The manometric heads were adjusted to kidney level and the polythene tubes secured with zinc oxide strapping to limit movements causing artefacts on the pressure trace. Respiratory waves were recorded by a piece of accordian tubing stretched round the dogs' chest, and pulse waves with an electrocardiograph. The animal was catheterised with a No.12 Ch. Malecot self retaining catheter and the intrapelvic pressure and urine flow rate were recorded. Diuresis was then induced and the pressures in the renal pelvis recorded at 5 minute intervals. Urine was allowed to drain freely from the catheter and the volume collected at the end of each 5 minute period noted. Maximal diuresis was reached at approximately 45 minutes and pressure records were generally taken to 60 minutes so that a total of 20-30 minutes of pressure tracings were recorded during each diuresis.

Osmotic diuresis was produced by infusing by vein 500 ml. of a solution composed of 200 ml 10% mannitol and 300 ml 0.9% Na Cl., over a period of 20 minutes followed by 500 ml. of a solution composed of 200 ml 10% mannitol and 300 ml 2% Na Cl., over a further

20 minutes. Water diuresis was produced by feeding the dog a litre of tepid water by gastric tube 15 minutes before pressures were recorded.

In a group of experiments, instead of measuring urine volume during diuresis, a polythene tube was threaded through the catheter into the bladder. This was connected to a manometric head and bladder pressures recorded. After maximal diuresis in both groups, the effect of either filling or emptying the bladder on pelvic pressures was also recorded, and in a few animals bladder pressure was raised by applying manual pressure to the abdomen during diuresis.

II

THE AUTOTRANSPLANTED KIDNEY PREPARATION: The technique of autotransplanting the kidney (Dempster 1954) was used to deprive the upper urinary tract of extrinsic innervation. In this group of preparations, with the exceptions of Nos. 40, 42 and 43, either the right or left kidney was transplanted to the iliac vessels on the same or the opposite side of the pelvis. The renal artery was anastomosed end to end to the internal iliac artery if this vessel was considered to have an adequate lumen, or end to side to the external iliac if the internal iliac artery was small. The renal vein was anastomosed

end to end to the common iliac vein. The ureter was implanted in the bladder by the double flap technique, care being taken to avoid constricting the ureter ic lumen. A manometric tube was implanted in the renal pelvis before the kidney was transplanted.

In dogs 10, 17, 22 and 23, the opposite kidney was also intubated with a nephrostomy tube.

The contralateral kidney was removed at the time of the autotransplantation operation in dogs 26, 27, 28, 29 and 30; in dogs 36, 37, 38, 39 and 44 the opposite kidney was removed 7 days before the autotransplantation operation. In dogs 31, 32, 33, 34, 40 and 42, the opposite kidney was removed 7 days after the autotransplantation.

In dogs 40, 42 and 43, the left kidney and ureter were completely mobilised to the uretero-vesical junction, the renal vessels being divided and the ends anastomosed.

In No.43, seven weeks after the original operation the control and the autotransplanted ureters were transected at the uretero-vesical junction and re-implanted in the bladder, and in No.22, the control ureter was also transected and implanted in the bladder.

The standard procedure for recording intrapelvic pressures was used in the autotransplanted preparations.

IIIa

URETERIC OBSTRUCTION: Simultaneous pelvic and ureteric pressures were recorded during acute obstruction in seven preparations. These records were made in five instances, (dogs No. 6, 7, 11, 15 and 20) before definitive operations, and in two (dogs No.14 and 23) as terminal experiments, by the following procedure: After the animal was anaesthetised, a diuresis was induced by infusing 500 ml. of 0.9% Na.Cl. into a vein. The abdomen was then opened by a mid-line incision, and pelvic pressures recorded. The ureter was then transected at the mid-point of the pelvic portion. A No.3 polythene tube (internal diameter 1.5 mm.) filled with saline and connected to a second manometric head, was inserted into the proximal end of the ureter and secured with thread. Pressures were recorded from the renal pelvis and ureter until no further change was observed in either pressure trace.

b.

URETERO-COLIC ANASTAMOSIS: Through a lower abdominal mid-line incision a Coffey uretero-colic anastamosis was made in eight dogs. Both ureters were transplanted in dog No. 5 and in dogs No. 1, 3, 7, 11, 14, 15 and 20, a single ureter was transplanted. Pelvic pressures were recorded at low urine flow rates and during

the standard diuresis but because of faecal contamination it was not possible to record urine flow accurately.

Descending pyelograms were made at known pelvic pressures to ascertain the degree of dilatation occurring in the upper urinary tract. After the animal was sacrificed, urine was aspirated from the nephrostomy tube and replaced with an equal volume of 50% W/V barium sulphate (Raybar Cream) in gelatine. The upper urinary tract was then removed from the animal and X-rayed.

IV ADDITIONAL STUDIES:

a) Intrapelvic pressure response to direct pelvic infusion: In dogs 21, 35, 45 and 46, the pelvis was infused with urine or saline at a constant rate by means of a Palmer slow-injection apparatus. Pelvic pressures were recorded through one manometric tube and the solution was infused through a second tube. The pressure in the injection line was also recorded.

b) Synchronous pelvic pressure and radiographic studies: These were obtained in two anaesthetised dogs. In the first (No.45) pyelograms were made at varying rates of infusion of 22% sodium diatrizoate (Hypaque) in

saline. In the second (No.46) pelvic and upper ureteric movements were studied with the X-ray image intensifier. Contrast was obtained with intravenous and infused Hypaque. Cineradiography was combined with manometric records during the infusion of 45% Hypaque into the left pelvis at 3.4 ml. per minute.

c) Pressures in pelvis and pelvi-ureteral zone: In dog 35, the tip of the Teflon tube was positioned in the pelvi-ureteral region of a solitary kidney, and the tip of a second manometric tube recorded from the pelvis. Pressures were recorded from both tubes during the standard diuresis and during infusion experiments.

d) Pressures in pelvis and ureter: Pressures were measured from the pelvis by the manometric nephrostomy tube, and from the ureter by means of various modifications of a T-or L-tube. Anaesthesia, exposure of the kidneys and the insertion of the nephrostomy tube, were carried out by the previously described methods. The ureter was then exposed by incising the peritoneum along its lateral margin. A nylon stilette was passed through the nephrostomy tube, down the ureter to the site selected for the insertion of the manometric tube. The ureteric wall was then

incised on the tip of the stilette.

In dogs No. 3, 4 and 23, a Teflon T tube 1½ cm. long, internal diameter 1.8 mm. was implanted through a 0.5 cm longitudinal incision in the ureter. The longer limb of the ureteric arm was inserted through the incision into the proximal ureter. The shorter limb could thus be introduced into the distal ureter and the T tube then moved distally.

The incision in the ureter was then repaired over the longer arm of the T tube with 5-0 interrupted silk sutures to obtain a watertight fit round the side arm. A No.8 Neoplex tube taken through a retroperitoneal tunnel and the flank muscles to a puncture wound on the skin, was pushed over the sidearm, avoiding angulation or rotation of the T tube. The Neoplex tube was anchored in the flank muscles by a cuff of Ivalon sponge.

In dog No.9 a puncture wound was made in the ureteric wall and the nylon stilette passed through it. The short limb of an L shaped Teflon tube was introduced over the stilette into the ureter and the stilette withdrawn.

In dog No.10, by a similar technique, a 1.3 c.m. stainless steel tube, internal diameter 1.2 mm. was

inserted into the ureter over the nylon stilette. A sidearm was then inserted into the puncture wound in the ureter and screwed into a side hole in the tube in the ureter to form a T-tube.

In dogs No. 13, 14, 18 and 19, similar T tubes, in which the surface had been coated with a thin film of Teflon, were inserted in the ureter. A modification in which the ureteric part was of U section was inserted in Dog 15.

The standard procedure was used to record pelvic and ureteric pressures.

SOURCES OF ERROR

Errors arising from the animals used:

The female greyhound was considered to be the most suitable dog available for study as the kidneys are large and relatively free from canine nephritis (Dempster and Daniel 1956). Animals were excluded from the series if pre-operative blood urea exceeded 25 mgm %, or if at operation there was more than minimal scarring of the kidney.

Errors arising from the manometric tubes:

As the initial method of inserting the manometric tube required a small incision in the renal pelvis, this was replaced with the modified Seldinger technique. With this method there was no damage to the pelvis, and it was considered that the trauma to the renal parenchyma was relatively insignificant. As the spaces in the Ivalon sponge are infiltrated by collagen fibres (Struthers 1960), a living bond thus anchored the nephrostomy tube. Minimal crusting occurred where the tube emerged at the skin wound and as the light weight

needle and plastic cap did not drag on the skin, most dogs seemed unaware of their inert appendage and the only protection required was a stockinette coat (Fig. 5). Occasionally, however, for no apparent reason, a dog would bite through its manometric tubes. Urine was not routinely cultured, but only in the hydronephrotic group did obvious infection develop. In this group, infection occurred rarely after osmotic diuresis, frequently after excretion and descending pyelography, and inevitably if the cap came off the manometric tube permitting the escape of urine. On bench testing, the manometric tube was shown not to damp phasic pressure waves. In the dog the tube was considered to function satisfactorily if pressure on the dog's abdomen was immediately reflected in a slight increase in intrapelvic pressure, and if flushing the tube with a few drops of saline produced an increase in pelvic pressure with phasic waves, followed by a return to the original pelvic pressure.

Temporary unsatisfactory function of the manometric tube occasionally occurred and was probably caused by the tip of the tube forming a flap valve with the pelvic mucosa. Permanent failure was caused by the tube retracting into the renal parenchyma or becoming blocked

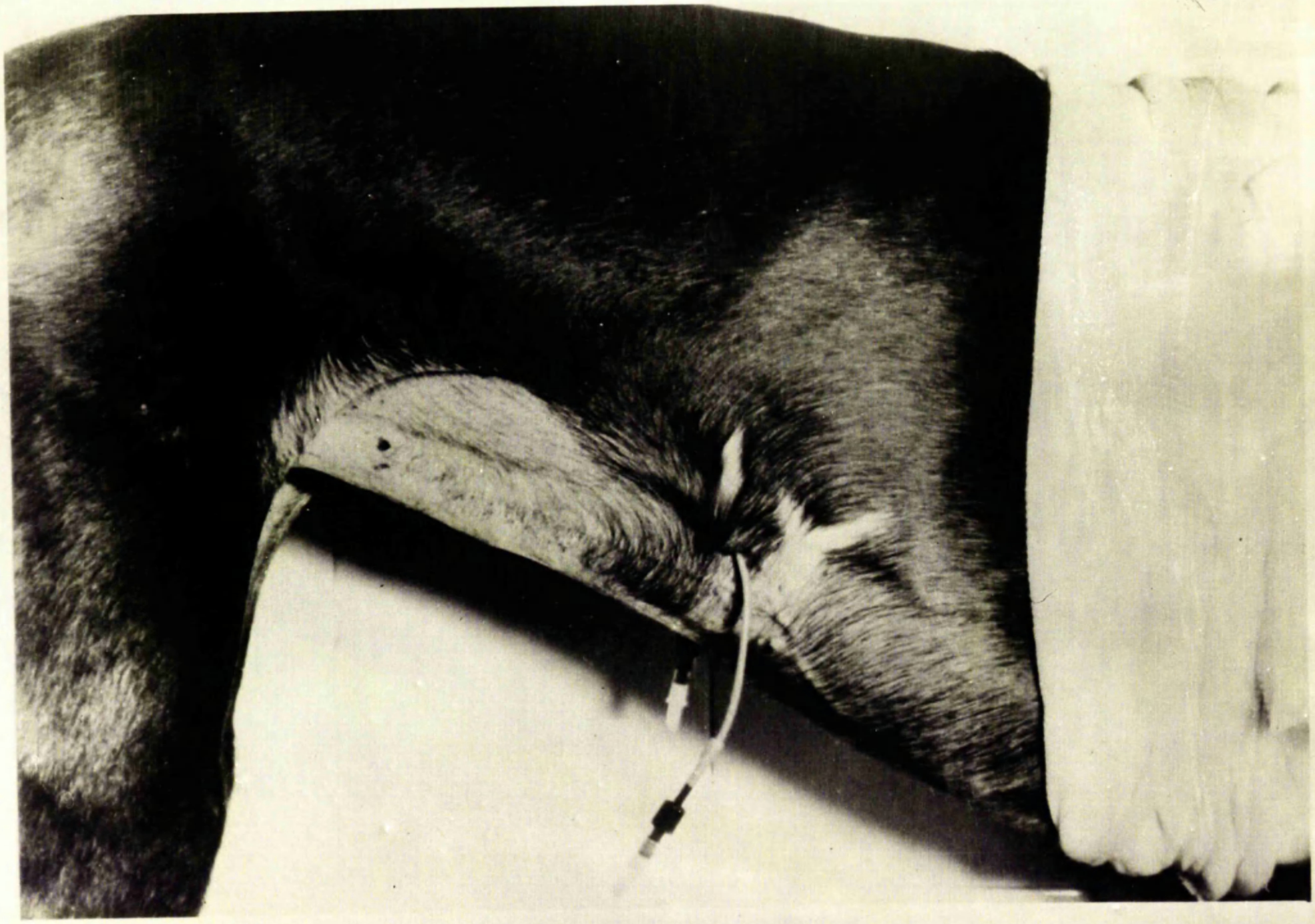

FIG. 5 STOCKINETTE COAT ROLLED TO DISPLAY MANOMETRIC TUBE.

with calcareous deposit.

So that the pressure records could be correctly interpreted certain extrinsic causes of pelvic pressure waves had to be identified. These waves were due to:

a) Respiration. Small fluctuations were frequently observed in the pelvic pressure trace. These fluctuations were synchronous with the respiratory cycle, the wave peaks coinciding with maximal inspiration (Fig. 6). The amplitude of the wave varied with the depth of respiration, and respiratory waves were most marked at high urine flow rates.

b) Pulse. Rapid oscillations, coinciding with the pulse and most obvious during diuresis (Fig. 7) were also noted in the pressure trace.

c) Muscular Movements. Irregular waves, due to muscle movements made some pressure records difficult to interpret. Attempts to identify these waves by a tube in the perirenal space were only temporarily successful (Fig. 8), as the perirenal tubes became encased in fibrous tissue. By recording pressures when the greyhound was recumbent in a padded metal trough, irregular voluntary and involuntary movements were reduced to a minimum. Simultaneous records from both

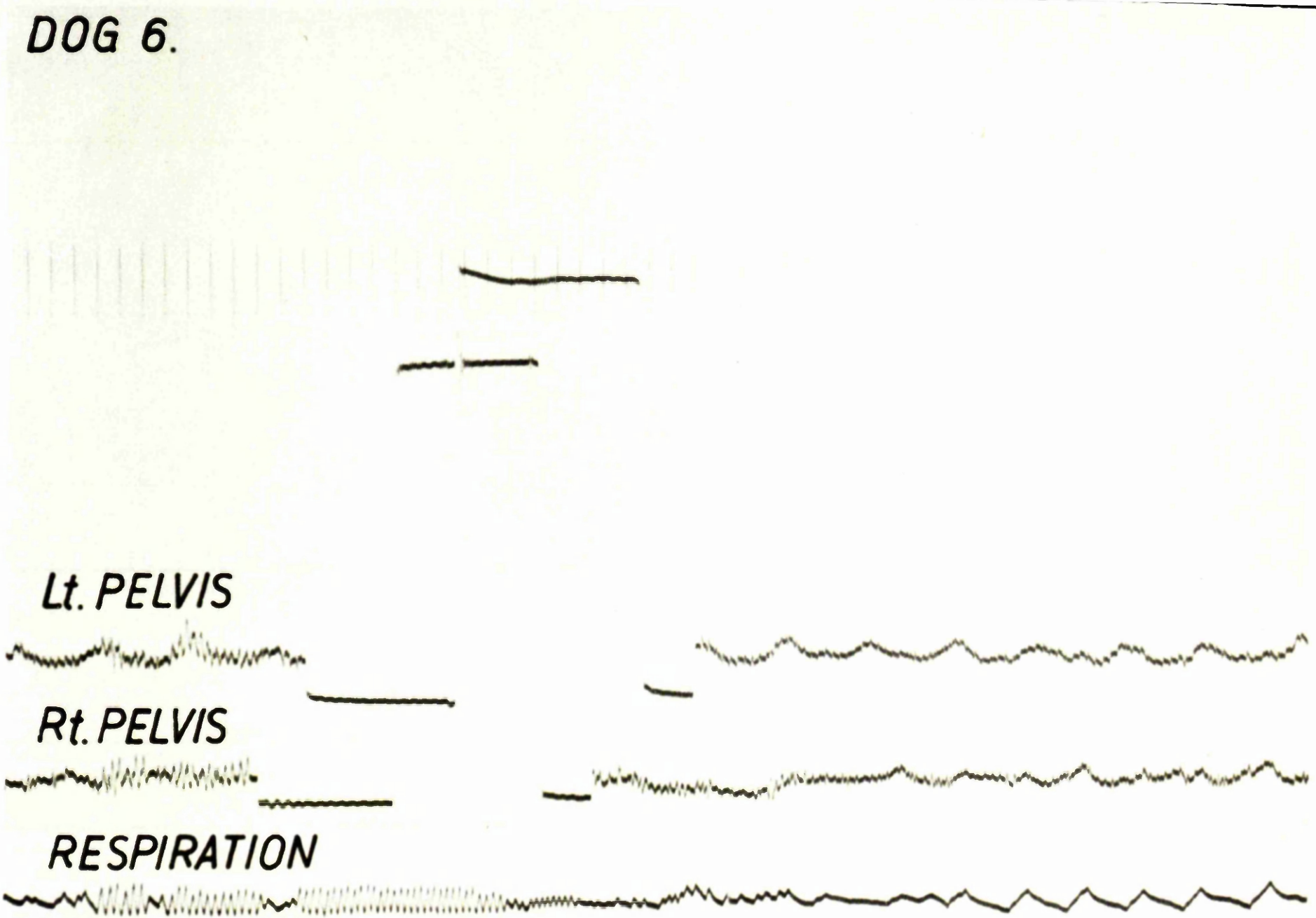

FIG. 6 EFFECT OF RESPIRATIONS ON PRESSURE RECORD.

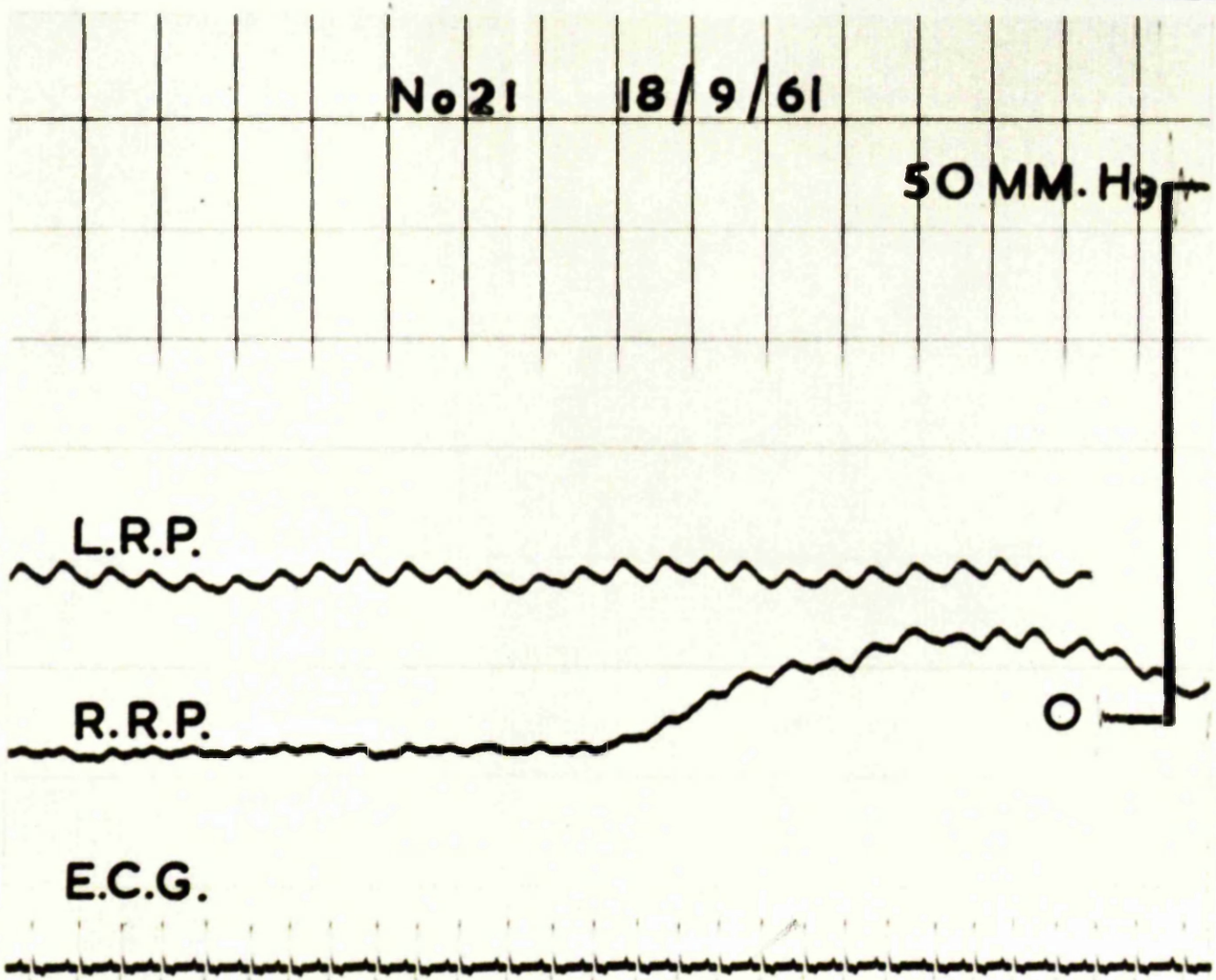

FIG. 7 EFFECT OF PULSE BEAT ON PRESSURE RECORD.

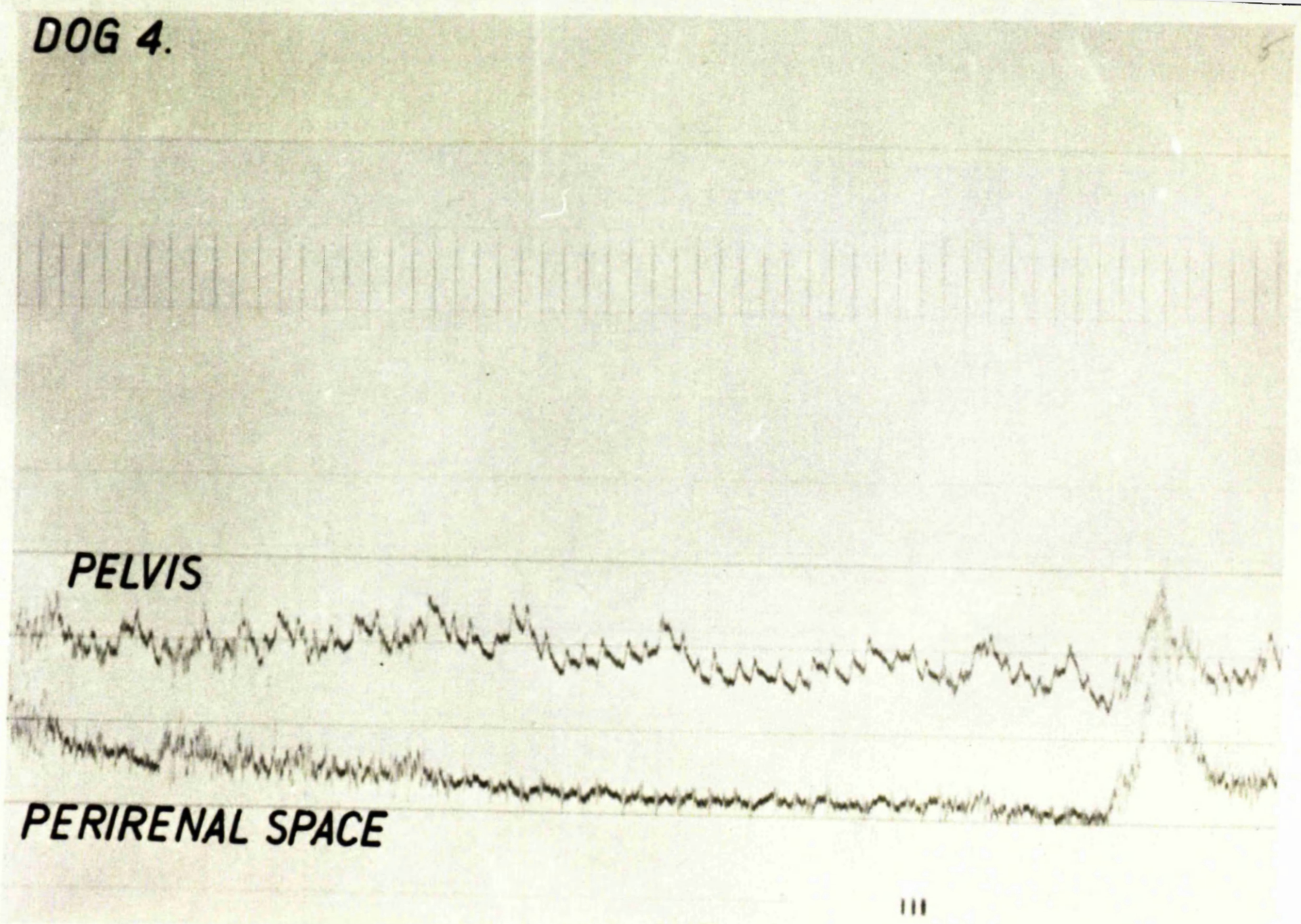

FIG. 8 IRREGULAR WAVES PRODUCED BY MUSCULAR MOVEMENTS.

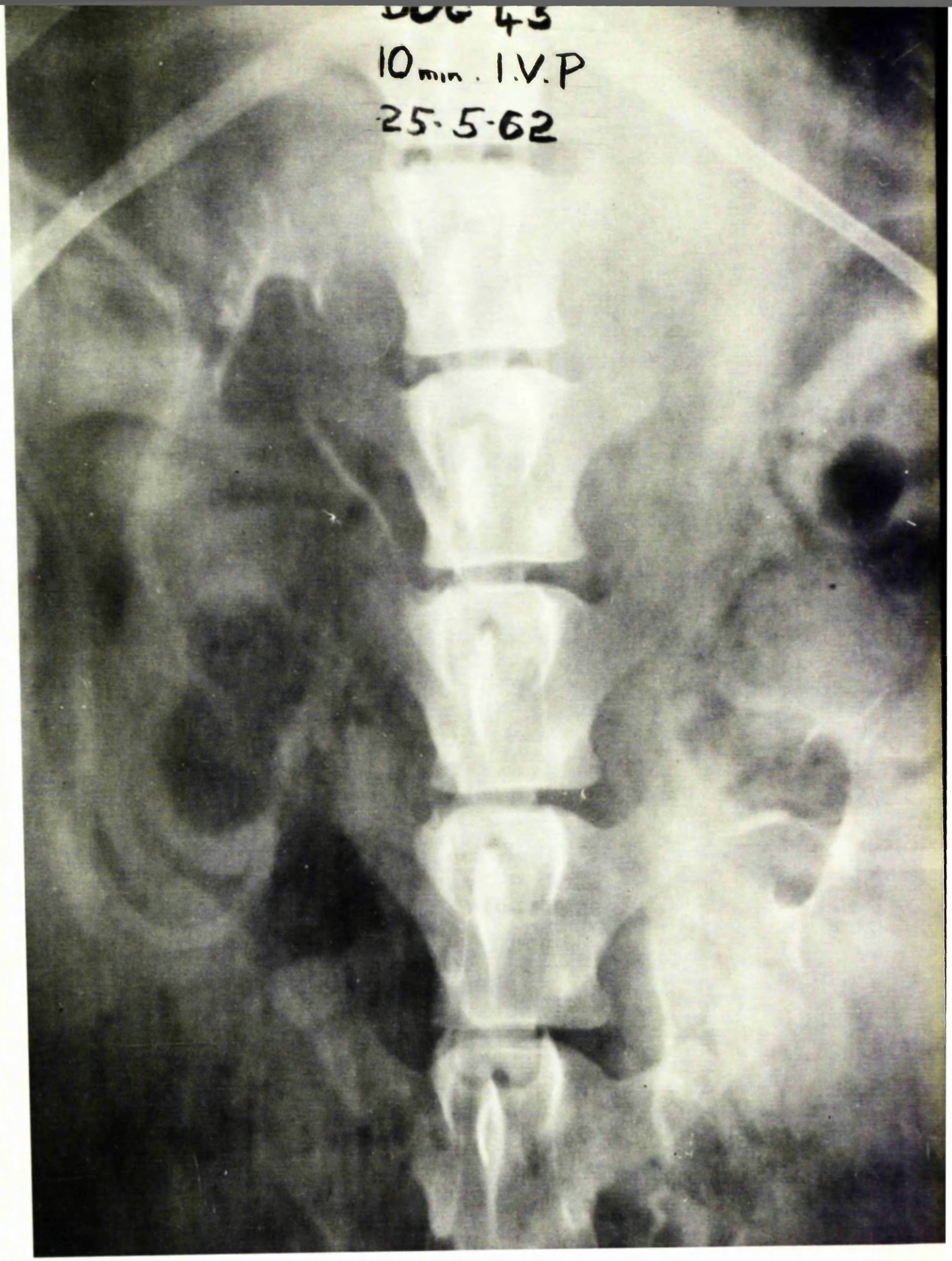

FIG. 9 EXCRETION PYELOGRAM ILLUSTRATING NORMAL APPEARANCE OF RENAL PELVES IN RIGHT AND LEFT(AUTOTRANSPLANTED) KIDNEYS.

pelves also facilitated recognition of these extraneous pressure waves.

It was shown by excretion urography (Fig. 9) that the outline of the upper urinary tract was not distorted by the manometric tubes, and after sacrifice of the animal it was confirmed that the manometric tube lay in the correct position.

RESULTS

I PELVIC PRESSURES IN THE "NORMAL" KIDNEY

BASAL PRESSURES IN THE RENAL PELVIS: Numerous basal pelvic pressure measurements were made in each dog and the results in 20 dogs are recorded in Table I. The pressure range was 0-10 mm Hg. with an average pressure of slightly less than 2 mm Hg. The basal pressure record (Fig. 10) was generally free from phasic contraction waves but if these did occur they were of low amplitude.

PELVIC PRESSURE AND URINE FLOW RATE: Osmotic diuresis produced a greater and more predictable increase in urine flow rate than water diuresis, and with a few exceptions was used throughout the study. With increasing urine flow rates the basal pressure was modified by an increase in the standing, or minimum pressure in the pelvis, and phasic pressure waves developed and tended to increase in amplitude with increasing urine flow. There appeared to be no constant flow rate at which these contraction complexes developed. In some recordings the phasic waves were of constant shape and rhythm (Fig. 11) while in others

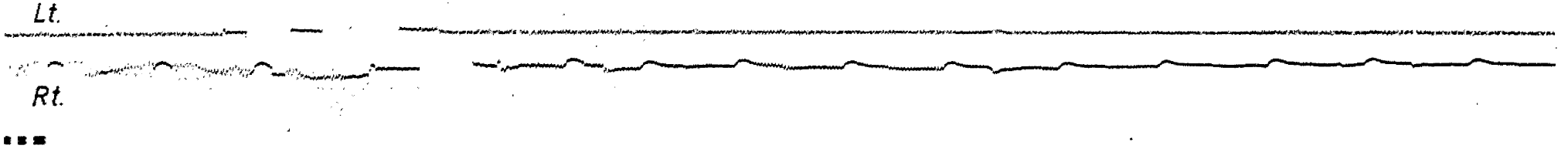

FIG. 10 TWO MINUTE RECORDING FROM RENAL PELVES DOG 8 ILLUSTRATING LOW BASAL PRESSURES. CALIBRATION 50 mm.Hg. SIGNALS DENOTE SECOND INTERVALS.

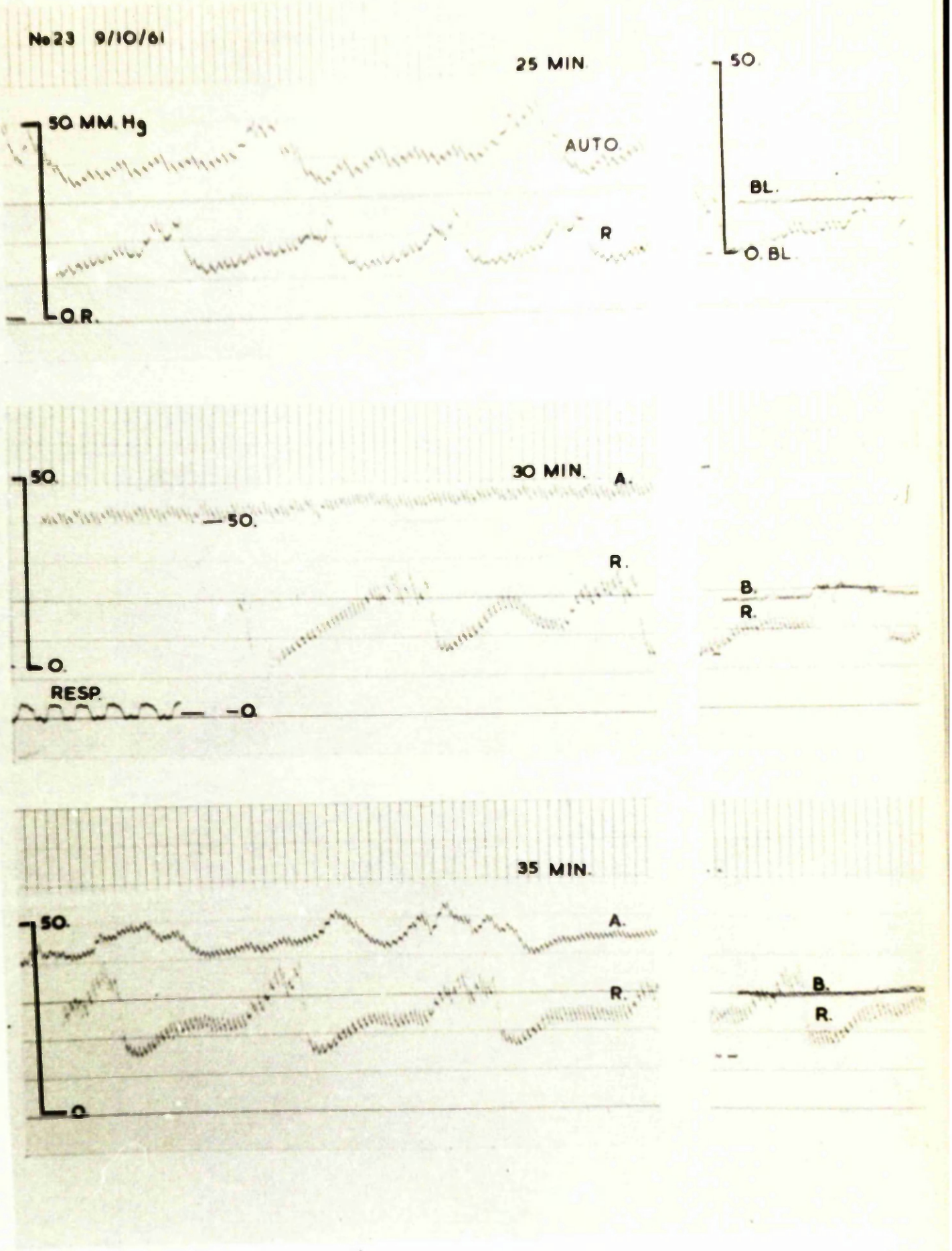

FIG. 11 ILLUSTRATES REGULAR PHASIC CONTRACTION WAVES IN RIGHT (R) PELVIS DURING DIURESIS

the waves were fused, and in others there was no apparent spatial or temporal pattern. Because of this irregularity, the maximum and minimum pelvic pressures were determined from a 20 second recording made at each five minute period. These pressures were plotted against the volume of urine secreted during the previous five minute interval and it was found that the increase in urine flow was directly reflected in an increase in intrapelvic pressure (Fig. 12 A B & C) although the relationship was not strictly linear. A similar pressure response occurred in repeated diuresis in the same dog and in the other dogs in the series. The highest standing pelvic pressures and the maximum pelvic pressures recorded for each animal during diuresis when the bladder was empty are reported in Table I

PELVIC PRESSURE AND INTRAVESICAL PRESSURE: Basal intrapelvic pressure was uninfluenced by changes in intravesical pressure. When pelvic pressures and bladder pressures were recorded during diuresis (Fig 13 A & B) the standing pelvic pressures and the amplitudes of the phasic contraction waves at each five minute period were much greater than those recorded when the bladder was maintained in an empty state by

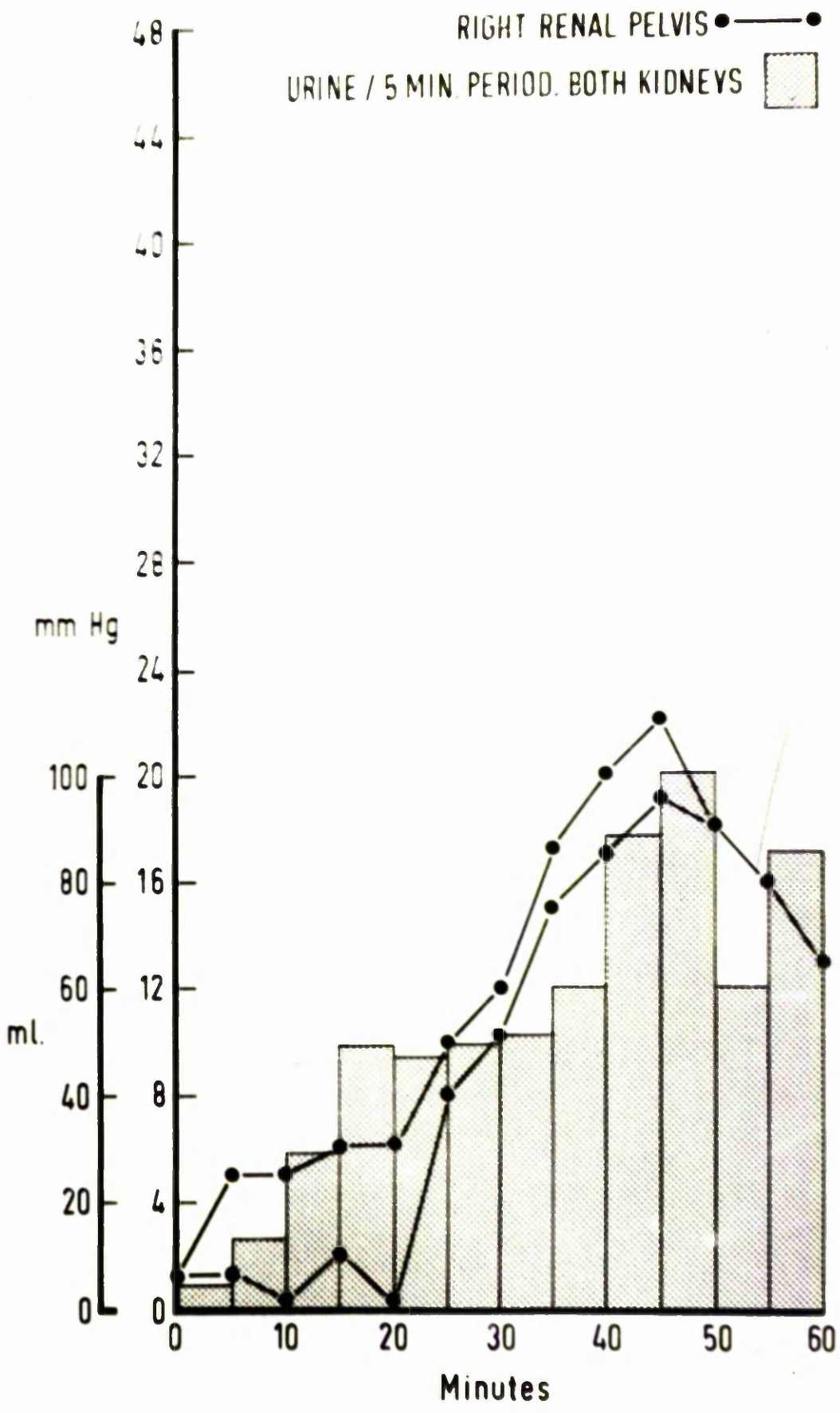

FIG. 12A MAXIMUM AND MINIMUM PELVIC PRESSURES RELATED TO URINE FLOW RATES DURING DIURESIS - DOG 20

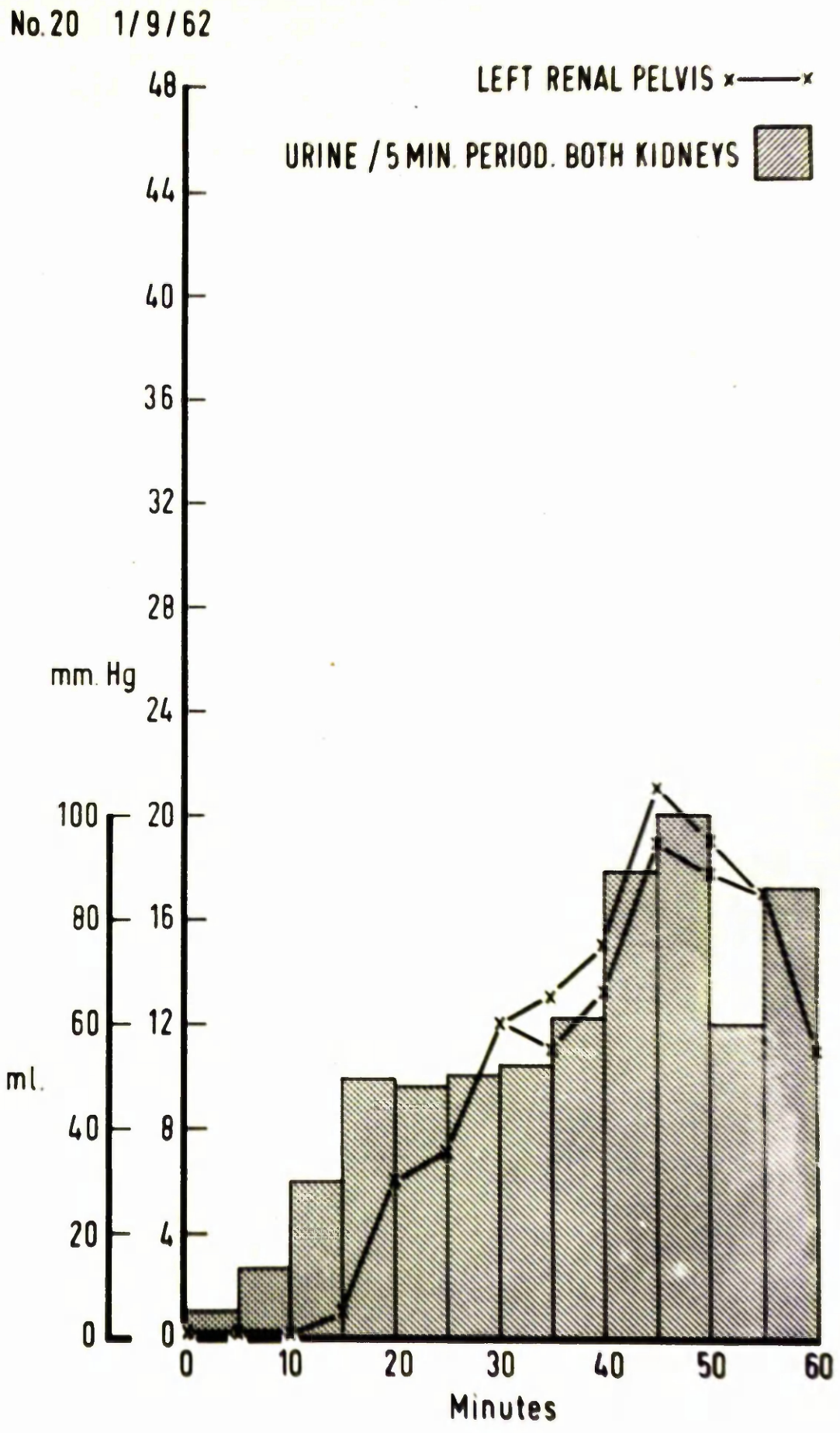

FIG. 12B MAXIMUM AND MINIMUM PELVIC PRESSURES RELATED TO URINE FLOW RATES DURING DIURESIS - DOG 20.

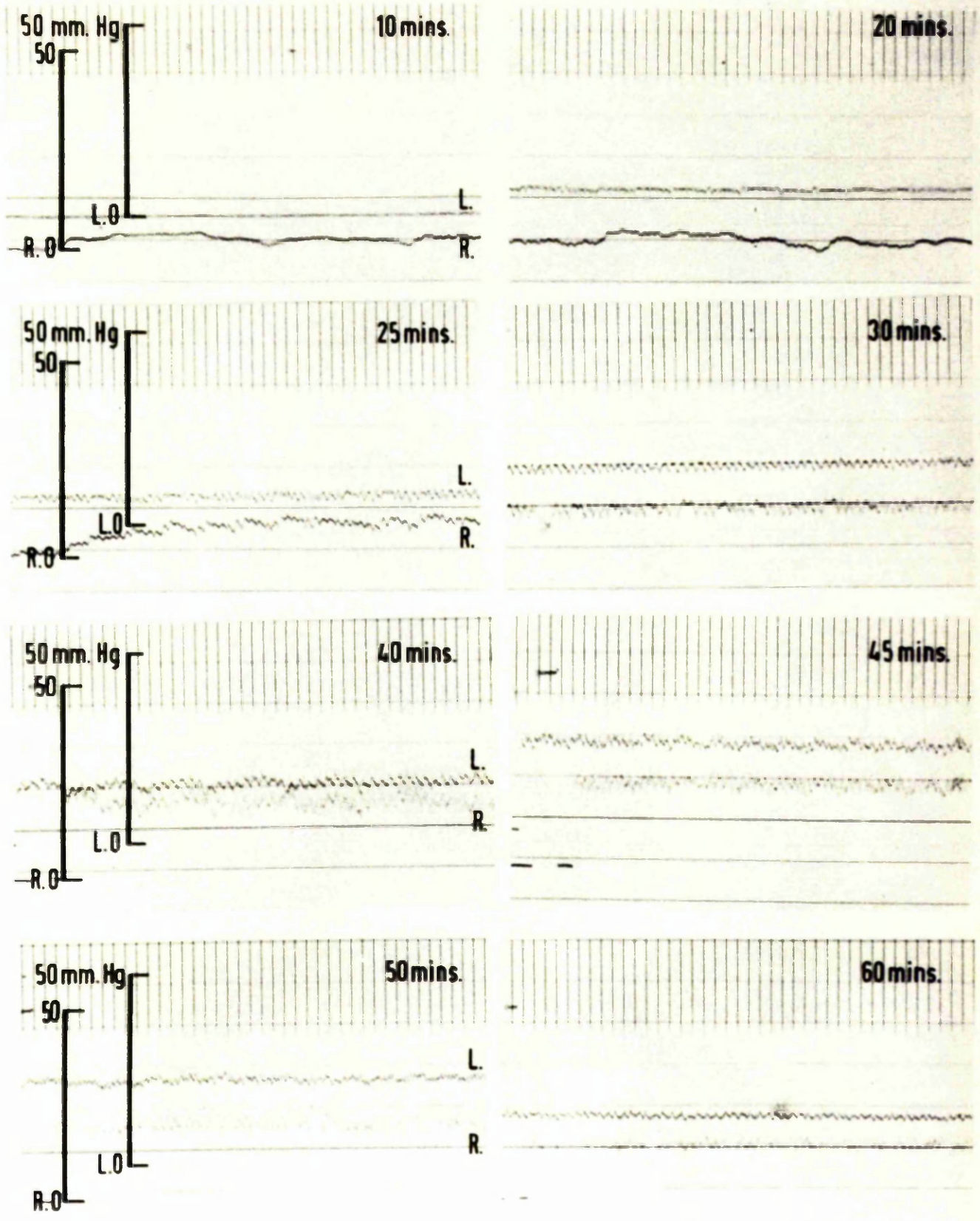

FIG. 12C RECORDINGS OF THE PRESSURES IN BOTH RENAL PELVES - DOG 20.

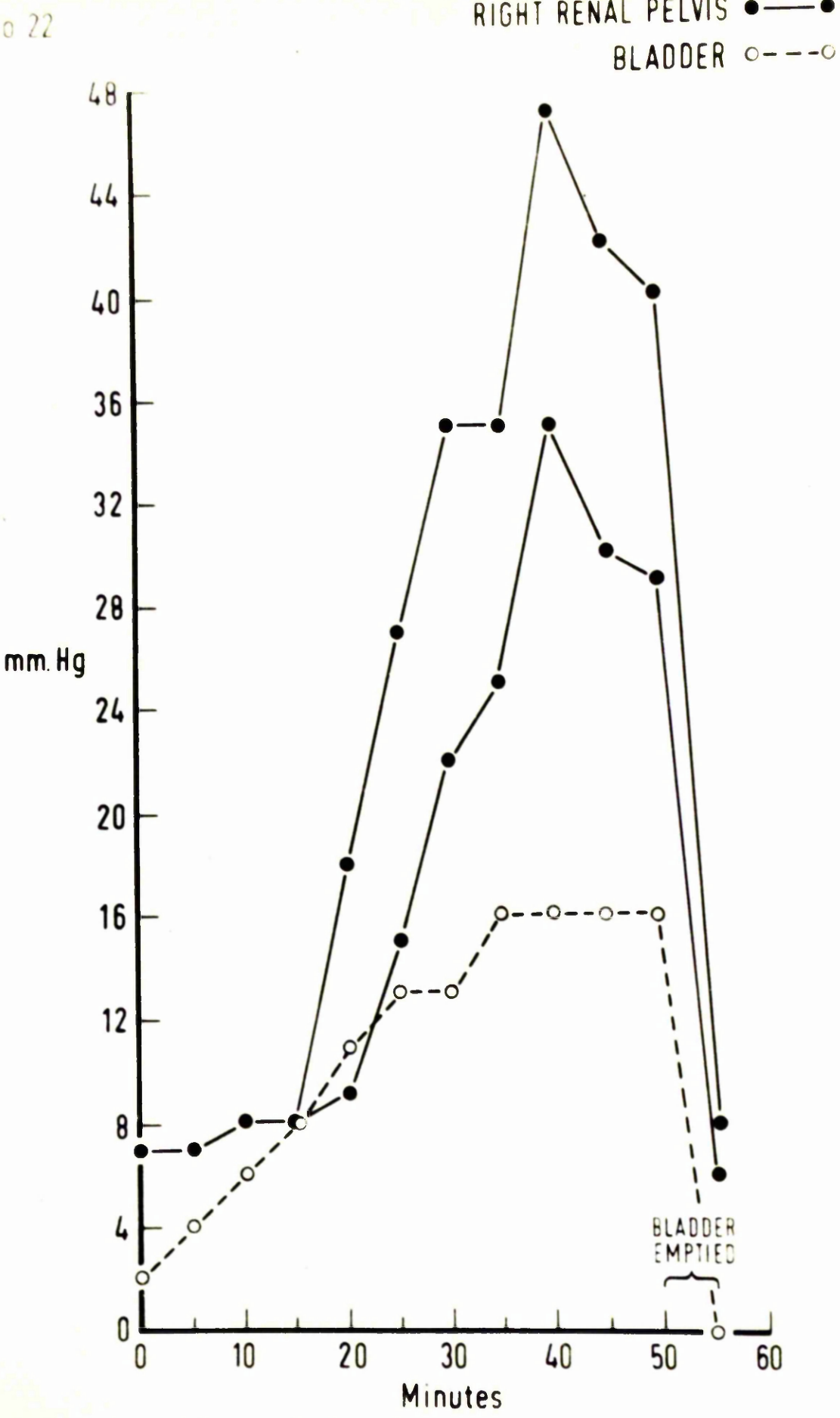

FIG. 13A PELVIC PRESSURES RELATED TO INCREASE IN BLADDER PRESSURE DURING OSMOTIC DIURESIS.

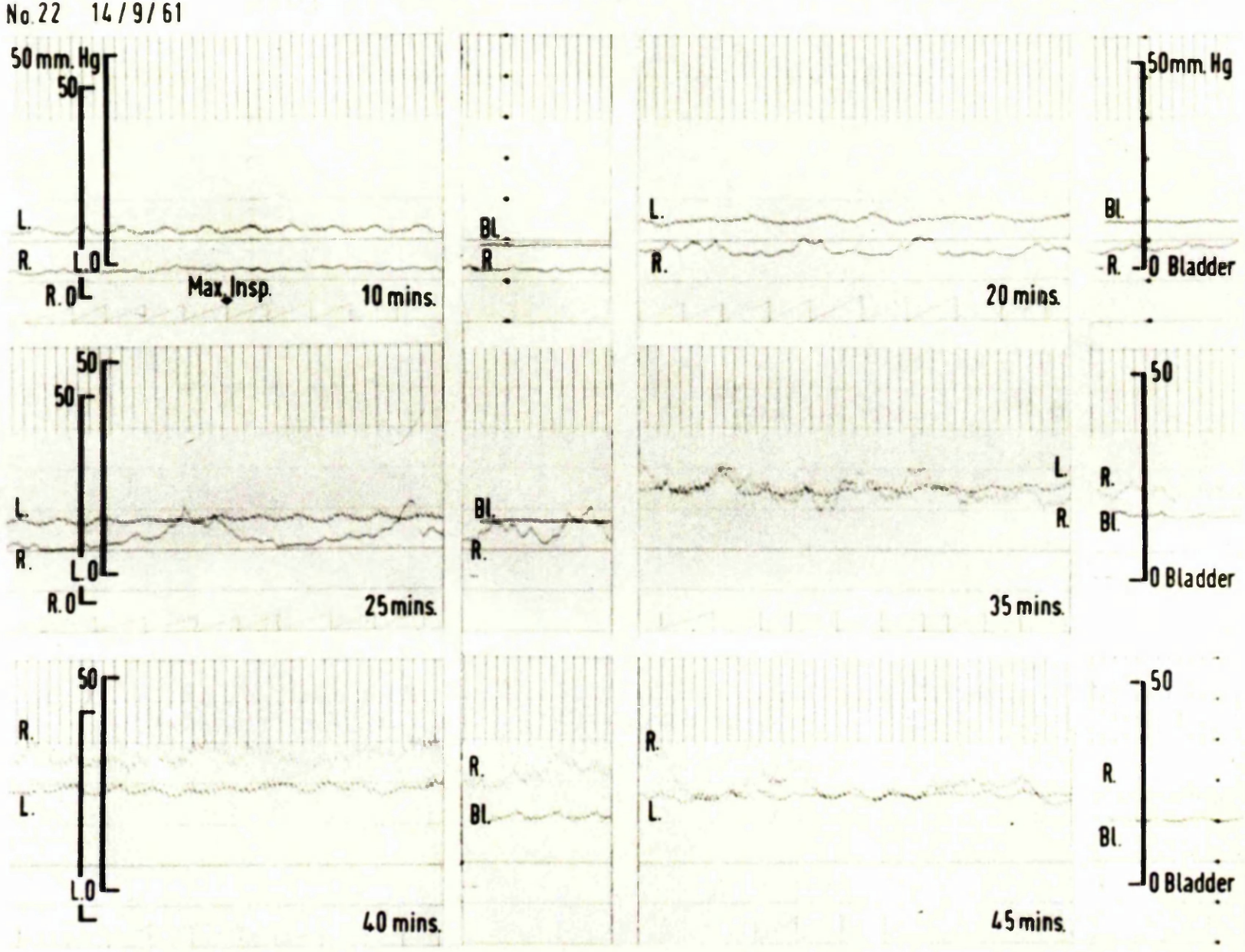

FIG. 13B RECORDINGS OF THE PRESSURES IN THE BLADDER AND RENAL PELVES.

catheter drainage.

It was also found that the pelvic pressures recorded during diuresis could be increased if the pressure in the bladder was raised by infusing saline into the bladder (Fig. 14) and that at comparable urine flow rates during diuresis alterations in bladder pressure were directly reflected by changes in intrapelvic pressures (Fig. 15). At these high pelvic pressures the arterial pulse wave produced deflections of a few mm Hg. in the pressure record (Fig. 16).

Emptying the bladder by catheter over a period of a few minutes resulted in a fall in the standing pressure in the renal pelvis, and the phasic contraction complexes decreased in amplitude (Fig. 17). It was demonstrated that the fall in pelvic pressures was not due to a lessening of the urine flow rate as refilling the bladder to its original pressure restored intra-pelvic pressures to the previous levels (Fig. 18). These changes in pelvic pressure were also demonstrated during water diuresis (Fig. 19). The modification of pelvic pressure response associated with changes in bladder pressure during diuresis, was recorded in repeated experiments in the same dog and in the other dogs in the series. The highest standing pressures and

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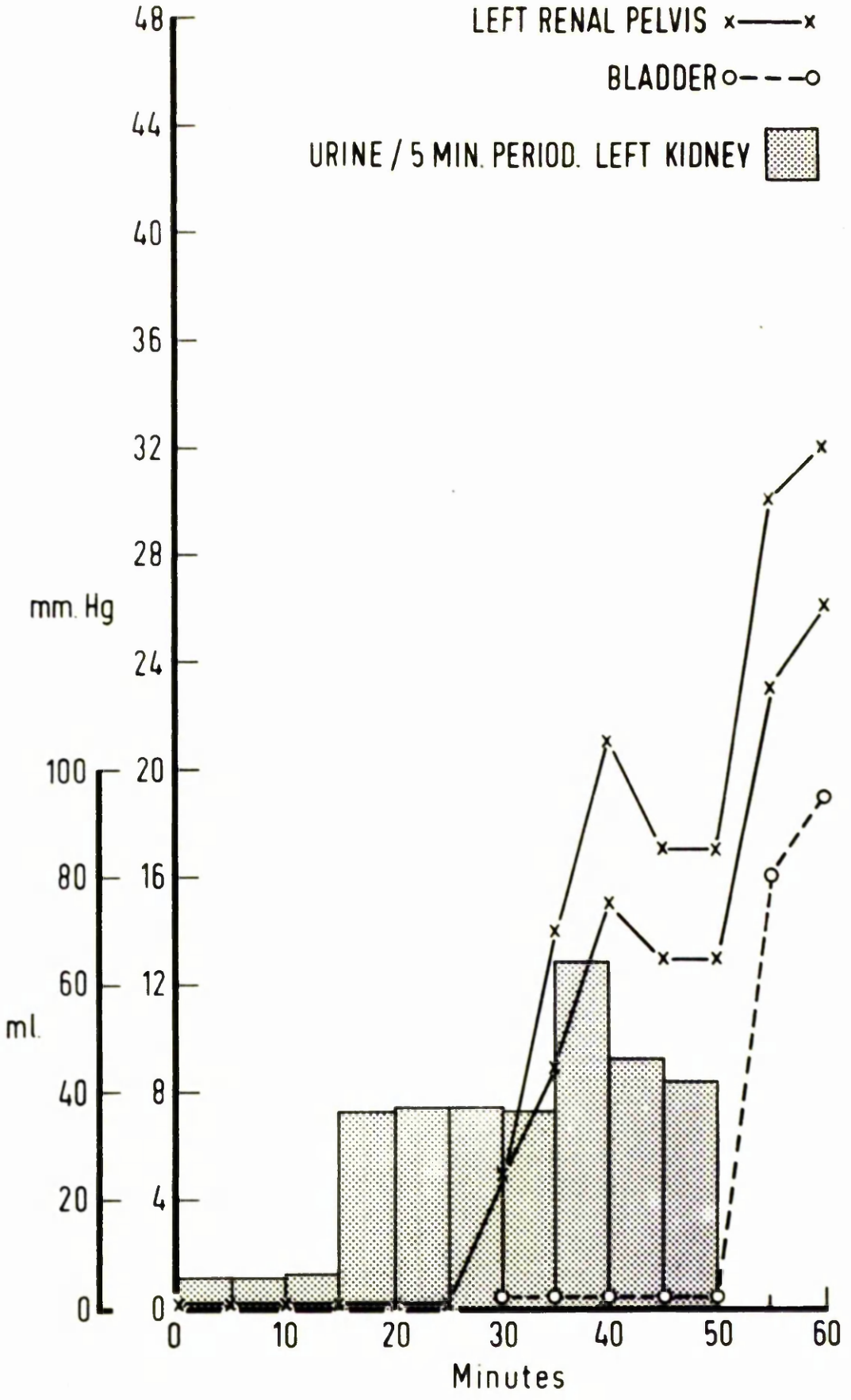

FIG. 14 ACUTE INCREASE IN BLADDER PRESSURE DURING DIURESIS RESULTS IN RISE IN RENAL PELVIC PRESSURE.

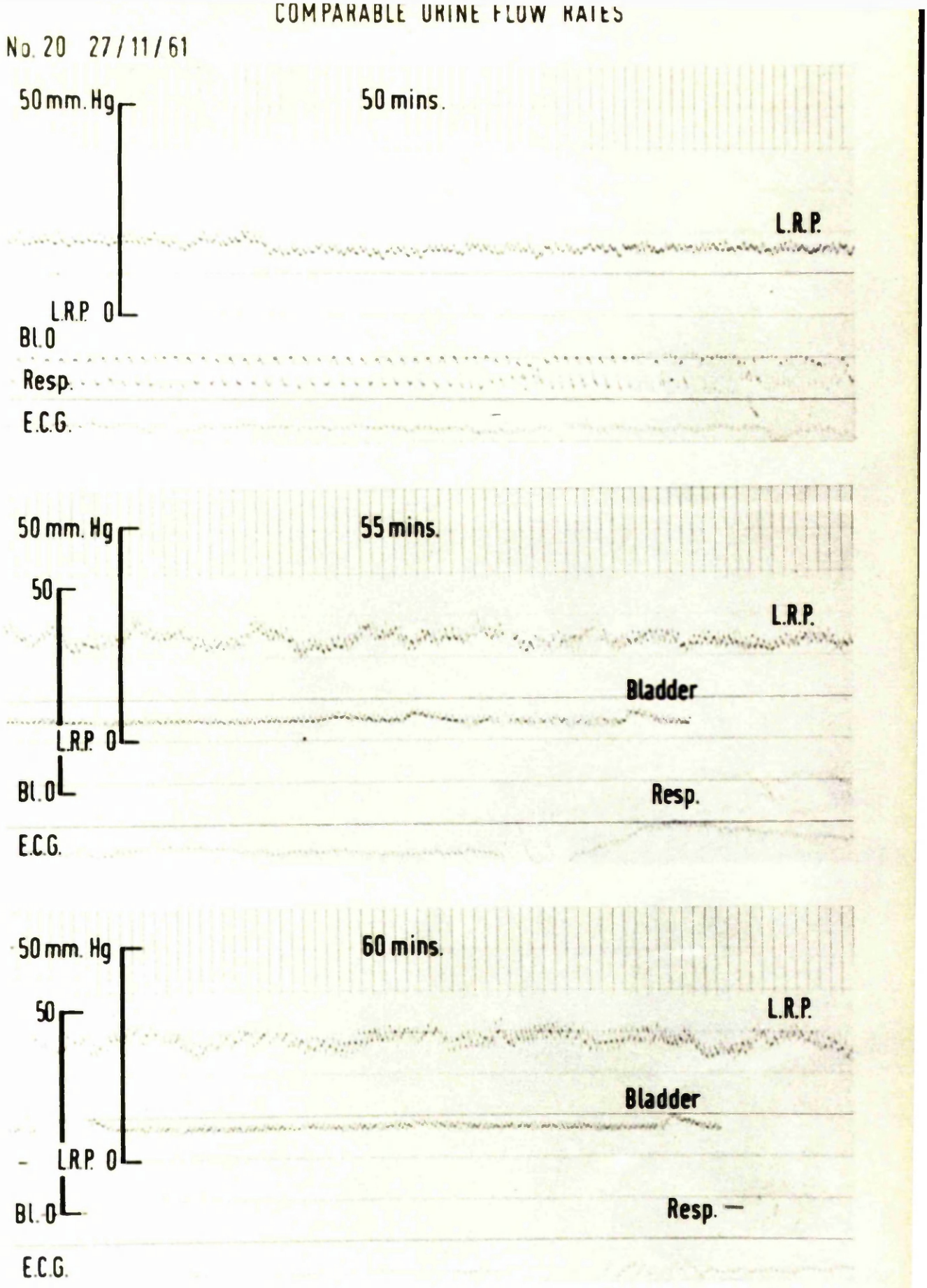

FIG. 15 AT COMPARABLE URINE FLOW RATES ALTERATIONS IN BLADDER PRESSURE ARE DIRECTLY REFLECTED BY CHANGES IN INTRAPELVIC PRESSURE.

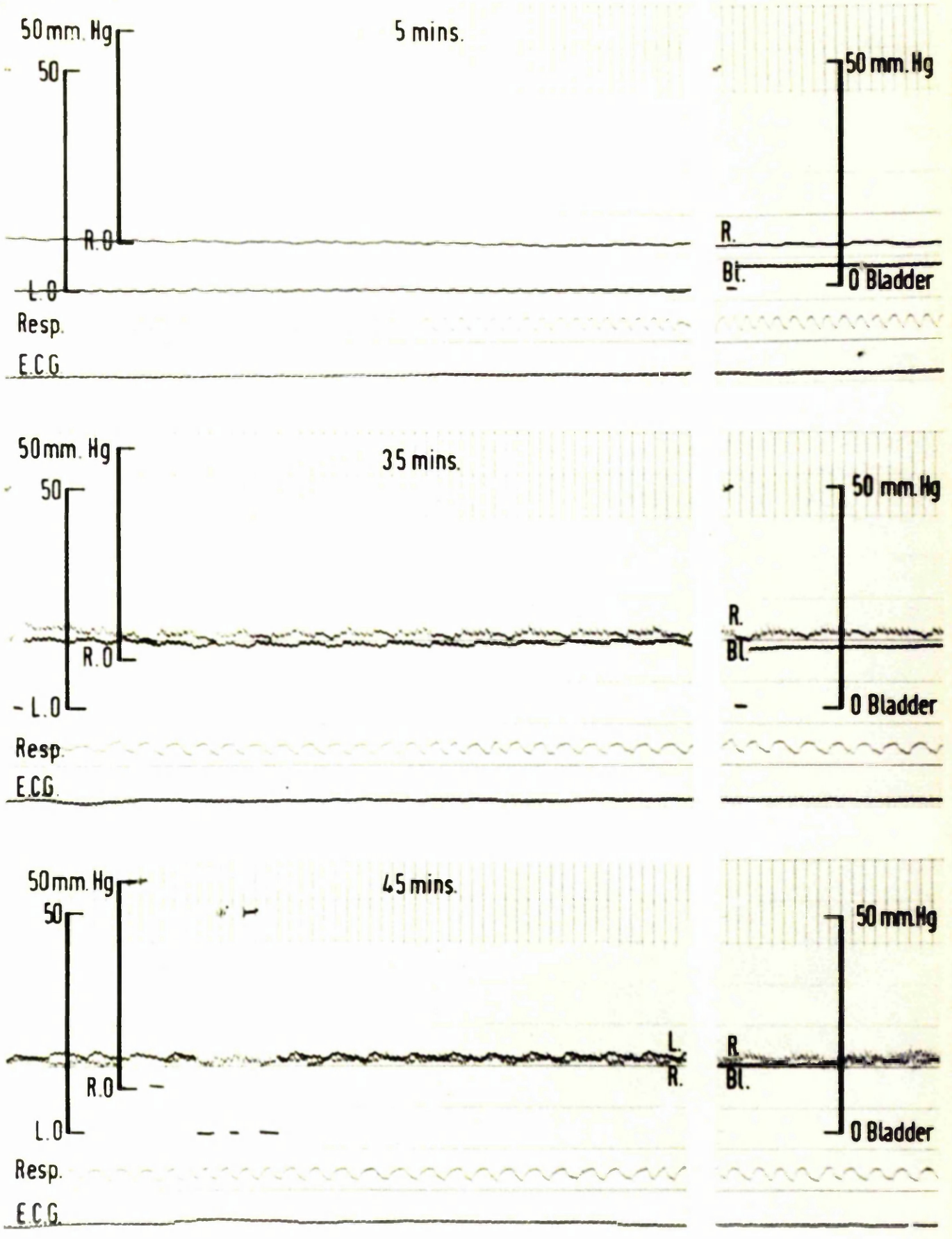

FIG. 16 THE PULSE WAVE PRODUCES A GREATER DEFLECTION IN THE PELVIC PRESSURE TRACE DURING DIURESIS.

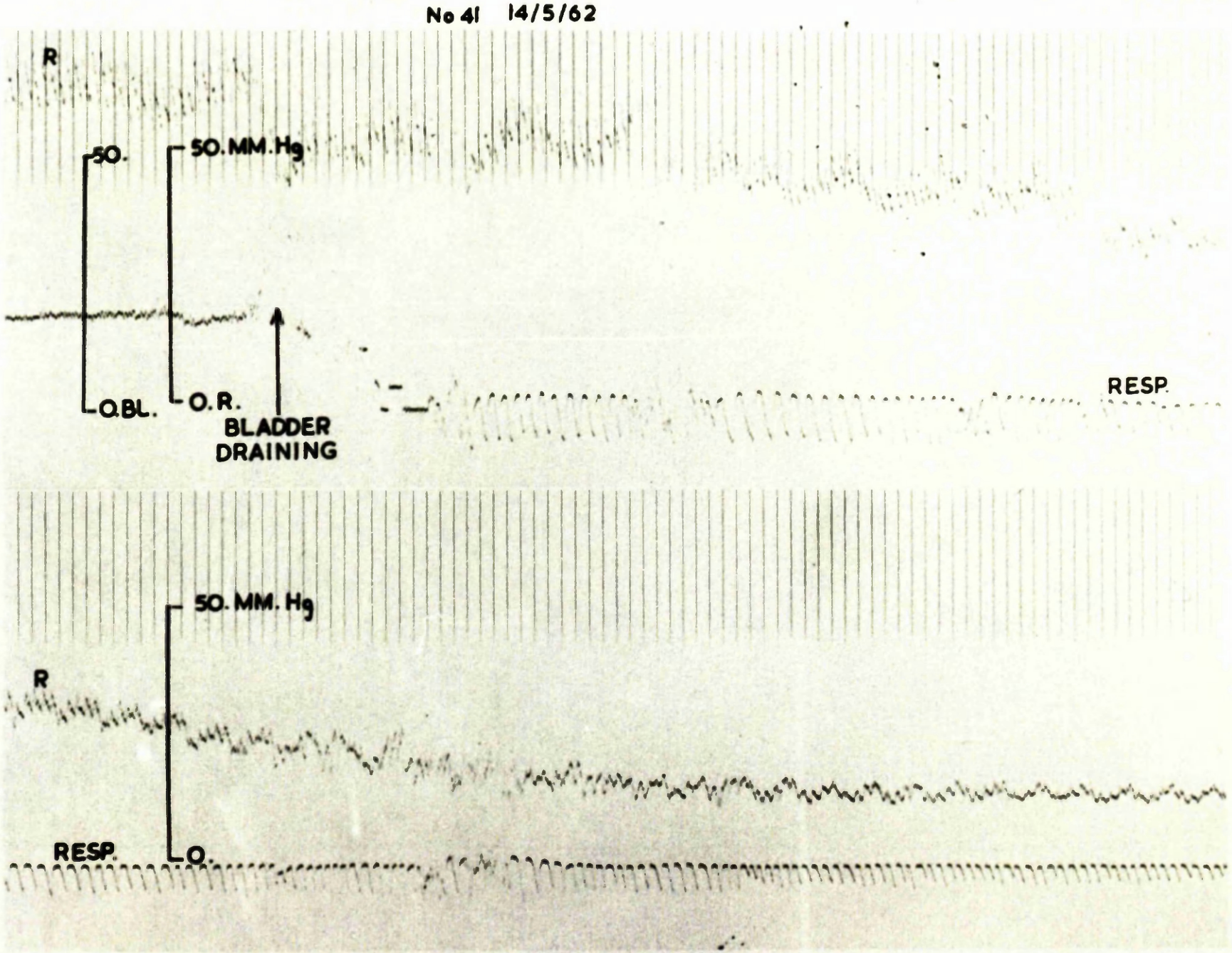

FIG. 17 ILLUSTRATES THE FALL IN PELVIC PRESSURE CAUSED BY CATHETER DRAINAGE OF THE BLADDER DURING DIURESIS.

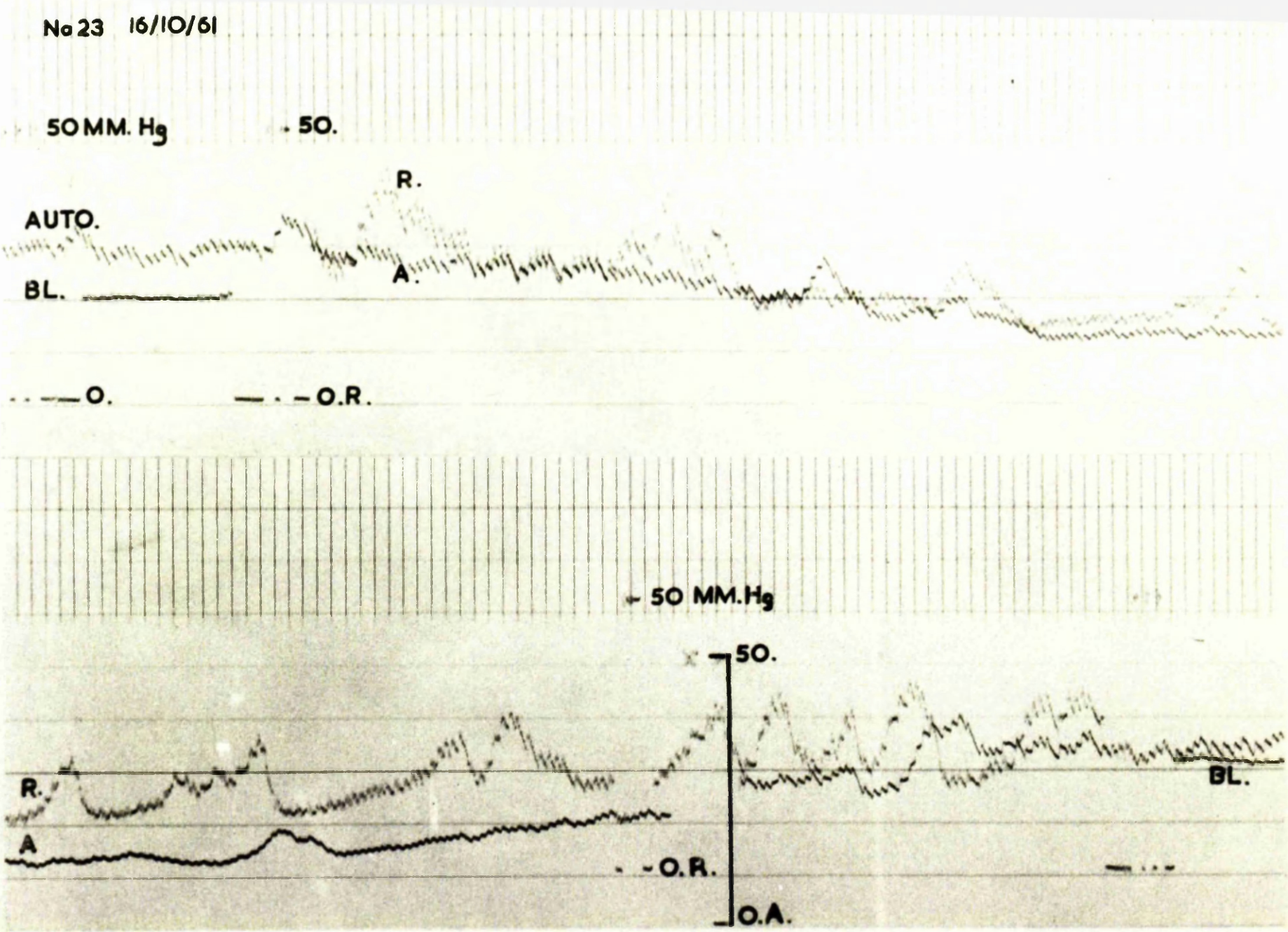

FIG. 18 PELVIC PRESSURE IN NORMAL (R) AND AUTOTRANSPLANTED (A) KIDNEYS RESTORED TO ORIGINAL LEVELS BY REFILLING BLADDER.

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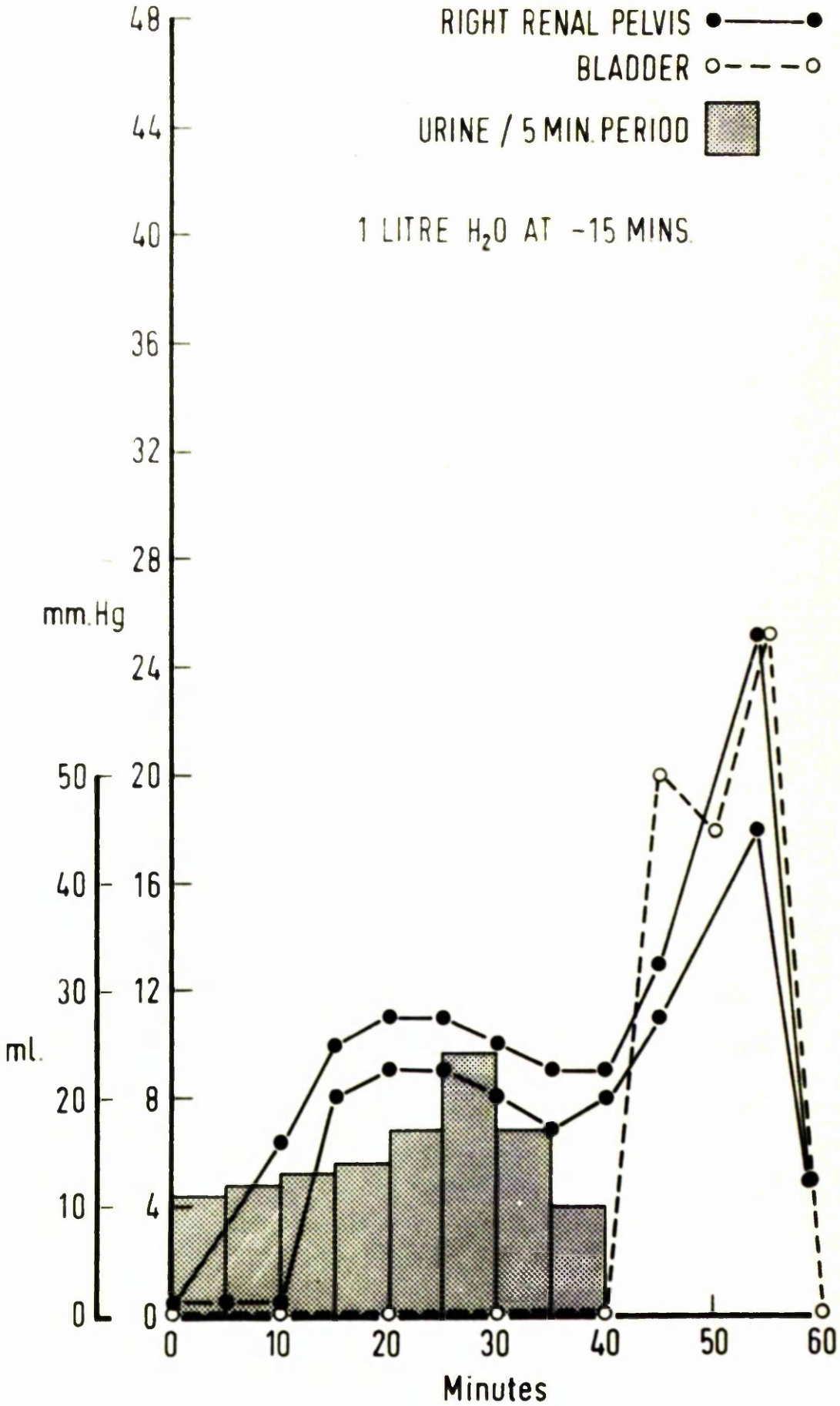

FIG. 19 PELVIC PRESSURES RELATED TO URINE FLOW RATE AND BLADDER PRESSURE DURING WATER DIURESIS.

maximum intrapelvic pressures for each animal measured during diuresis when the bladder was distended with urine are reported in Table I

II PELVIC PRESSURES IN THE AUTOTRANSPLANTED KIDNEY

The site to which the kidney was transplanted, the procedure with the contralateral kidney, and the complications which occurred, are recorded in Table II. Only eight of the 23 preparations were available for pelvic pressure studies, and the basal pelvic pressures and the maximum pelvic pressures recorded during diuresis are reported in Table III. Three preparations (Nos. 17, 22 and 43) had basal pelvic pressures within the range for a normal kidney immediately after the transplant operation, and the basal pressure trace (Fig. 20) was indistinguishable from that of a normal kidney. In these preparations there was no gross evidence of dilatation of the urinary tract on excretion pyelography (Fig. 9)

In four animals (Nos. 23, 31, 33 and 34), the basal pelvic pressure was maximally elevated during the first week after the autotransplant operation, and the basal pressure trace at this time showed regular

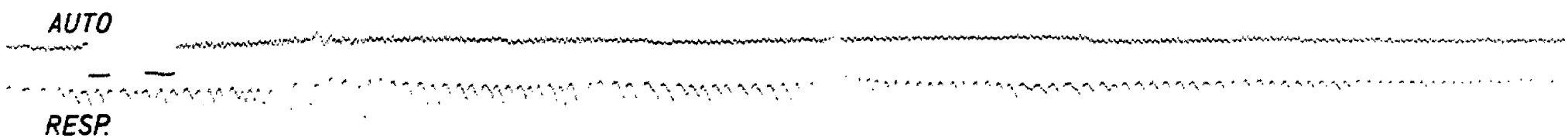

FIG. 20 TWO MINUTE RECORDING FROM PELVIS OF AUTOTRANSPLANTED KIDNEY, DOG 22, ILLUSTRATING LOW BASAL PRESSURE.

contraction complexes (Fig. 21) while the diuretic trace contained large irregular contraction waves. Descending pyelography (Fig.22) demonstrated a grossly dilated pelvis and upper ureter. The obstruction was temporary as serial pressure studies at comparable urine flow rates (Fig. 23) showed a gradual fall in pelvic pressures and also a return of the pelvic pressure response to increased bladder pressure. Within four weeks the basal pelvic pressures in Nos. 23,31 and 33 had fallen to within the normal range. Further pressure studies were not obtained in No. 34 as this animal developed an intussusception, and pelvic pressures were not measured in No. 44 during this period as the nephrostomy tube was not functioning satisfactorily.

There was no apparent difference between the pressure response in the autotransplanted pelvis and the normal renal pelvis to diuresis (Fig. 24 A & B). As the urine flow rate increased the standing pelvic pressure and the amplitude of the phasic waves also increased. Higher pelvic pressures occurred when the bladder was permitted to fill with urine or when it was distended with saline.

Only one of the animals (No. 31), which had shown

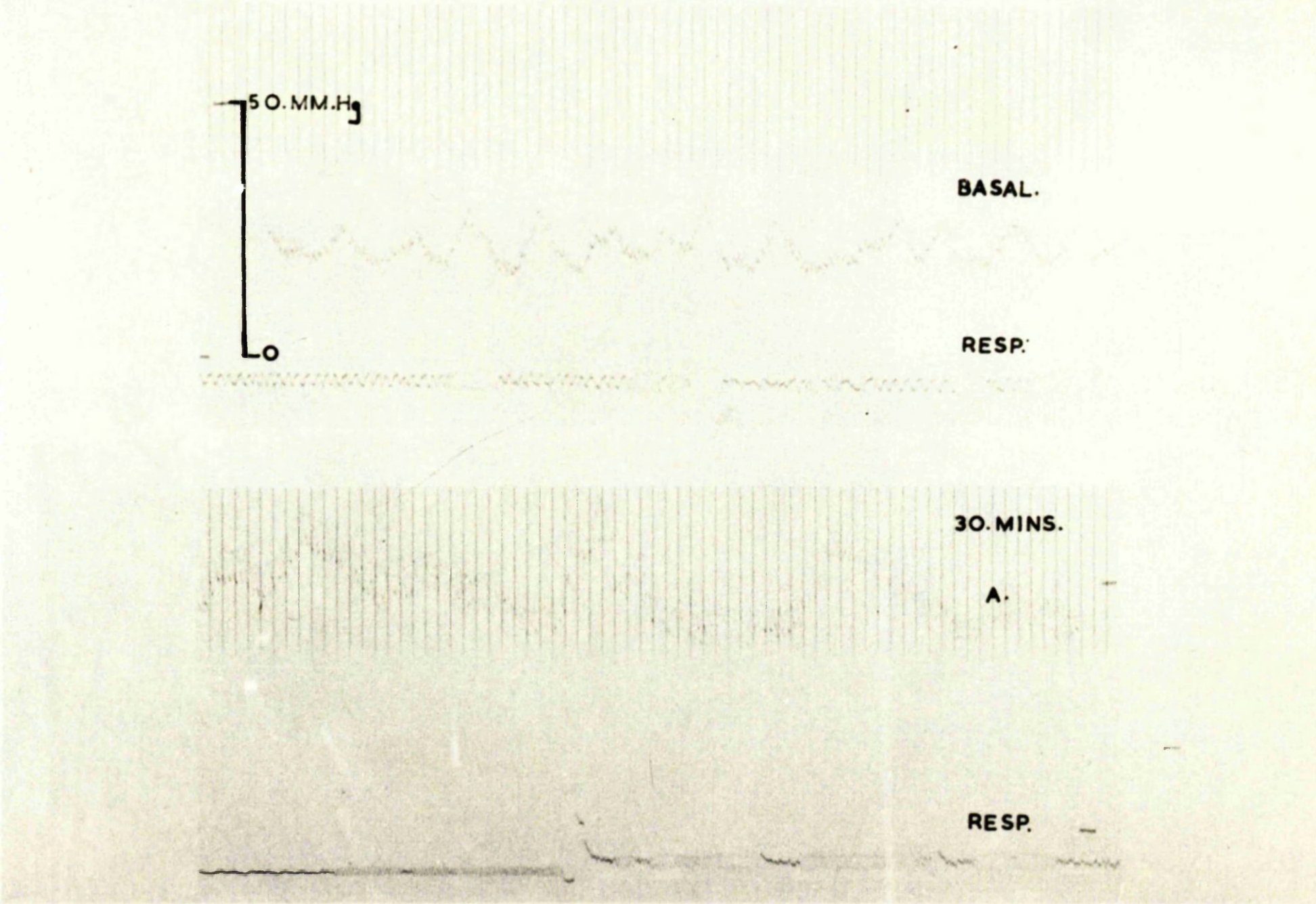

FIG. 21 OBSTRUCTION AT URETERO-VESICAL ANASTAMOSIS AFTER RENAL TRANSPLANT RESULTS IN HIGH BASAL AND DIURETIC PELVIC PRESSURES.

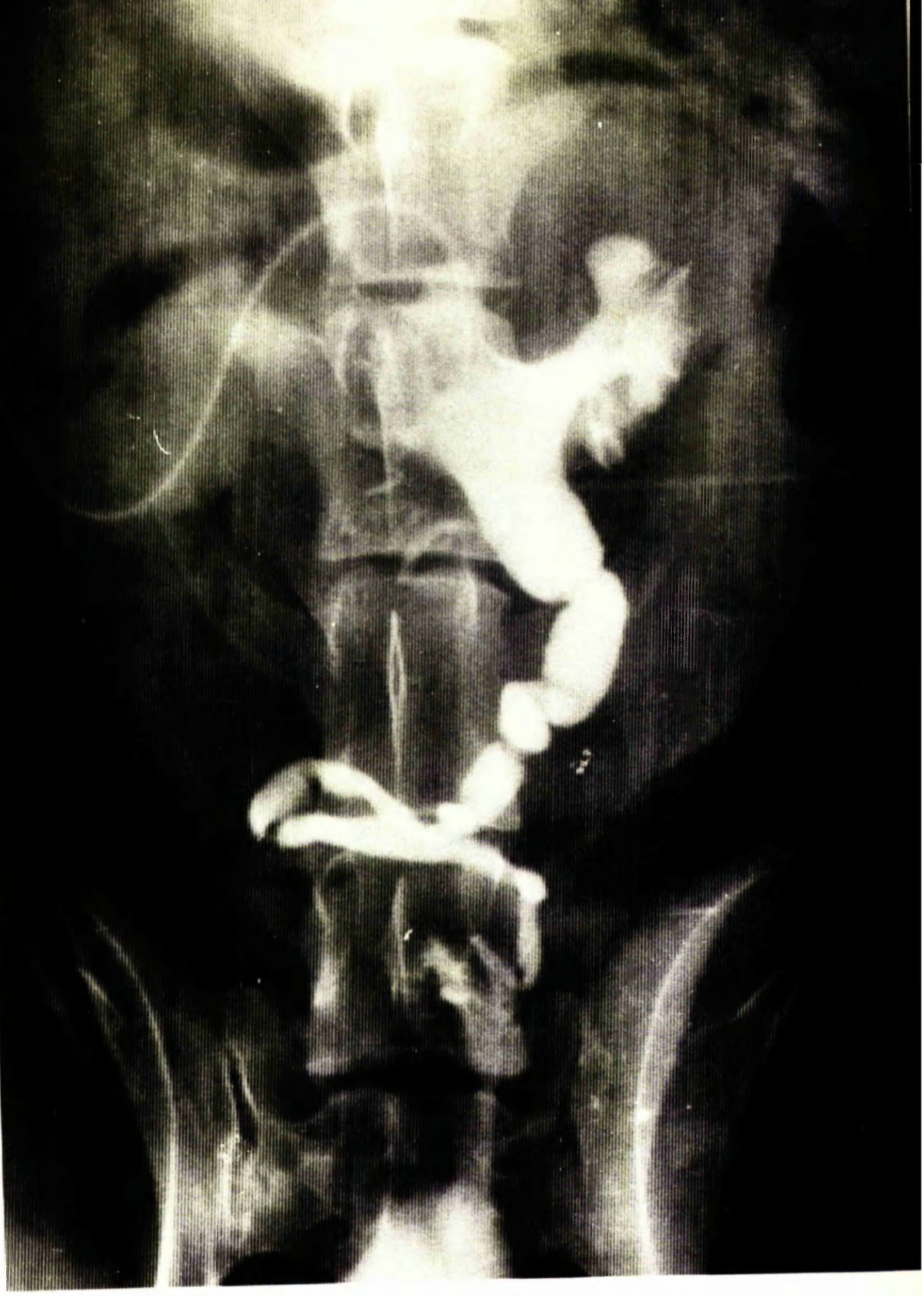

FIG.22 DESCENDING PYELOGRAM SHOWS HYDRONEPHROSIS AFTER RENAL TRANSPLANTATION.

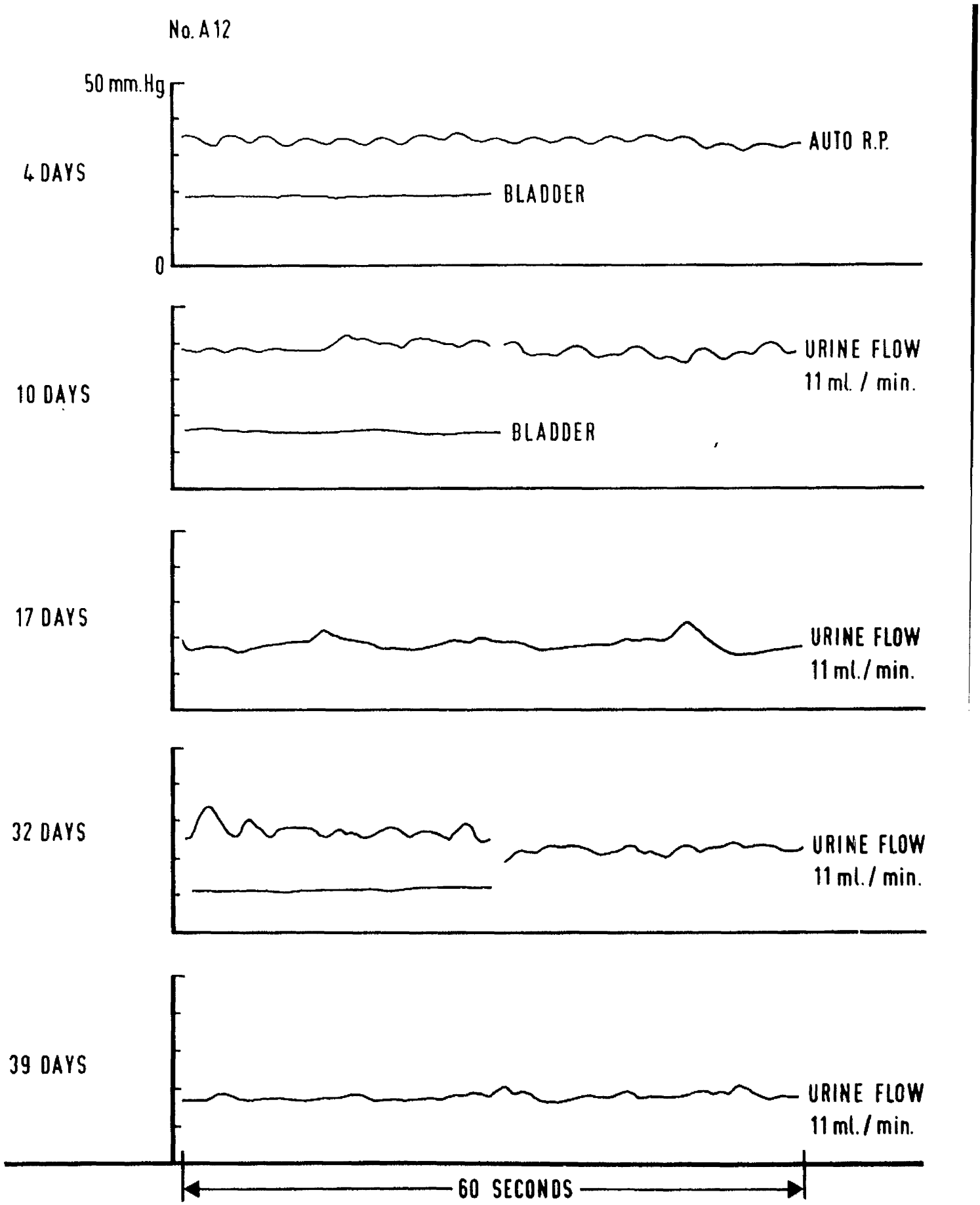

FIG. 23 TEMPORARY NATURE OF INCREASE OF PELVIC PRESSURE AFTER KIDNEY TRANSPLANT.

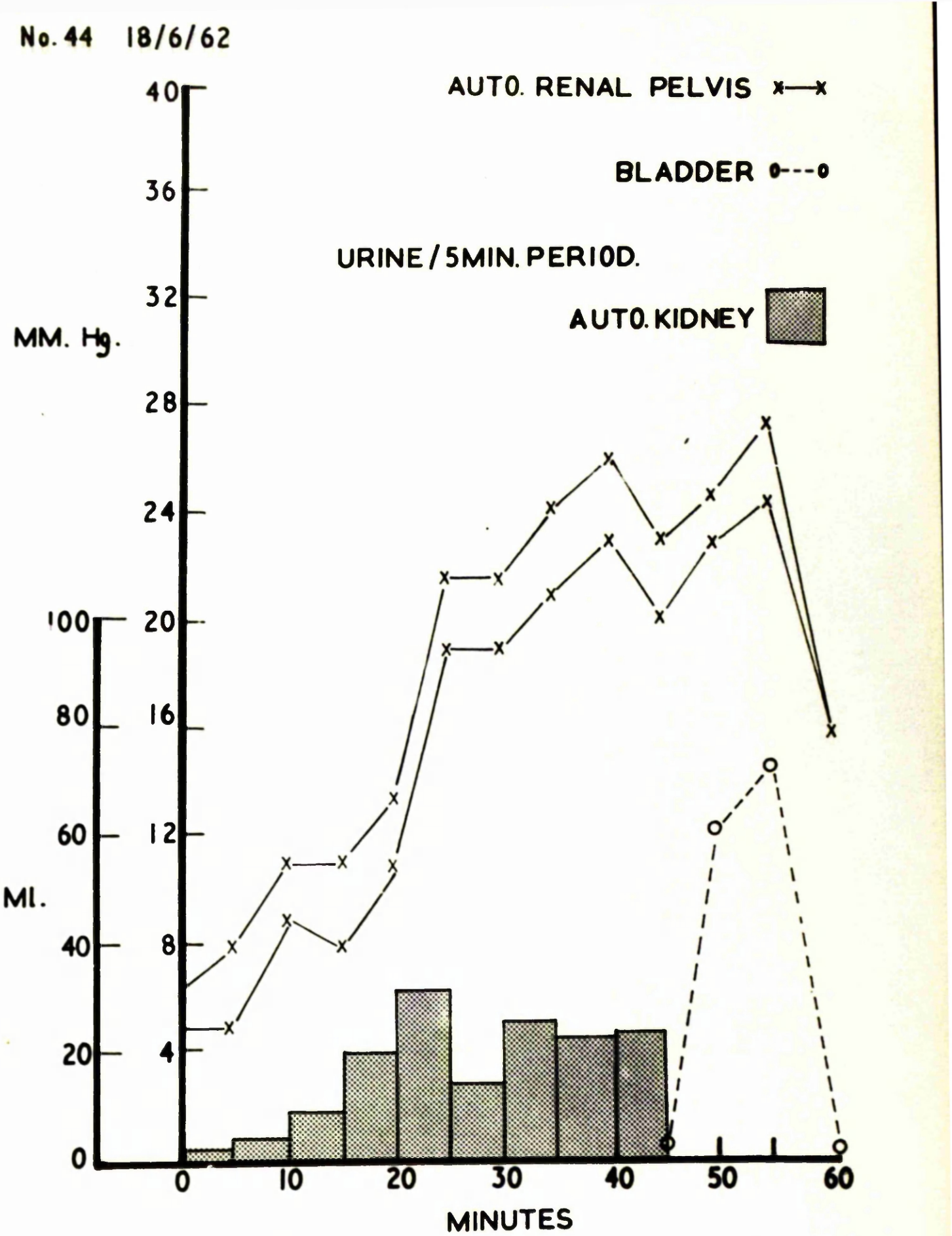

FIG. 24A PELVIC PRESSURE IN THE AUTOTRANSPLANTED KIDNEY RELATED TO URINE FLOW RATE AND BLADDER PRESSURE DURING OSMOTIC DIURESIS.

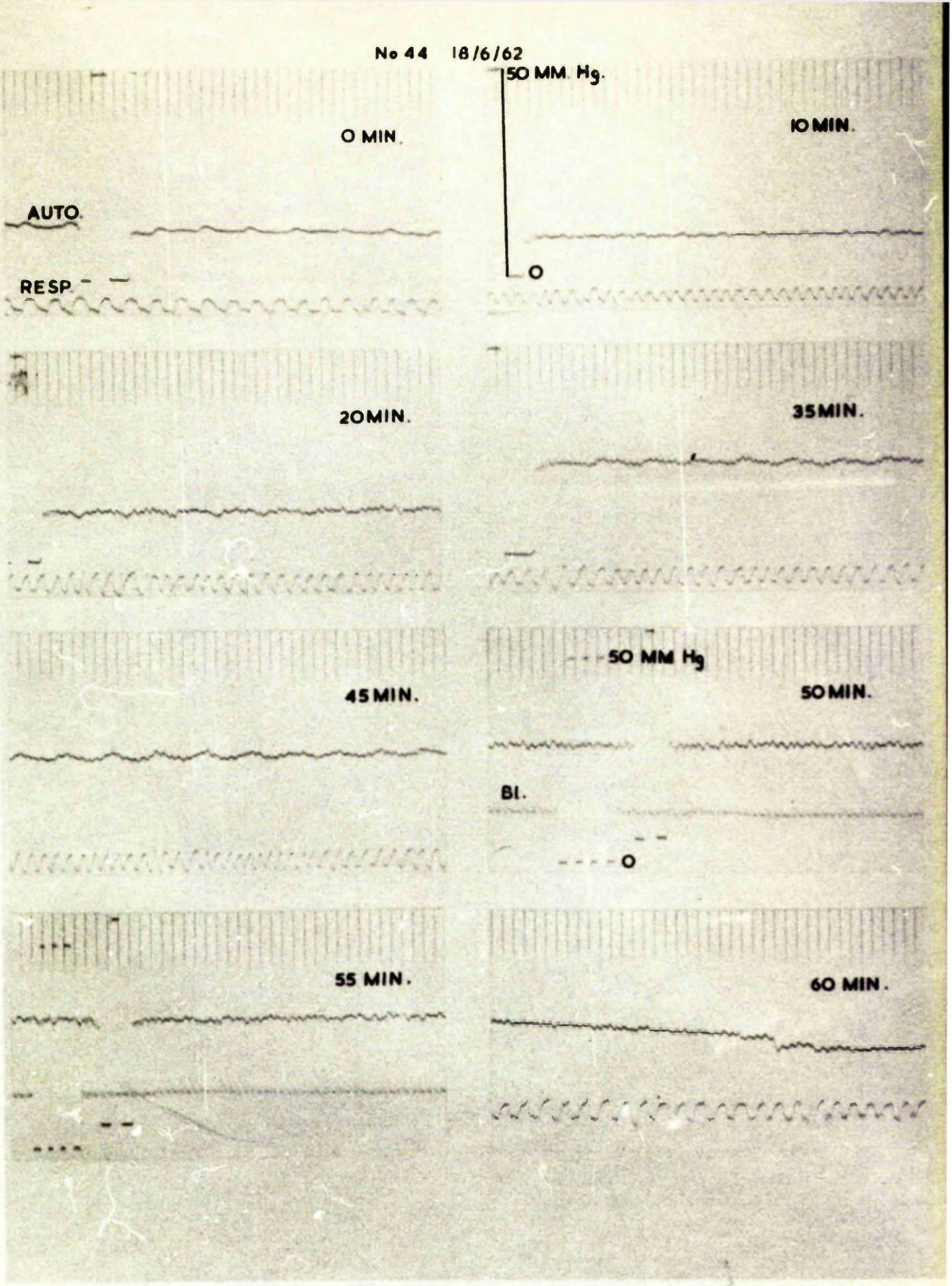

FIG. 24B RECORDINGS OF THE PELVIC PRESSURES IN THE AUTOTRANSPLANTED KIDNEY.

evidence of a temporary obstruction to urine drainage from the upper urinary tract, was observed over an extended period. Four weeks after kidney transplantation the basal pelvic pressure was 4 mm Hg. and a descending pyelogram (Fig. 25) showed no dilatation of the upper urinary tract. At 19 weeks, however, the basal pressure was moderately elevated (8-12 mm Hg.) and the descending pyelogram showed dilatation of the pelvis and ureter (Fig. 26). At 22 weeks the basal pressure was 19-31 mm.Hg. and the maximum pressure during water diuresis was 37-72 mm.Hg. at a urine flow rate of 3 mls per minute. The blood urea at this time was 28 mgm %. At autopsy the upper urinary tract contained 46 mls of urine and a pin hole stenosis was present at the uretero-vesical junction.

To obtain further information about the cause of the increase in intrapelvic pressure after renal autotransplant, uretero-vesical anastomosis was performed in two otherwise normal ureters in dogs No. 22 and 43. After this procedure the maximum increase in basal intrapelvic pressure was 12 mm.Hg. in No.22, and 9 mm.Hg. in 43. The ureter of the autotransplanted kidney in dog No.43 which had not been severed from the bladder at the time of transplant, was also re-implanted into

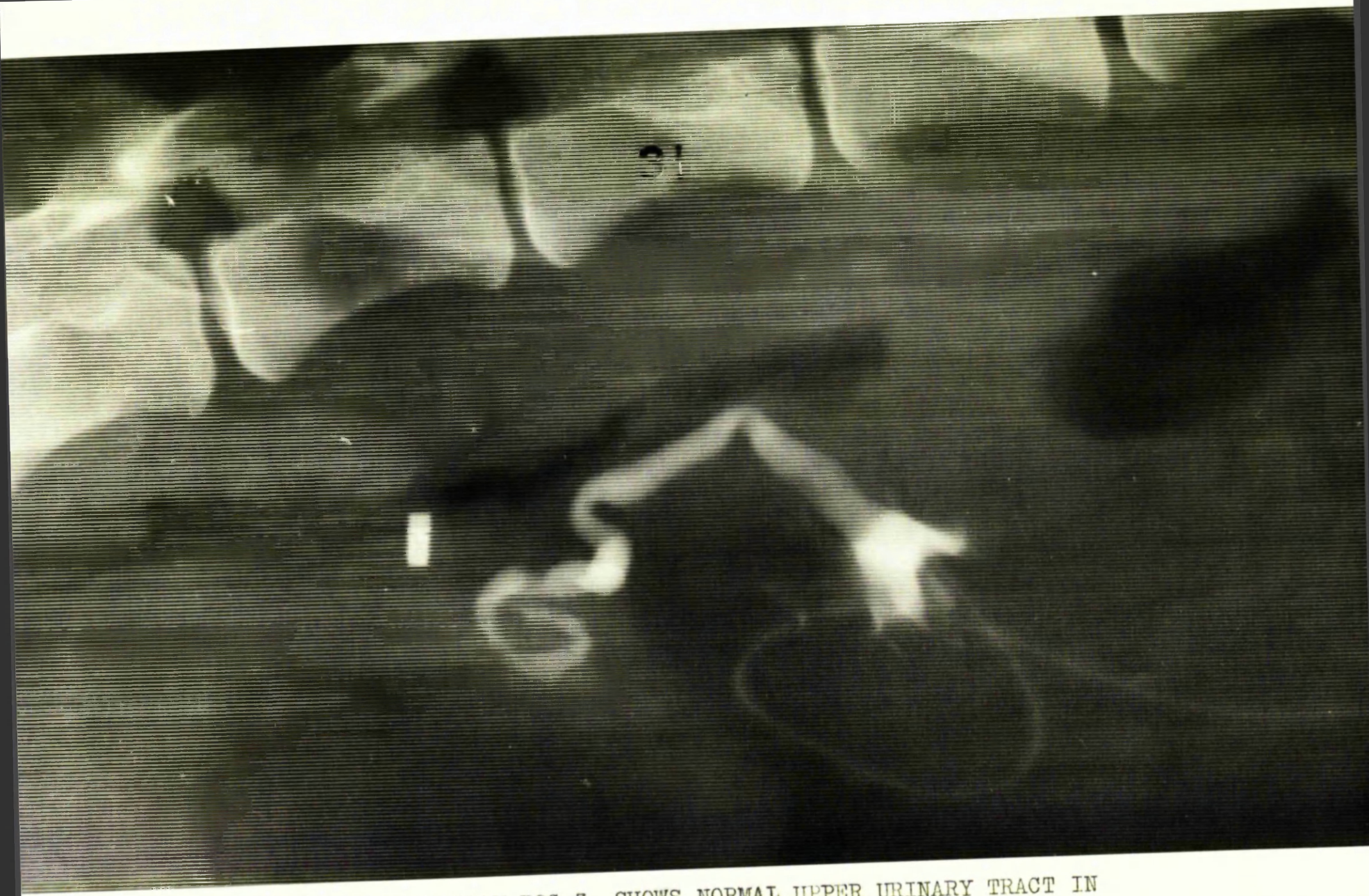

FIG. 25 DESCENDING PYELOGRAM DOG 3 SHOWS NORMAL UPPER URINARY TRACT IN AUTOTRANSPLANTED KIDNEY.

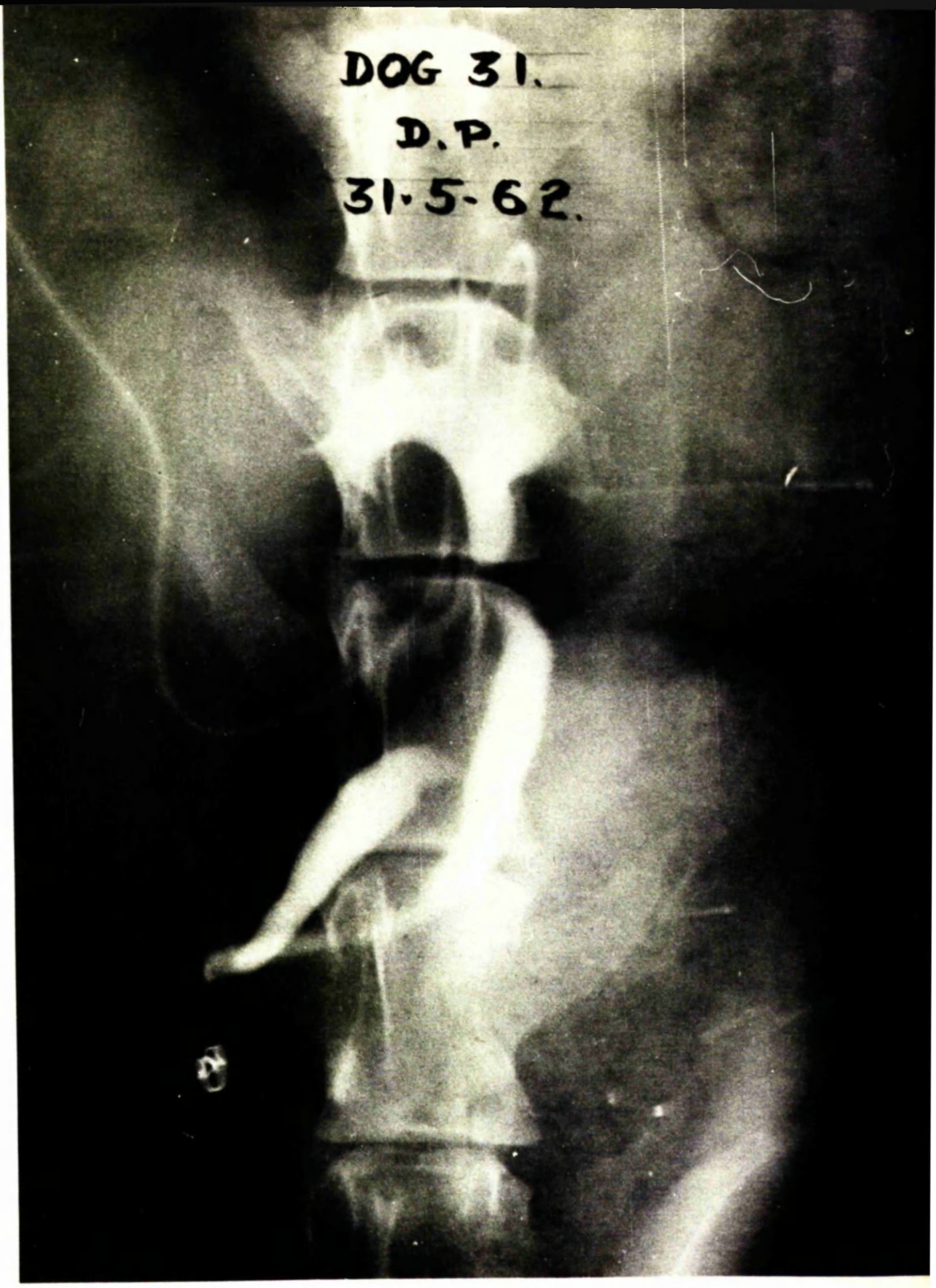

FIG. 26 DESCENDING PYELOGRAM. DOG 31, SHOWS HYDRONEPHROSIS. BASAL PELVIC PRESSURE 8-12 mm.Hg

the bladder and a maximum increase of basal intrapelvic pressure of 5 mm.Hg. was recorded. In these preparations the excretion urograms showed no evidence of upper urinary tract dilatation.

The reasons for the small proportion of satisfactory renal transplant preparations are of incidental interest.

Arterial thrombosis was the major technical complication and accounted for almost half of the failures. Intussusception of the bowel occurred in five animals on the second to the seventh day after operation. The symptoms of intussusception were often minimal and consisted only of an occasional small vomit. The condition was recognised and successfully reduced by operation on two occasions but recurred in one dog and required resection. What appeared to be a classical picture of intussusception was exhibited by No. 30; colic, vomiting and the passage of blood and mucus per rectum, but at laparotomy the only abnormality was a spastic large bowel. Unfortunately the dog died under the anaesthetic. Gastric dilatation, renal artery spasm, and renal vein thrombosis, accounted for the remaining failures. Perihilar abscess developed as a late complication in No.33 and the experiment was terminated.

III

a) ACUTE URETERIC OBSTRUCTION: The general pattern of the pressure changes recorded from the lower end of the ureter and the renal pelvis in the acutely obstructed upper urinary tract, was the same in seven experiments, although the rate and degree of pressure changes varied. With obstruction, the ureteric contraction complexes immediately increased in amplitude until the wave peaks exceeded a pressure of 50 mm.Hg. (Fig. 27). The standing pressure in the ureter which was of the order of zero to a few mm.Hg., suddenly increased and thereafter continued to climb gradually, and as this happened there was an inverse fall in the height of the ureteric waves.

At first the pressure in the renal pelvis remained lower than the ureteric standing pressure, but as the obstruction continued, the pelvic pressure also increased until a common pressure and wave form was recorded from the ureter and the pelvis. At this stage there was a continuous column of fluid in the ureter and pelvis.

b) URETERO-COLIC ANASTAMOSIS: The Coffey technique of uretero-colic anastamosis was carried out in eight dogs which had previously had nephrostomy tubes

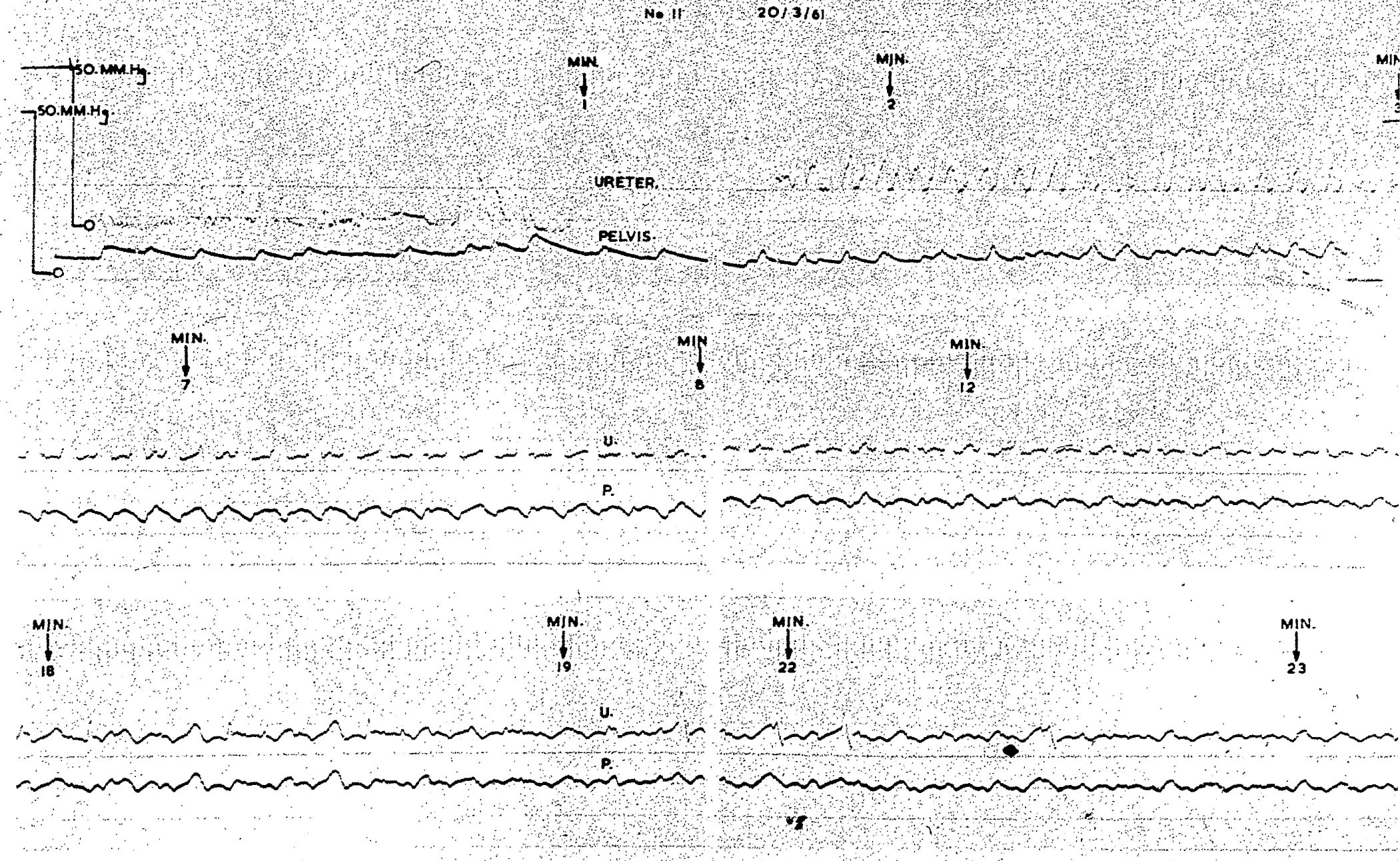

FIG. 27 ILLUSTRATES CHANGES IN PELVIC AND URETERIC PRESSURES FOLLOWING ACUTE URETERIC OBSTRUCTION.

implanted in the corresponding kidneys. In all instances a degree of subacute obstruction to urine drainage was produced. The intrapelvic pressures recorded one week after anastamosis at low urine flow rates (resting pressure) and the maximum pressures recording during diuresis are reported in Table V. It was found that osmotic diuresis produced a further increase in the already raised standing pelvic pressures and in the amplitude of the phasic contraction waves (Fig. 28 A & B). Pyelography demonstrated a continuous column of fluid in the upper urinary tract, but the degree of dilatation could not be correlated with the pressure recorded from the renal pelvis.

Pyonephrosis occurred in five dogs (Nos. 5, 7, 11, 14 and 15) after manometry or pyelography, and the experiments were terminated. The remaining dogs (Nos. 1, 3 and 20) were studied for 14 weeks. It was found that, as the duration of the partial obstruction progressed, there was a gradual fall in resting pelvic pressures and in the maximal response to diuresis, (Fig.29). Ultimately the resting pressure lay within the basal range of the normal pelvis, and the response of the standing pelvic pressure to diuresis and the amplitude of the contraction complexes were markedly

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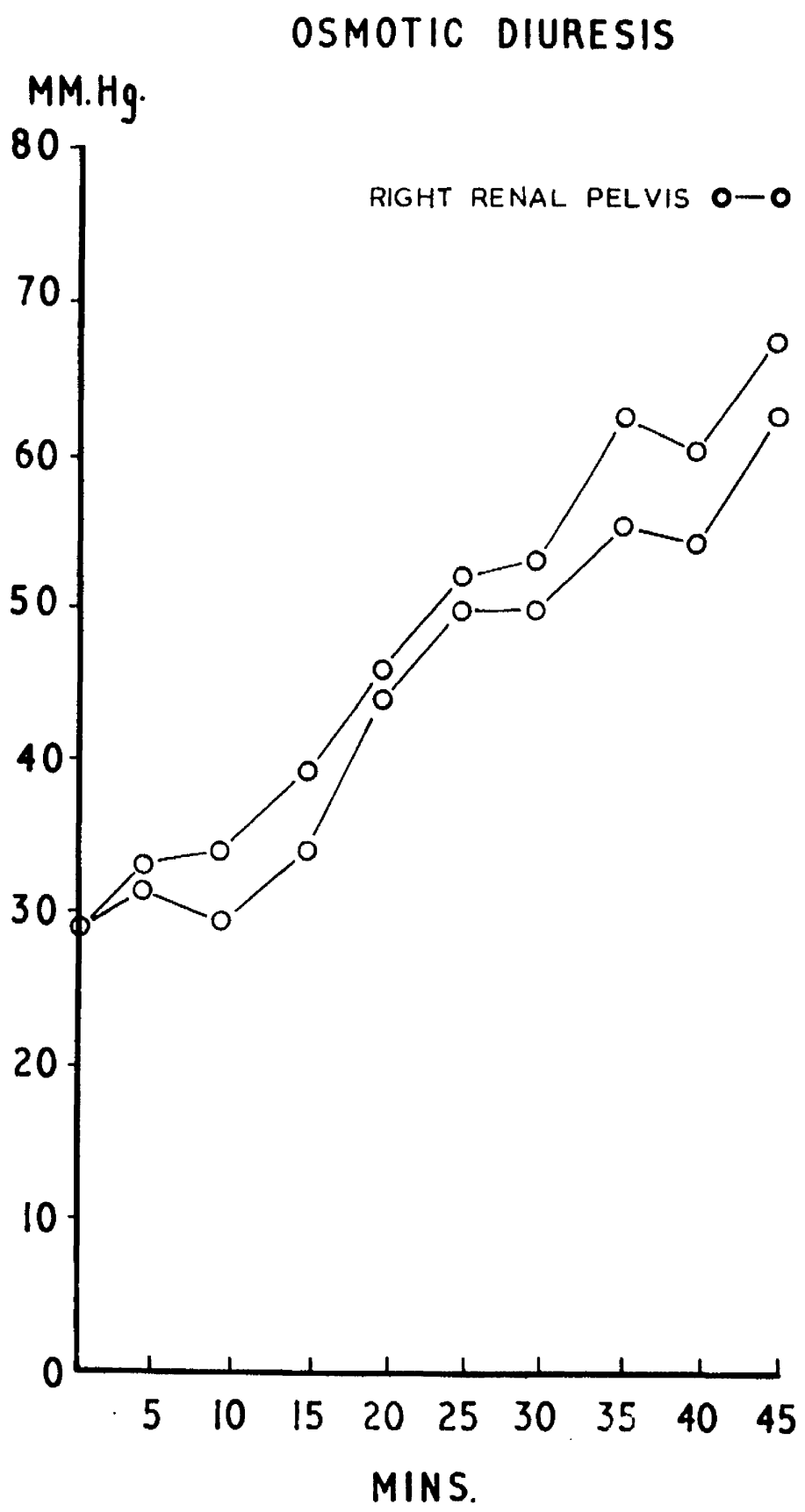

FIG. 28A HIGH RESTING PELVIC PRESSURE AFTER URETERO-COLIC ANASTAMOSIS FURTHER INCREASED BY OSMOTIC DIURESIS.

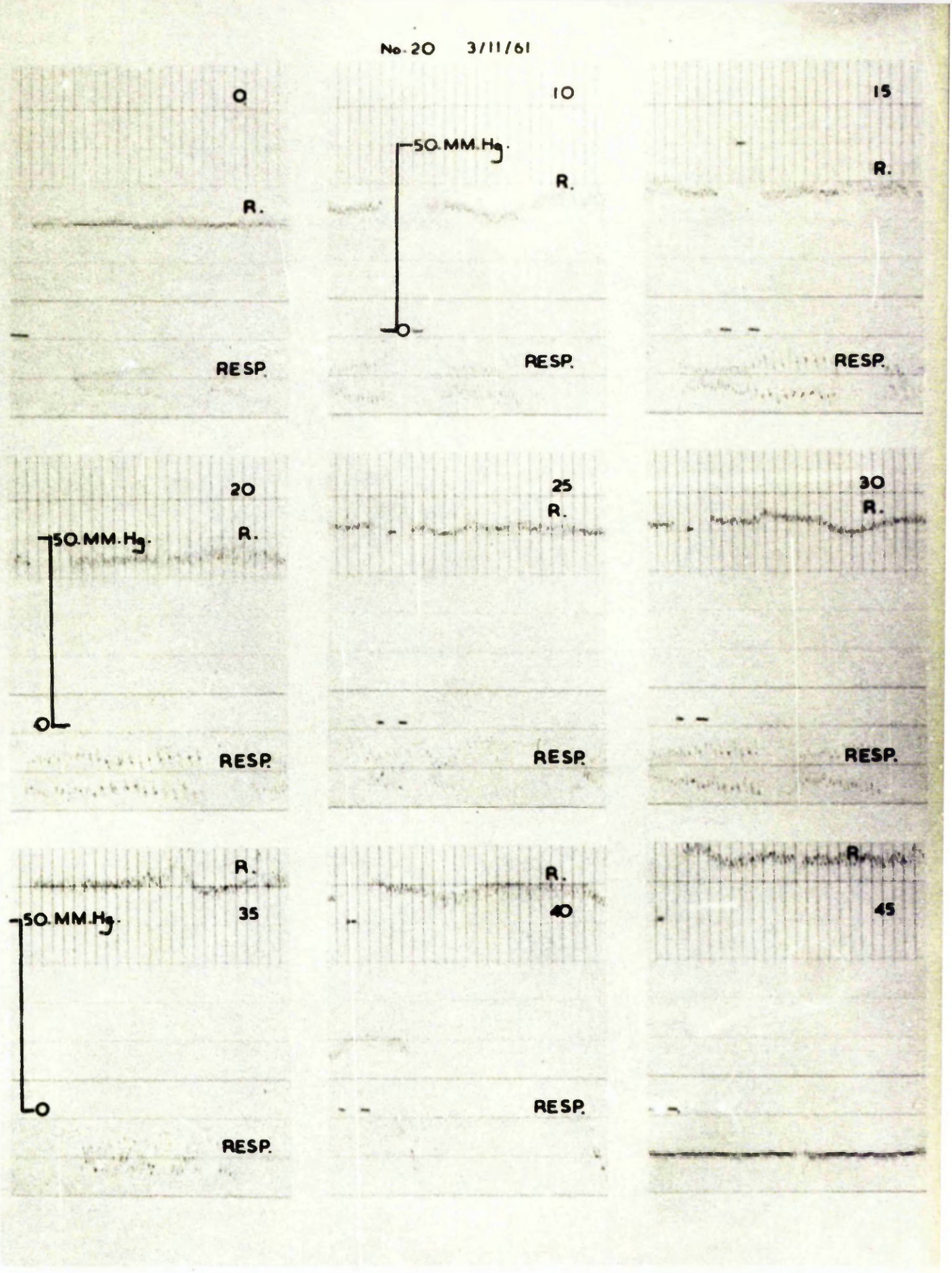

FIG. 28B RECORDINGS OF THE PRESSURES IN THE RENAL PELVIS

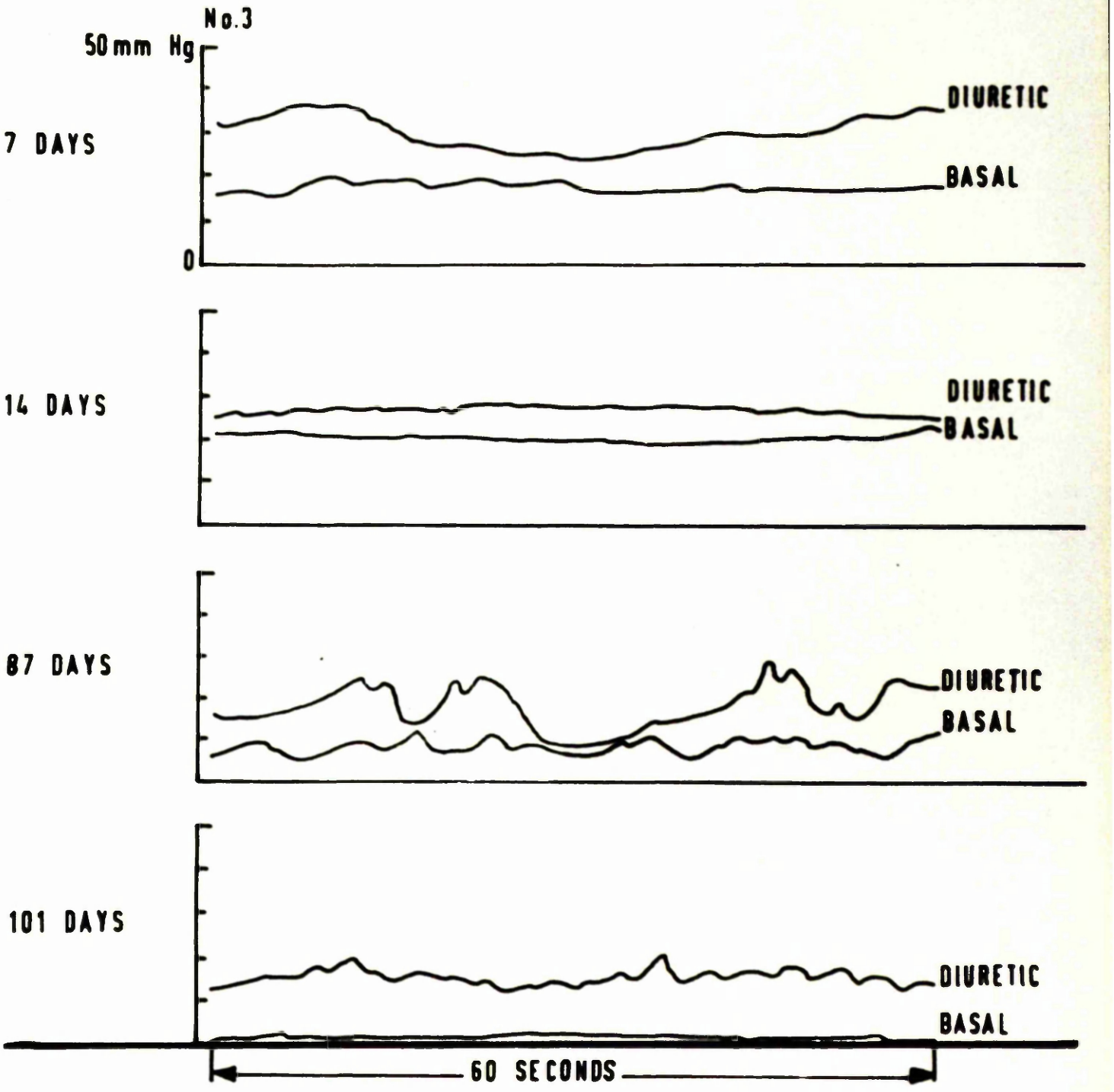

FIG. 29 ILLUSTRATES THE GRADUAL FALL IN BASAL AND MAXIMAL DIURETIC PRESSURES IN THE RENAL PELVIS AFTER URETERO-COLIC ANASTAMOSIS.

impaired, Table IV. The radiograph of each injected post-mortem specimen showed a marked stenosis at the uretero-colic anastamosis and a varying amount of atrophy of the renal parenchyma, (Fig.30).

IV ADDITIONAL STUDIES

a) Pelvic pressure response to direct infusion: As there were two tubes in the renal pelvis, the intrapelvic pressure could be recorded throughout the infusion. It was found that within a few seconds of even an extremely rapid infusion, intrapelvic pressures returned to preinfusion levels (Fig.31). The type of infusate used, urine, saline, or Hypaque saline, did not affect the pelvic pressure response to infusion. The duration of infusion appeared to be important (Fig. 32), as, with the same infusion rate at the same initial pelvic and bladder pressures, a short period of infusion resulted in a slight rise in intrapelvic pressure, whereas a more prolonged infusion, while causing a similar increase in the standing pelvic pressure, also gave rise to a series of phasic contraction waves. There was little apparent quantitative pelvic pressure response to different rates of infusion, (Fig. 33) Tr. I and Tr. 2. If, however, the pelvis was infused at a fixed rate during an osmotic

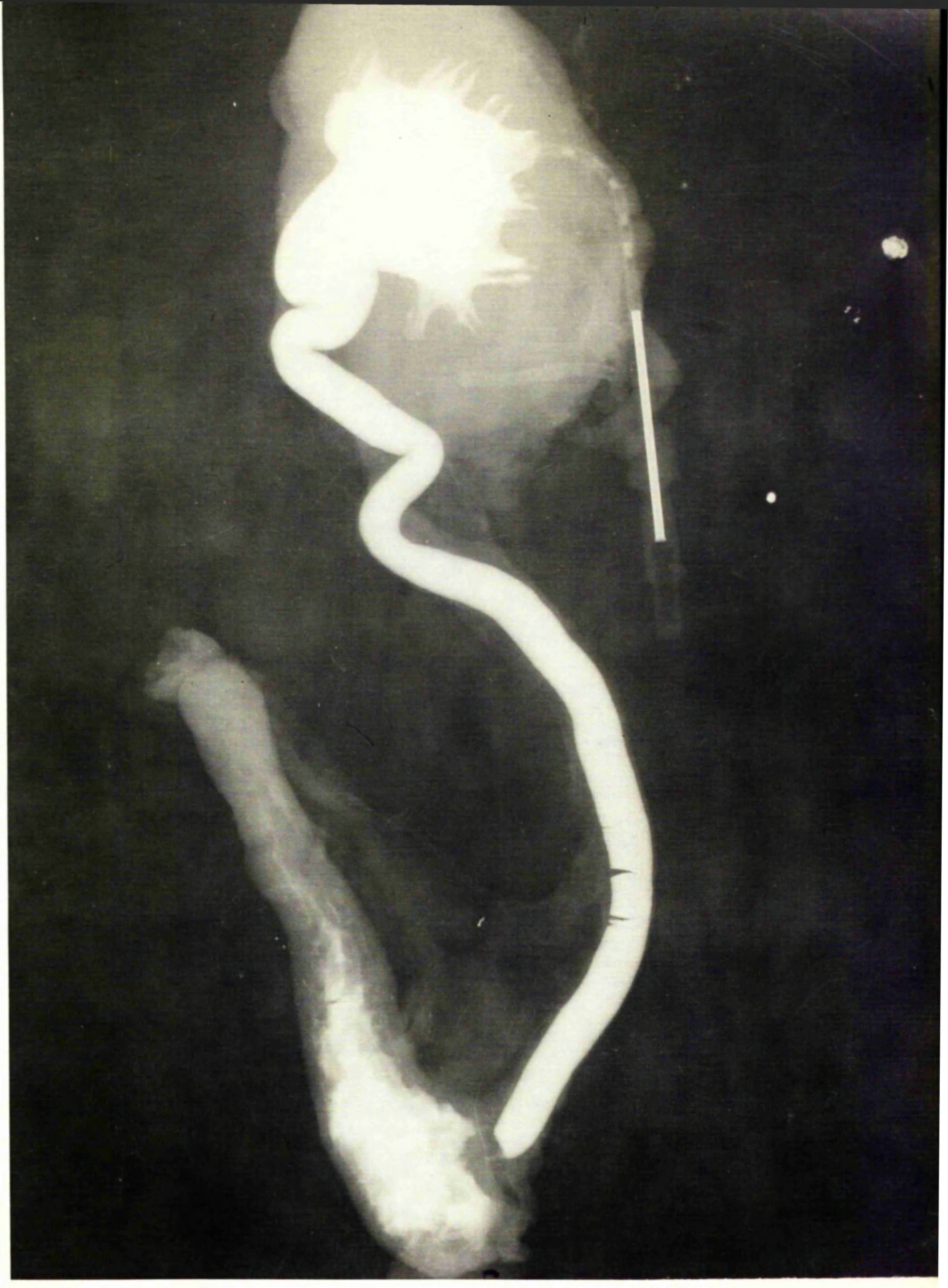

FIG. 30 RADIOGRAPH OF INJECTED POST-MORTEM SPECIMEN SHOWING STENOSIS AT THE URETERO-COLIC ANASTAMOSIS

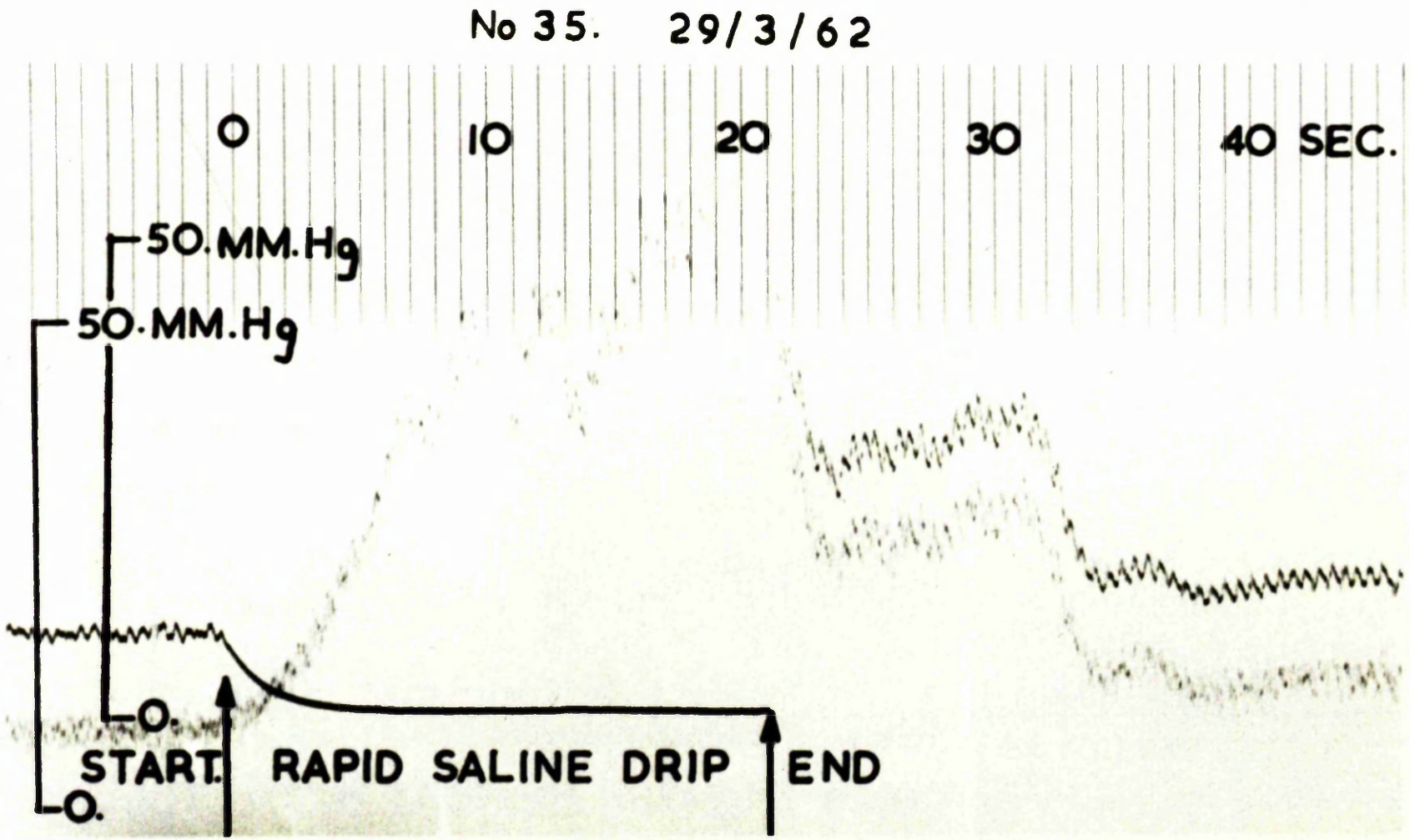

FIG. 31 ILLUSTRATES RAPID FALL IN PELVIC PRESSURE AT END OF SALINE INFUSION.

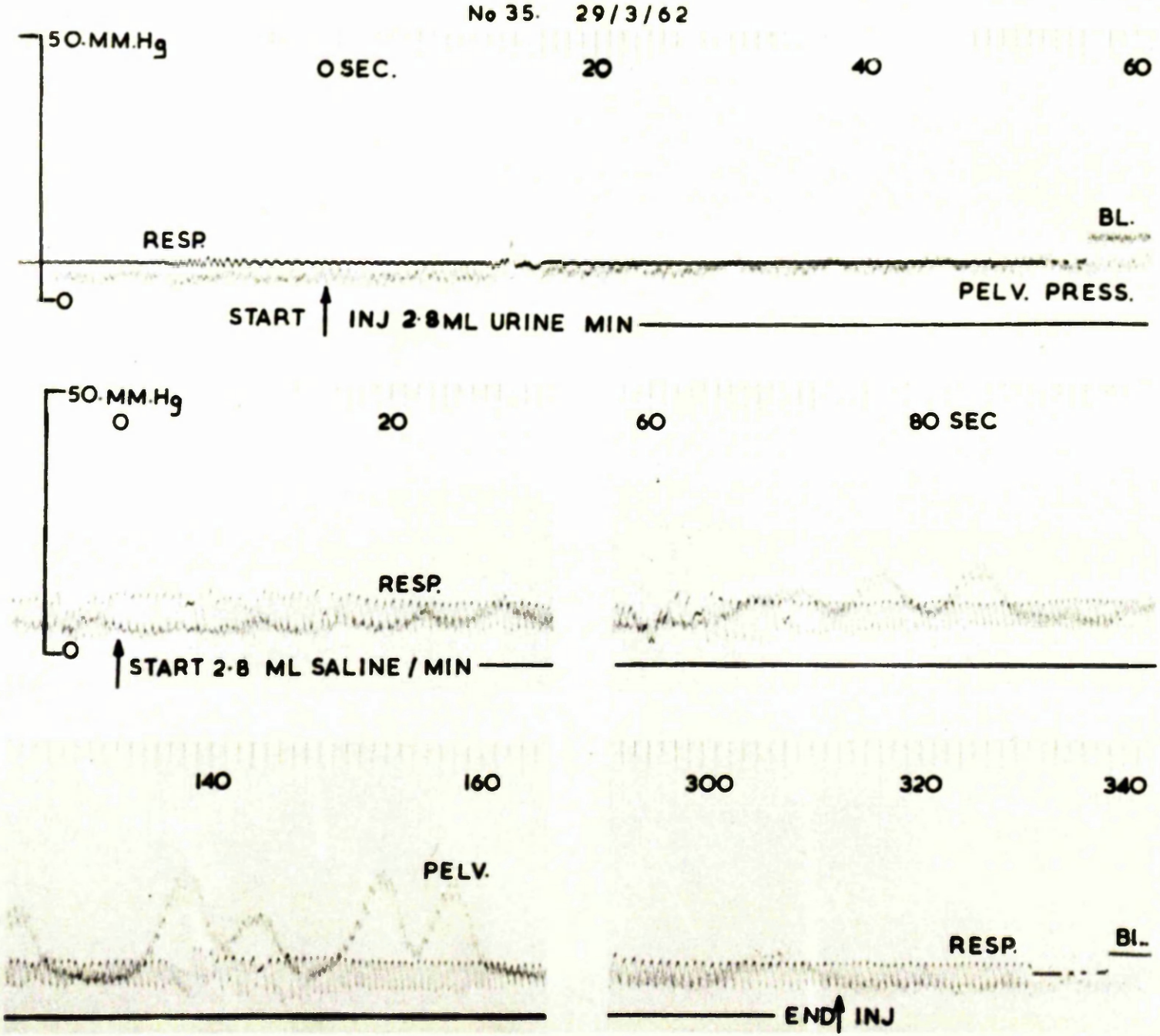

FIG 32 HIGH PELVIC CONTRACTION WAVES PRODUCED BY PROLONGED INFUSION.

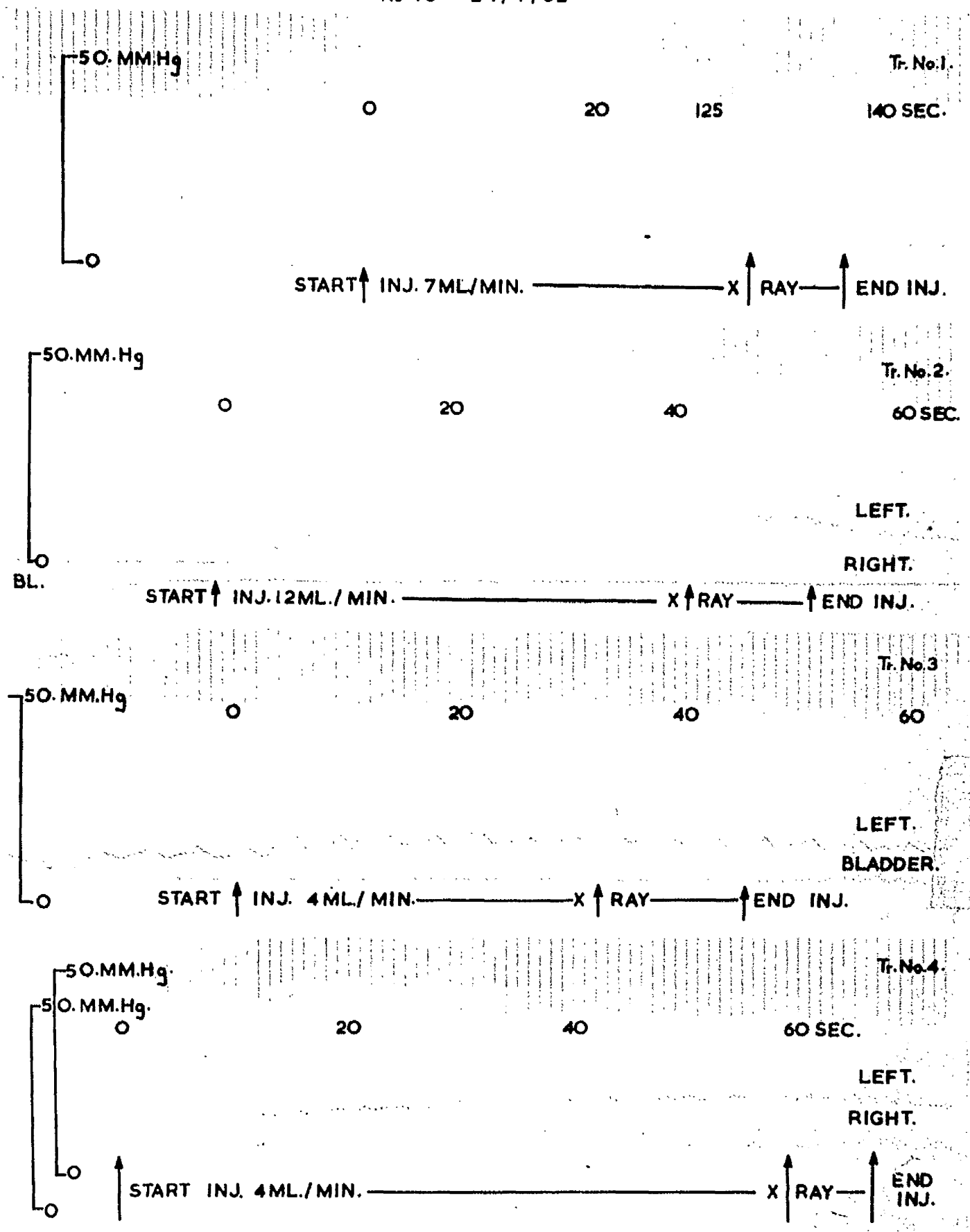

FIG. 33

DIRECT INFUSION OF LEFT RENAL PELVIS AT 7 ml/MIN (Tr.1) AND 12 ml/MIN (Tr.2) RAISE PRESSURE 8 AND 10 mm.Hg. RESPECTIVELY. INFUSION AT 4 ml/MIN (Tr.3) AT BEGINNING OF STANDARD DIURESIS PRODUCES 4 mm.Hg. INCREASE IN PRESSURE WHEREAS THE SAME RATE AT THE MID POINT OF STANDARD DIURESIS PRODUCES 8 mm.Hg. INCREASE.

diuresis, the increase in pelvic pressure produced by the infusion was very much less at the low urine flow rates occurring early in diuresis (Fig. 33, Tr.3) than at the higher urine flows later in diuresis (Fig.33 Tr. 4).

b) Pelvic pressures and pelvic movements: No gross variation in pelvic size or shape was noted in the pyelograms made at the different pelvic pressures during the above infusion experiments (Fig. 34). Under the X-ray image intensifier pyelo-ureteral movements appeared similar when contrast medium was being excreted by the kidney or when it was being infused through the second nephrostomy tube. Infusion of Hypaque was used to provide the necessary contrast for the cineradiographic studies made during diuresis. Repeated examination of the cinefilm records failed to reveal any marked alteration in the size or shape of the intrarenal pelvis at different pelvic pressures, flow rates, and bladder pressures.

The following interpretation of pelvic emptying was made from the cineradiograph. The infused contrast medium streamed from the intrarenal pelvis filling in continuity the extrarenal pyelo-ureteral segment and as this segment changed from a conical to a globular shape,

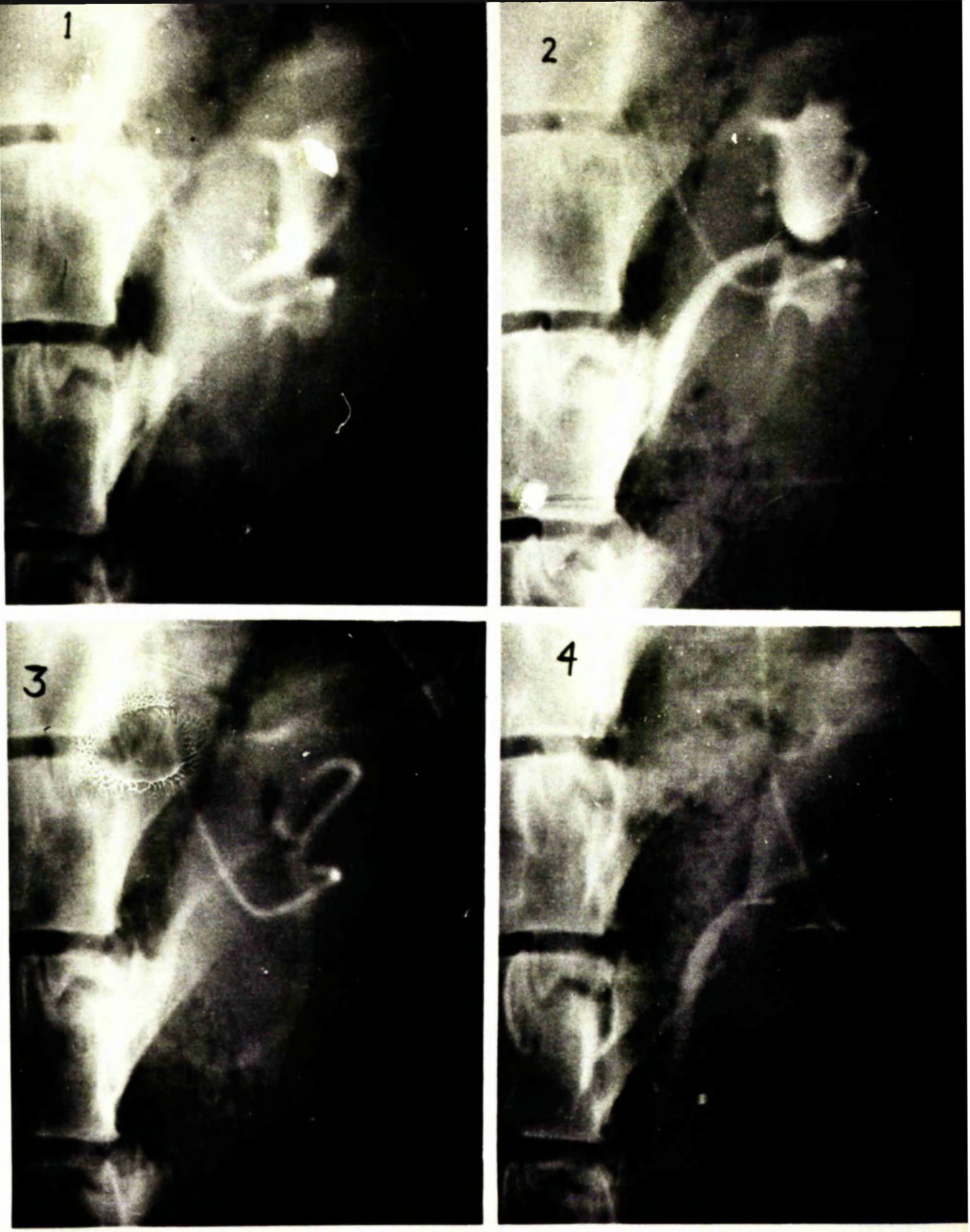

FIG. 34 PYELOGRAMS MADE IN DOG 45 DURING THE INFUSION EXPERIMENTS ILLUSTRATED IN FIG. 33. LESS CONTRAST OBTAINED DURING DIURESIS BUT NO SIGNIFICANT ALTERATION IN PELVIC CONTOUR.

a constriction occurred separating the intra and extrarenal parts of the pelvis. The pyelo-ureteral bolus of contrast medium then became spindle shaped and passed down the ureter, and simultaneously contrast medium from the intrarenal pelvis started to reform the extrarenal pyelo-ureteral segment. This sequence of movements could not be correlated with the pressures measured from the intrarenal pelvis.

c) Pressures in the pelvi-ureteral zone: the pressure traces from the left nephrostomy tube in dog 5 at low urine flow rates featured phasic waves of the form associated with ureteric contraction complexes (Fig.35). At autopsy the tip of the left nephrostomy tube was found to lie in the extrarenal pelvis whereas the tip of the right tube lay in the intrarenal pelvis.

In No.35, the tip of the PTFE tube was positioned in the pelvi-ureteral region of the solitary right kidney and the tip of the second tube in the pelvis, (Fig.36).

The trace from the pelvi-ureteral region at low urine flow rates was at times similar to the pelvic trace Fig. 37A, whereas at other periods during basal flow, contractions similar to ureteric waves were present Fig. 37B. As the flow of urine increased during

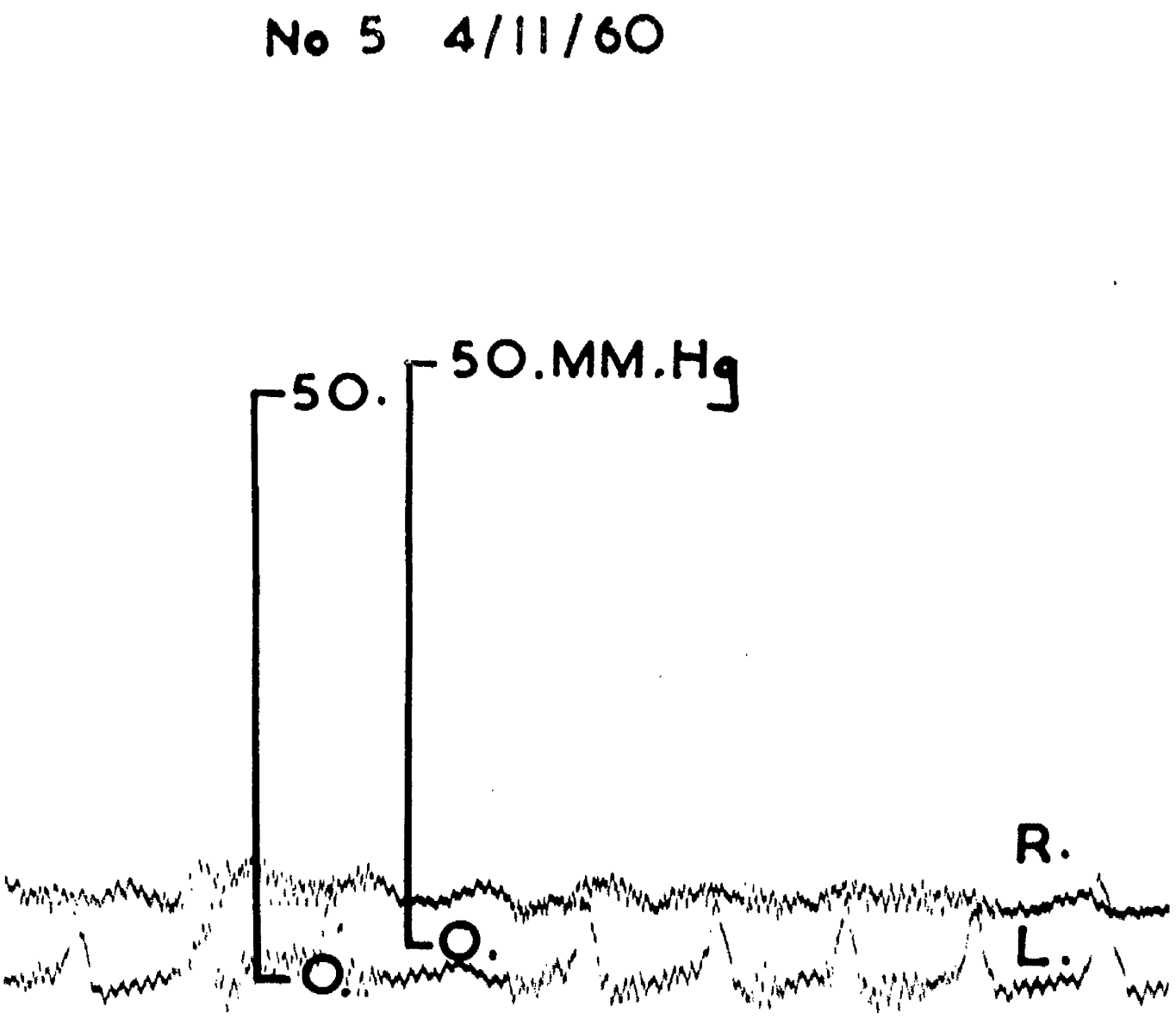

FIG. 35 "URETERIC" WAVES RECORDED FROM LEFT MANOMETRIC TUBE IN EXTRARENAL PELVIS.

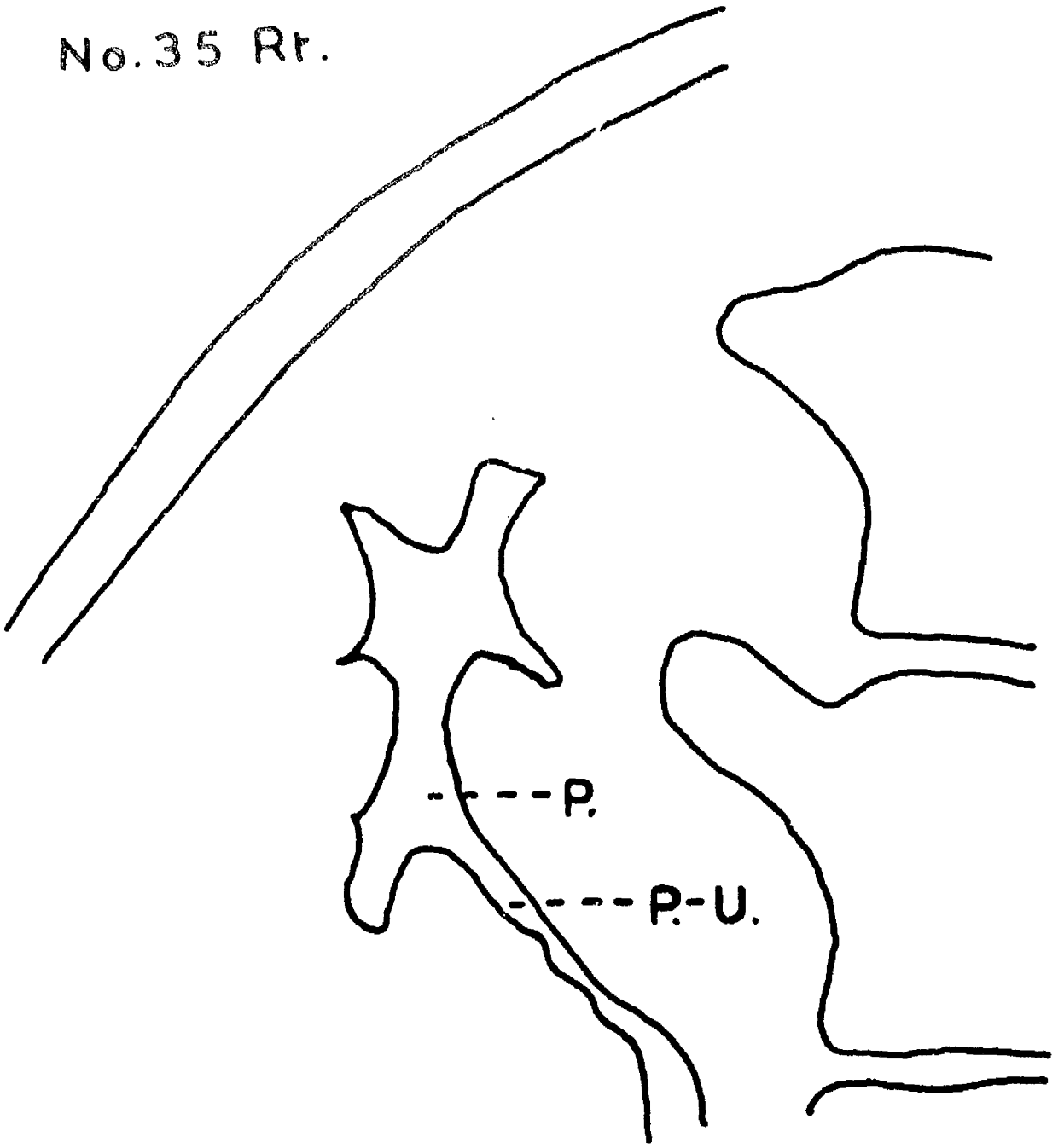

FIG. 36 TRACING OF PYELOGRAM INDICATING POSITIONS OF TIPS OF MANOMETRIC TUBES IN PELVIS (P) AND PELVI-URETERAL (P.U.) ZONE.

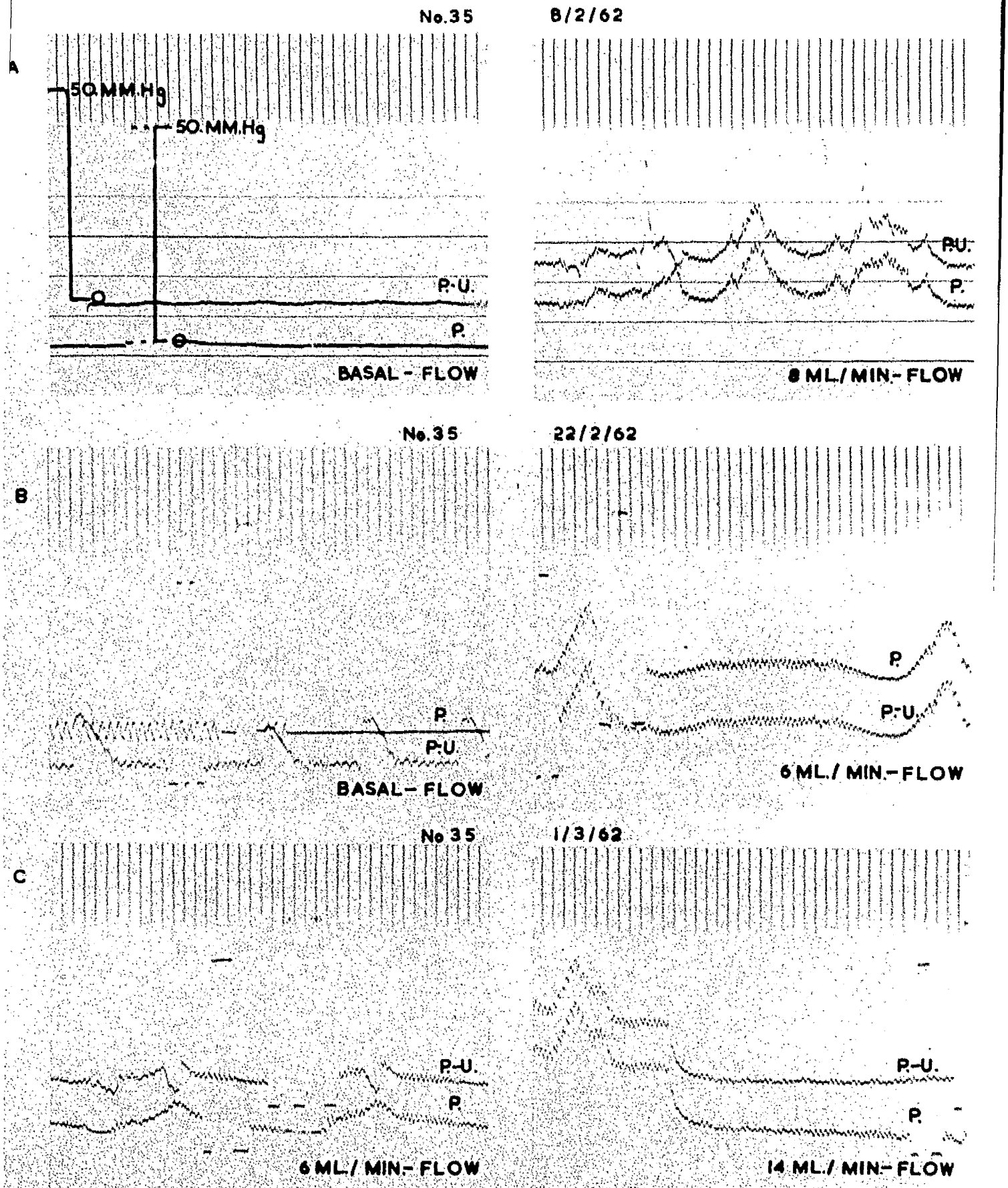

FIG. 37

A PELVIC (P) AND PELVI-URETERAL (PU) BASAL PRESSURE TRACES SIMILAR

B PELVI-URETERAL (PU) BASAL PRESSURE TRACE OF "URETERIC" TYPE

C AT HIGH URINE FLOW RATES SIMILAR WAVE FORM AND PRESSURE IN PELVIS (P) AND PELVI-URETERAL (PU) ZONE.

type of wave
diuresis, the urete_ _ _ _ _ _ ave disappeared and the pelvic and pelvi-ureteral pressure complexes became identical. Fig. 37 C. Infusion experiments also showed a similar change from "ureteric" to "pelvic" waves (Fig.38) - at low infusion rates "ureteric" waves were recorded from the pelvi-ureteral tube, whereas at higher flow rates the contraction complexes were more characteristic of those occurring in the pelvis.

PELVIC AND URETERIC PRESSURES: None of the implanted ureteric tubes remained satisfactory and diuretic pressure studies were obtained in only four of the ten preparations. The position of the ureteric tube, the number of diuretic studies and the cause of failure of function of the tube are recorded in Table V.

Recordings from the functioning T-tubes during diuresis, showed that the ureteral contraction wave complex might vary in detail (Fig.39). There was, however, a gross overall pattern during diuresis. At basal pelvic pressures and in the early part of the standard osmotic diuresis, the standing pressure in the ureter was low and ureteric waves of medium amplitude occurred at regular intervals, (Fig.40 A). As flow increased the amplitude of the waves increased, then

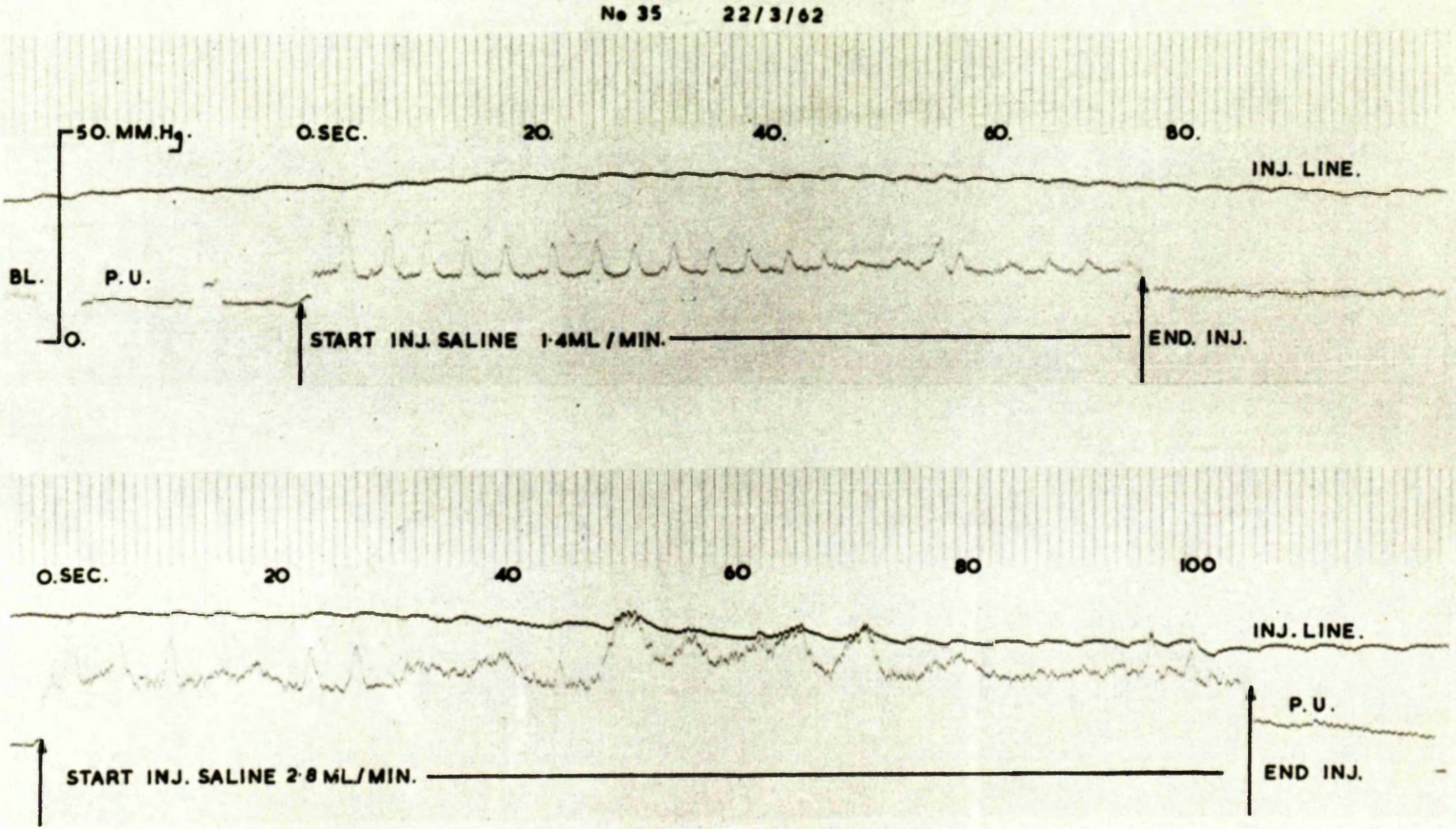

FIG. 38 CHANGE IN FORM OF WAVE AT PELVI-URETERAL ZONE CAUSED BY HIGHER RATE OF INFUSION.

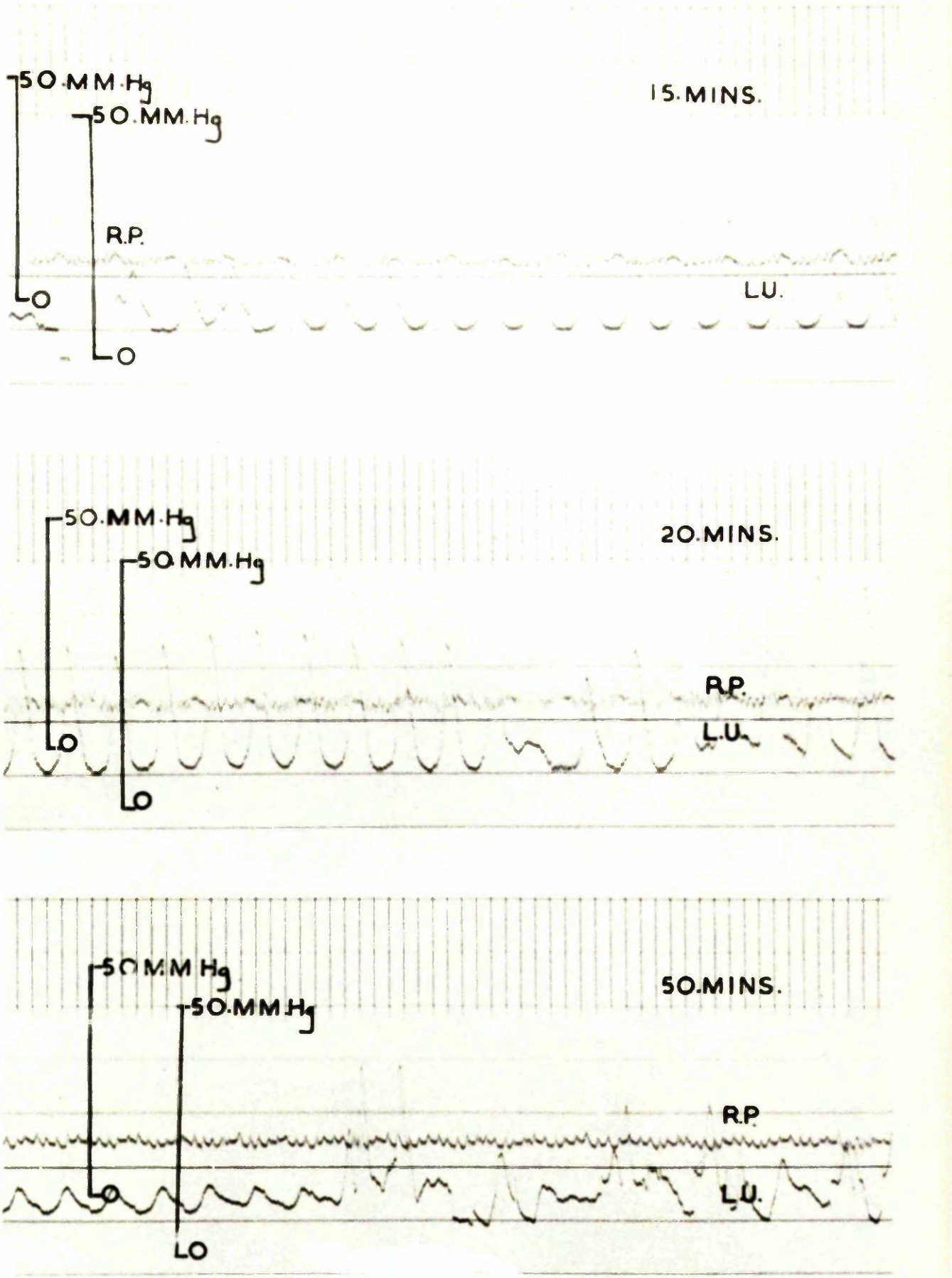

FIG. 39 DIFFERENT FORMS OF URETERIC WAVE RECORDED FROM LEFT URETER.

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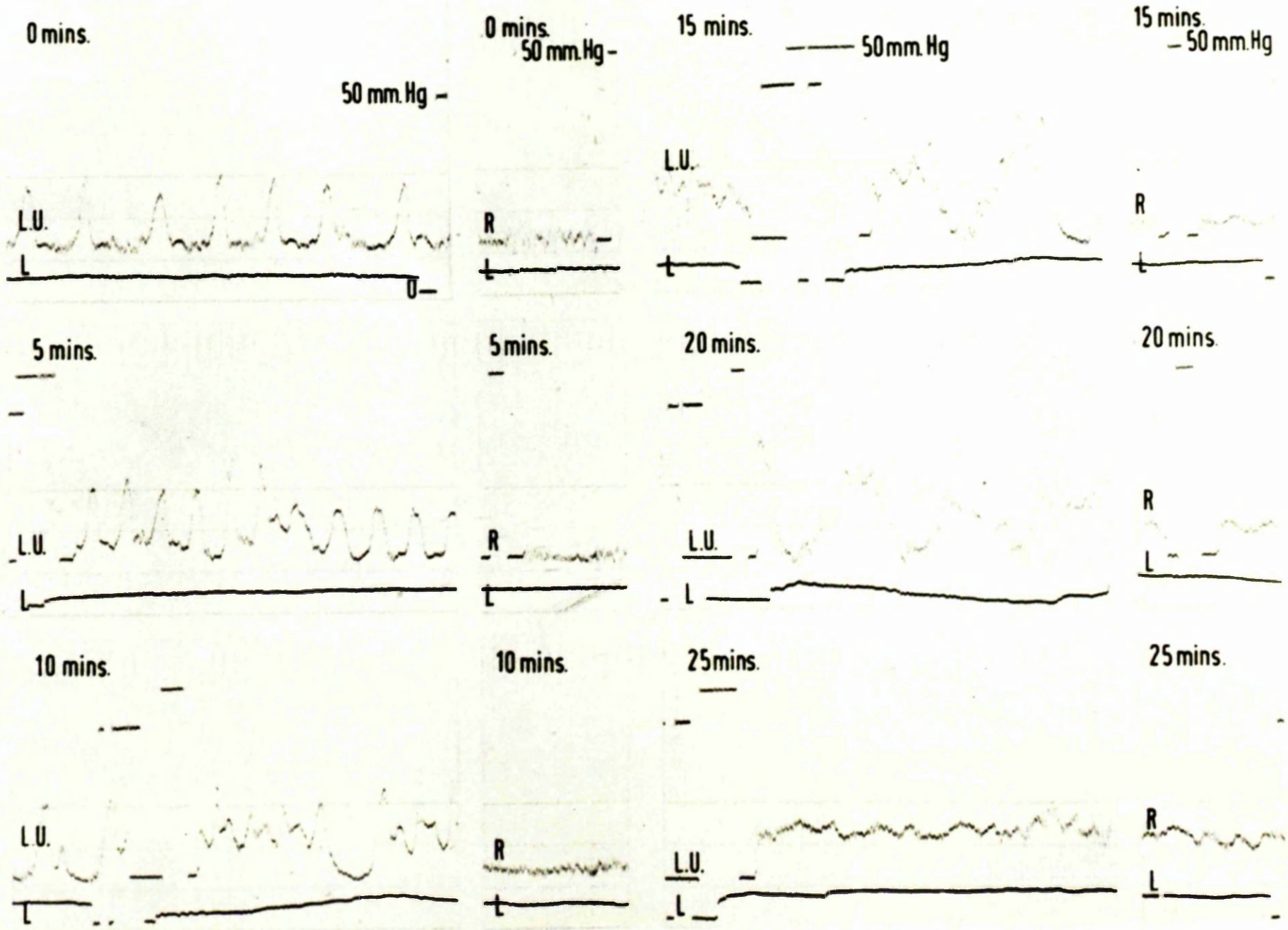

FIG. 40A PELVIC AND URETERIC PRESSURES RECORDING DURING DIURESIS

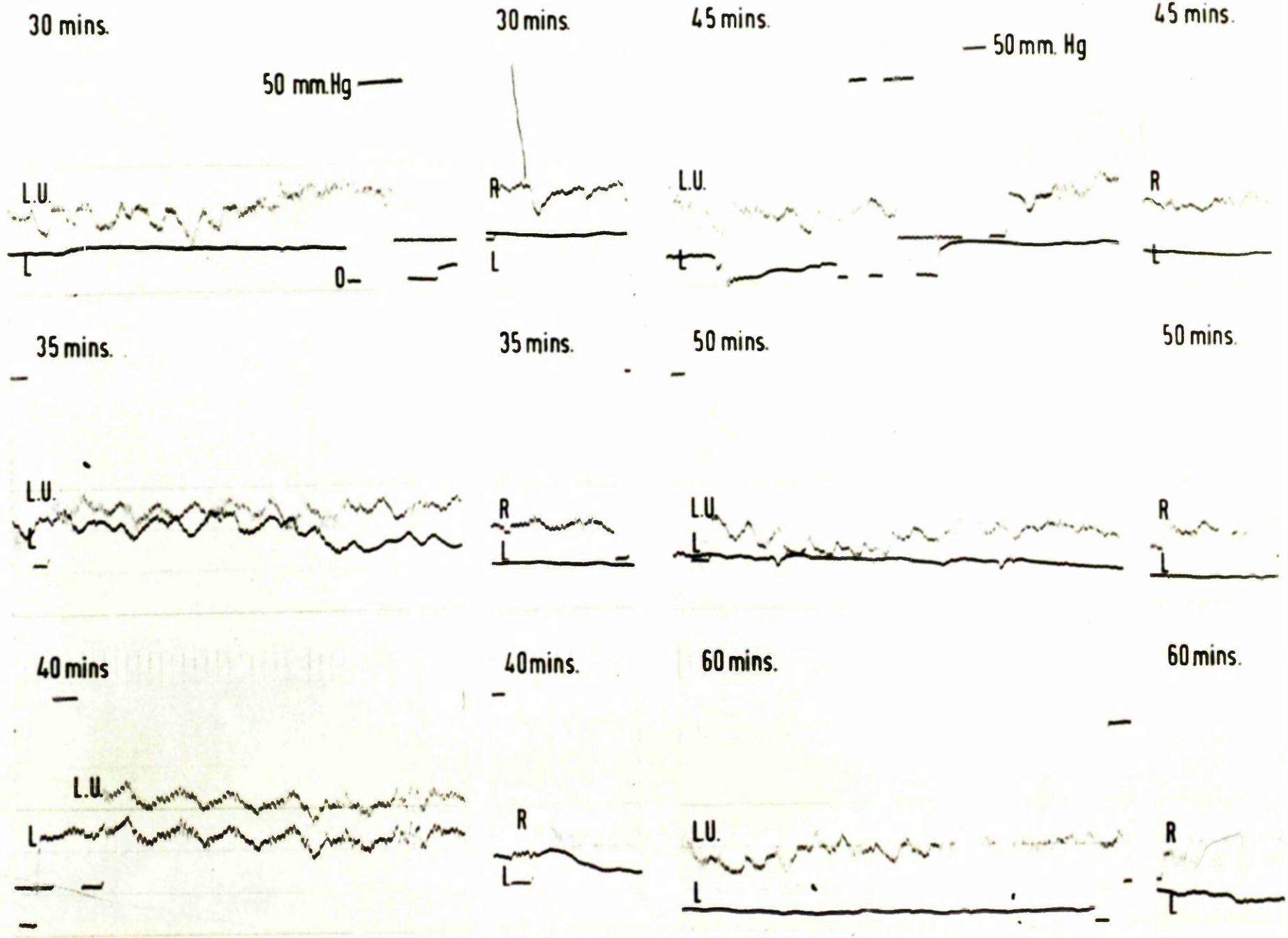

FIG. 40B COMMON WAVE PATTERN IN PELVIS AND URETER AT HIGH URINE FLOW RATES.

the standing ureteric pressure increased, and the contraction complexes became irregular, with a broader base and diminishing amplitude Fig. 40 B. The pelvic and ureteric waves, which had been dissociated, became, during the full flow of diuresis, identical in form and amplitude. Finally, as the diuretic flow diminished, the ureteric and pelvic waves were again dissociated. In this sequence, comparison of the pressure in the control renal pelvis and the pelvis drained by the ureter with the T-tube did not suggest that the T-tube was causing obstruction even at a flow rate of 10 ml of urine per minute from both kidneys.

Pressure on the dog's lower abdomen during diuresis demonstrated that this manoeuvre produced obstruction to the flow of urine down the ureter, (Fig. 41), and also impeded pelvic drainage.

The reasons for the malfunction of the implanted ureteric T-tubes and the subsequent abandonment of the experiments are described below.

Leakage of urine round the tube with the formation of retroperitoneal abscess occurred in No. 9, and the experiment was terminated. The plain stainless steel T-tube implant in No.13 blocked within a few days with

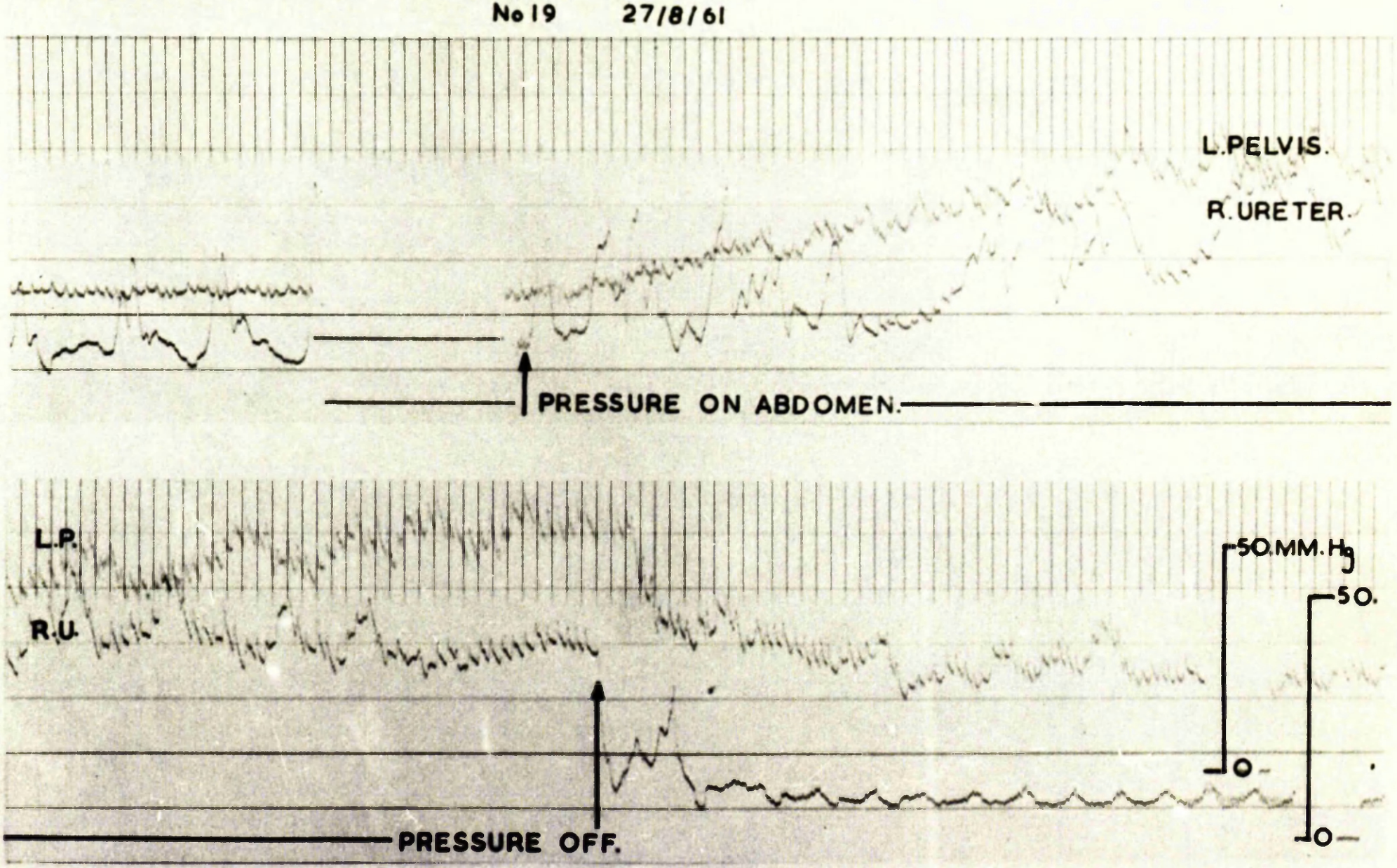

FIG. 41 ILLUSTRATES THE EFFECT OF INCREASED INTRA-ABDOMINAL PRESSURE ON PELVIC AND URETERIC PRESSURES.

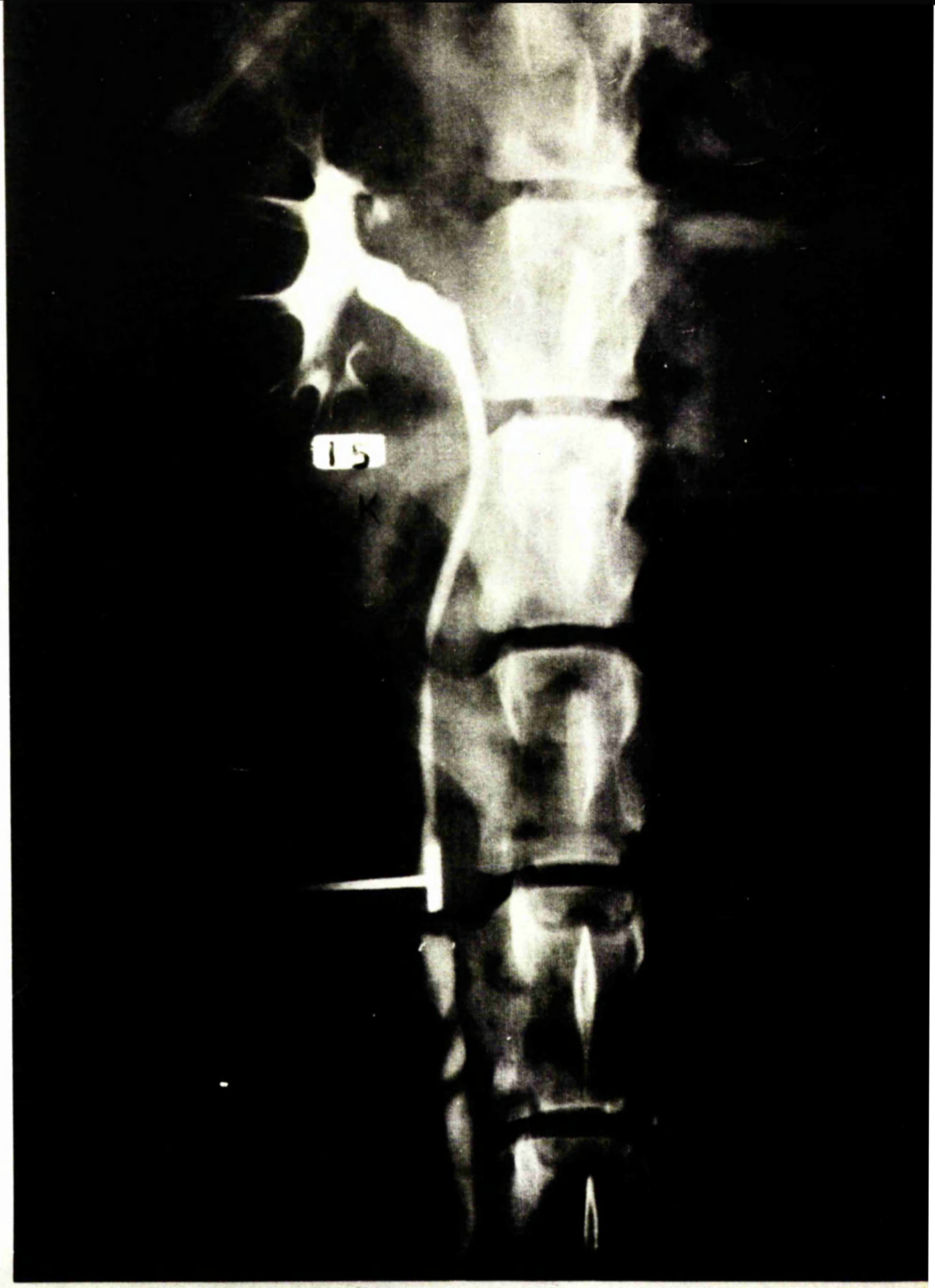

FIG. 42 ILLUSTRATES SATISFACTORY POSITION OF URETERIC T-TUBE

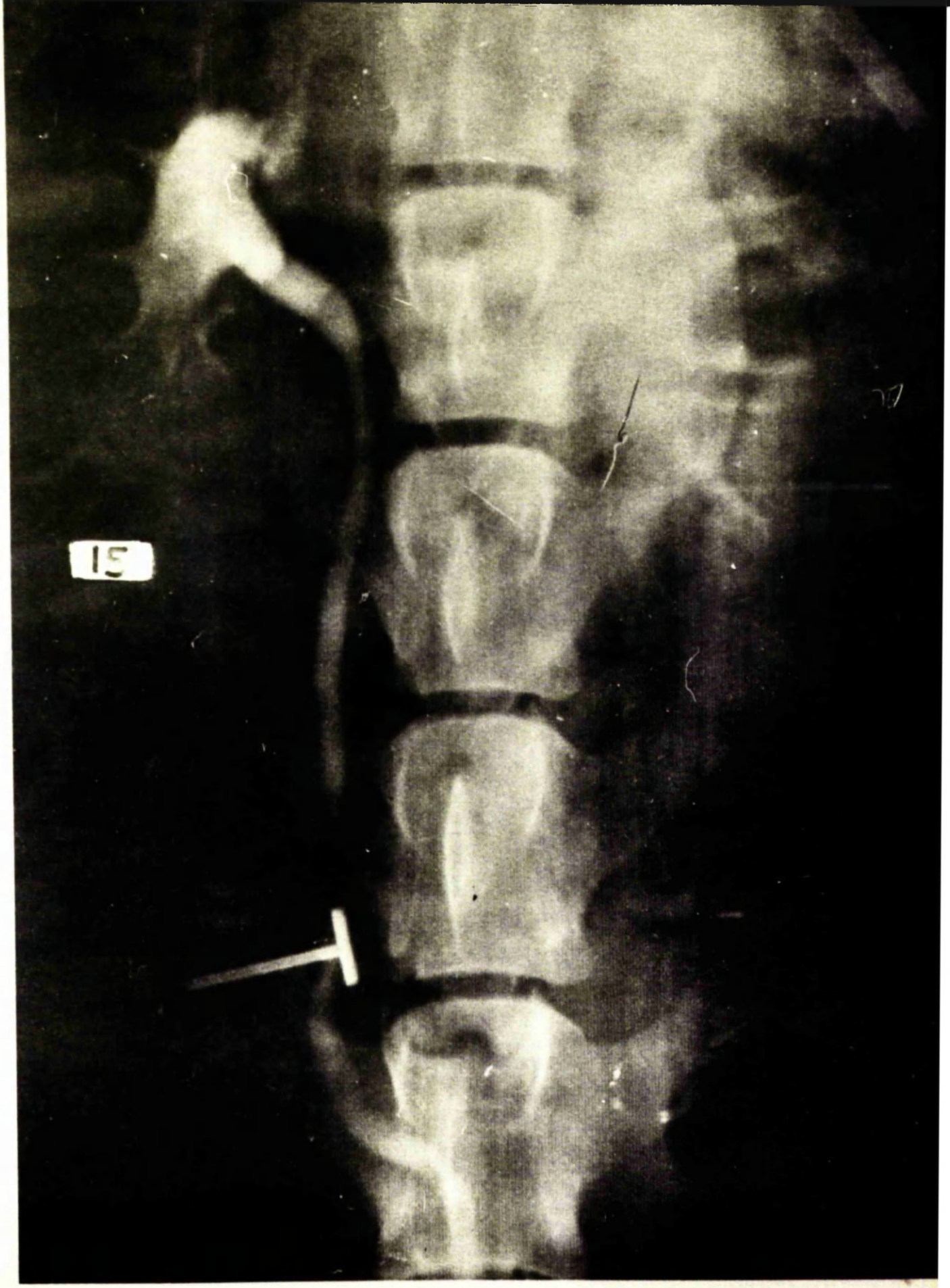

FIG. 43 T-TUBE ROTATED CAUSING HYDRONEPHROSIS.

calcareous deposit. Coating the stainless steel with PTFE proved a fairly satisfactory method of maintaining patency but in two preparations, Nos. 13 and 19, the tube blocked after pyelography. Infection of the skin track occurred in one instance (No. 4) after the dog had bitten the manometric tubes. An apparently satisfactory position on X-ray (Fig. 42) was no guarantee that the tube would not later become angled (Fig. 43), and some degree of angulation ultimately occurred in all the functioning tubes.

In Nos. 14, 18 and 19, at subsequent exploration or post-mortem examination, the ureters remained mobile in the retro-peritoneal tissue but the segment of ureter encompassing the T-tube was thickened and fibrosed. In No.4 there was considerable fibrosis of both the ureter and the retro-peritoneal tissue.

The details of the above experiments are given in the section, Protocols to Experiments.

DISCUSSION

The above studies will be discussed in the same order as that used for the presentation of the results.

I PELVIC PRESSURE IN THE NORMAL KIDNEY:

Lapides (1948) has indicated the need for measuring urine flow rates during studies on the upper urinary tract, and in man, there is evidence that, in some individuals, diuresis may produce distention of the renal pelvis (Covington and Roesser 1950). For these reasons it seemed important to correlate measurements of pelvic pressure with urine flow rates in this study of the hydrodynamics of the renal pelvis.

To provide a baseline for subsequent pressure measurements it was decided that pelvic pressures recorded when the urine flow rate was less than one ml per kidney, per minute, would be basal pelvic pressures. In favour of this choice of baseline was the finding that unless a dog had recently taken a drink of water, the urine flow rate was less than this level, and it was also considered that collection errors would be significant with smaller volumes of urine. It can, however, be argued that this flow rate is higher than

the average flow calculated from a 24 hour urine collection in the dog. In practice it proved a satisfactory baseline as basal pelvic pressures were found to be low, ranging from zero to a few mm.Hg. The basal pressure records contained no significant contraction waves and no phases of negative pressure.

PELVIC PRESSURE AND URINE FLOW RATE:

As we have seen from the review of the literature there have been few studies of pressures in the renal pelvis and in none of these has the urine flow rate been systematically varied. Previous workers (Kiil 1957) when they have desired to alter the rate of fluid transport, have infused fluid directly into the pelvis at a known injection speed, but it has not been shown that this technique reproduces in full the conditions of diuresis. Osmotic diuresis was used in this study to obtain the widest possible range of urine flow rates. With this method it was found that as urine flow rate increased the basal pelvic pressure was modified by an increased standing pelvic pressure and by the development of phasic contraction waves. In general, highest pelvic pressures were associated with highest urine flow rates but it was not found possible

to correlate changes in pelvis pressure with changes in urine flow rates. A similar increase in renal pelvic pressure in the anaesthetised dog was recorded by Morales and others (1952). As pelvic infusion techniques in dog and man (Kill 1957) have indicated that pressure in the renal pelvis remains low and that the pelvis musculature is only capable of small pressure fluctuations, there is a strong suggestion that a factor other than liquid volume affects the dynamics of the renal pelvis during diuresis.

PELVIC PRESSURE AND BLADDER PRESSURE:

Kill (1957) found that, in man, intravesical pressure was not reflected in the renal pelvis and in this study at low urine flow rates, similar observations were made in the dog. During diuresis, however, it was noted in an animal in which, for technical reasons, the bladder was not catheterised, that the renal pelvic pressures were significantly higher than when the bladder had been continuously drained. Further studies were therefore made and these confirmed that an increasing vesical pressure during diuresis, either naturally or artificially induced, resulted in higher standing pelvic pressures and larger contraction complexes than were found at comparable stages in diuresis when the bladder

was empty. It was also found that emptying the bladder by catheter caused a fall in the pelvic pressure. This phenomenon is at variance with the concept derived from work in dogs by Lucas (1908) that the pressure in the renal pelvis is isolated by the ureter from changes in bladder pressure. A possible mechanism for the increased pelvic pressures will be discussed, in the section on the autotransplanted kidney.

Although the renal pelvic pressures reported in this study were measured in the unipapillary dog pelvis, the pelvic pressures recorded at casual urine flow rates in man, by Rattner and others (1957) suggested that pressures at least as high as those which have been reported above in the dog, may develop in the multipapillary human pelvis under comparable conditions.

The effects of sustained diuresis and raised bladder pressures on pelvic pressures observed in this study suggest a possible mechanism for the production of the dilatation of the upper urinary tract observed in a high percentage of pregnancies in the human female, Baird (1932). It has generally been considered that this "physiological" dilatation was due to a "hormonal atony" of the ureter (Van Wagenan and Jenkins 1939) or

alternatively, that it was caused by direct pressure of the uterus against the ureters at the pelvic brim. The absence of ureteric dilatation in the pregnant quadruped (Mongert 1934) is against a purely hormonal mechanism, but on the other hand, Baird (1952) has pointed out that a purely mechanical explanation is unlikely as large ovarian cysts are associated with a lesser degree of dilatation. Recent observations on the urinary tract in pregnancy are of interest in the light of the above findings in the dog. Polydipsia and polyuria occur from the second trimister of pregnancy (Jeffcoate 1962) and the observations of Gould, Hsieh and Tinklor (1955) have shown that bladder pressure at the seventh month of pregnancy in the erect female was 40 cms of water compared with 15 cms in the supine position. Bladder pressures for normal controls were 22 cms and 6 cms respectively. The figure of 40 cms water is considerably higher than the bladder pressures recorded by Murphy and Schoenberg (1960) in cases of bladder outlet obstruction. Kiil (1957) found evidence of increased pressure in the renal pelvis during the fifth month of pregnancy, but these pelvic pressures were recorded when the bladder pressure was only a few mm.Hg.

There is thus some evidence that the dilatation of the upper urinary tract in pregnancy is produced by a hydrodynamic mechanism, and it would therefore be interesting to determine the pressure in the renal pelvis of the pregnant female when the intravesical pressure was maintained at the level found when the patient was in the erect posture.

II PELVIC PRESSURES: AUTOTRANSPLANTED KIDNEYS.

There is no agreement in the literature on the role that the autonomic nervous system plays in the dynamics of the upper urinary tract. It has been variously considered to control mobility (Hatz 1950), to regulate tone (Gould and others 1955) or to have no effect at all (Lapides 1948).

The pelvic pressures in the autotransplanted kidneys reported in this thesis suggest that division of the extrinsic nerve supply to the upper urinary tract has no effect on the dynamics of the renal pelvis. Basal pelvic pressures, the changes in pelvic pressures during diuresis and the pelvic pressure response to variations in bladder pressure were the same as those found in the normal kidney. No evidence was found to support the suggestion by Goodwin and Kaufmann (1956) that kidney transplantation, by destroying the "safety valve" of

lymphatic drainage from the kidney, would result in excessively high pelvic pressures during diuresis. Pyelograms made at low pelvic pressures indicated an apparently normal calibre of the upper urinary tract. These findings support the impression of Quinby (1916), derived from dog experiments, and of Murray, Merrill and Harrison (1958) from kidney transplantation in man, that urinary track motility is normal after transplantation of the kidney.

If the tone is regulated by the autonomic nervous system, one would expect some evidence of loss of regulation after kidney transplantation. From the literature it is not clear what changes could be expected as there is no uniform concept of tone or indeed of how changes in tone might be measured. A common view (Marath 1951) is that an alteration of tone would produce a change in calibre of the upper urinary track while the intra-luminal pressure remains constant. Denervation, however, did not affect either the calibre of the tract as demonstrated by pyelography, or the pressure in the pelvis. In certain preparations there was transient dilatation of the pelvis and ureter but this was associated with a temporary increase in intrapelvic pressure. It was probable that these

changes were caused by oedema at the uretero-vesical anastamosis, as this was a cause of temporary obstruction in a proportion of autotransplanted kidneys (O'Conor 1961). Tinckler (1956) considered that the renal pelvis was protected from high bladder pressures by changes in tone in the ureter. The most marked changes in tone were thought to occur in the lower ureter and these changes were probably mediated by a reflex arc with different impulses arising in the bladder wall. This present study fails to support such a mechanism as not only was the normal renal pelvis not protected from changes in intravesical pressure, but autotransplantation of the kidney, which removed the extrinsic nerve supply and the lower ureter, did not produce any different intra-pelvic pressure response from that occurring in the normal pelvis when the intravesical pressure was altered during diuresis.

III

a) PELVIC PRESSURES AND ACUTE URETERIC OBSTRUCTION

The pressure changes recorded from the renal pelvis and distal ureter in acute ureteric obstruction in anaesthetised dogs have been previously documented

by Risholm and others (1959) and in man a similar ureteric response was recorded by Kiil (1957). The decrease in the peristaltic waves in acute obstruction was held by Campbell (1954) to be caused by ureteral overdistension causing anaesthesia of nerve endings thus disrupting the peristaltic arc. Kiil (1957), however, commenting on the correlation between the reduction in amplitude of the contraction complexes and the elevation of the standing pressure, thought there was a "functional dependency between the activity of the tract and the standing pressure". He considered that the increase in the standing pressure was partly due to passive distention and partly due to an increase in tone. An alternative mechanism is suggested from this study by the development of the common pressure pattern in the pelvis and ureter. It is postulated that a continuous high pressure column of fluid produced by the force of renal secretion (Hinman 1954) distends the ureter and pelvis progressively stretching the spiral muscle fibres described by Schneider (1938) thus impairing the contractile power of these fibres with a consequent fall in the amplitude of the contraction complexes.

b) PELVIC PRESSURES AND URETERO-COLIC ANASTAMOSIS:

As transplantation of the ureter into the colon frequently leads to hydronephrosis, Dempster (1957) suggested that this would be a suitable technique for evaluating the changes produced by subacute ureteric obstruction. It was found that uretero-colic anastamosis of the Coffey type produced an immediate increase in the standing pelvic pressure accompanied by phasic contraction waves of varying amplitude. Although the pressures were not higher than the pressure recorded from the normal pelvis during diuresis, pyelography demonstrated that the upper urinary tract was dilated and this finding suggests that a continuous increase in standing pressure is a factor in the aetiology of hydronephrosis.

There are no studies in the literature on pelvic pressure in chronic hydronephrosis in the experimental animal, but in man measurements have failed to show any significant increase in intrapelvic pressure (Underwood 1937). Kiil (1957) considered that although pelvic pressure was probably increased in periods of pelvic expansion, a state of equilibrium would be reached when the renal pelvic pressure was not appreciably increased, whereas Nolick, Karellis

and Maryka (1961) postulated some as yet unknown mechanism of the pelvic or ureteral muscle being responsible for the low pressures.

The pressure measurements in the small number of preparations which were available for prolonged study indicated that there was a gradual fall in resting intrapelvic pressure and in the pressure response to diuresis. In spite of the finding of "normal" pelvic pressures, pyelograms demonstrated that the hydronephrotic state persisted.

The diminishing pressure response to diuresis is most satisfactorily explained by gradual destruction of renal secretory tissue, either by direct back-pressure from the pelvis (Hjort 1953) or indirectly by the hydronephrotic pelvis obstructing venous drainage from the kidney (Sheehan and Davis 1959). An alternative explanation for the fall in pressure would be the re-absorption of urine by pyelotubular and pyelointerstitial back-flow (Kasin, Perchy and Staroaski 1960), possibly supplemented by re-absorption through the ureter (Rodrigues 1960). These mechanisms should, however, tend to protect the kidney parenchyma from atrophy.

IV ADDITIONAL STUDIES

a) The pattern of pelvic pressures observed in this study in the infusion experiments in dogs is very similar to that found by Kiil (1957) in man. This study suggests that the method of recording pelvic pressures in man by a small indwelling ureteric catheter (Kiil 1953) does not significantly affect the emptying mechanism of the renal pelvis, and it also provides evidence which indicates that the apparently conflicting renal pelvic pressures recorded in man and dog by previous workers (Kiil 1957, Rattner and others 1957) could be the result of different urine flow rates and bladder pressures obtaining in the respective experiments. It would, therefore, be of interest to make further studies of pelvic pressures in man employing Kiil's "urometric" technique under controlled conditions of urine flow rates and bladder pressure.

b) Pelvic pressures and pelvic movements: Considerable information about the emptying mechanism of the renal pelvis might be gained from a simultaneous study of pelvic pressures and movements. Unfortunately, the conditions necessary for adequate definition during excretion urography preclude observations during high urine flow rates. Because

of this, radiographic studies were made using direct infusion of the contrast medium, combined in some instances with osmotic diuresis. Possibly because pressures were being recorded from the intrarenal pelvis and peri-ureteral region, no correlation was found between pelvic pressures and pelvic movements. Another possible explanation is suggested by calculations of Bojesen (1954) - he determined that the volume of the canine renal pelvis increased in a linear fashion with urine flow rate until the pelvis reached a maximum volume at a flow rate of 4-5 ml per minute. As the total flow rates during radiography generally exceeded this critical figure it is probable that the pelvis was maximally distended and that the pressure changes reflected isometric contractions of the pelvic muscle.

c) Pressures in the pelvi-ureteral junction: It was thought that a knowledge of the pressures in the zone of transition from the pelvis to the ureter might also contribute to an understanding of the pelvic emptying mechanism. In physiological terms, Kiil (1957) has defined the pelvi-ureteral junction as "the proximal level where the contraction complexes characteristic of the ureter are recorded". He found that, in man, this area lay in the anatomical pelvis

and that pelvic distention caused it to move distally. He concluded that the pressure and wave characteristics of the pelvi-ureteral junction, could be explained by the peristaltic contraction wave occluding the lumen of the distal pelvis. In this study it was also found that the area of transition from the low, pelvic wave, to the peaked, ureteric wave, occurred in the extra-renal pelvis and that this area of transition was not fixed, but apparently moved distally during diuresis and during the direct infusion of fluid into the pelvis.

d) Pelvic and ureteric pressures: Simultaneous pressure studies from the pelvis and ureter would be expected to provide information about the integration of the hydrodynamics of these parts of the upper urinary tract. In this study pressures were recorded from the ureter through a small T-tube, a technique which had previously been used in acute animal experiments by Lucas (1908). Scott and deLuca (1960), considered that a T-tube technique would not grossly affect ureteric peristalsis and that it was a preferable method to a catheter passed in a retrograde fashion which must interfere with the uretero-vesical valve mechanism. It became clear during the study, however, that any transgression of the ureteral wall

interfered with the longitudinal movement of the ureter which occurs in the peri-ureteral sheath during each peristaltic wave (Engelmann 1869). Although pelvic pressures in some of the preparations did not indicate a significant obstruction to urine flow, no precise significance can be attached to these combined pressure observations. There was, however, a suggestion that at low and moderate urine flow rates ureteric waves transported urine, and that at higher flow rates, the pelvis and upper part of the ureter formed a common pumping chamber with a high standing pressure. Such a change in pattern of urine transport might explain the observations of Morales and others (1952) that during extreme diuresis the peristaltic nature of the efflux from the ureteric orifice was replaced by a continuous flow of urine.

SUMMARY

1. A new technique for recording pressures in the renal pelvis is described in which a "permanent" monomotric nephrostomy tube is inserted into the dog's kidney. This method is not subject to the disadvantages of previous techniques and permits the systematic recording of pressures under physiological and pathological conditions of the upper urinary tract.

2. This method has made possible the study of pressures in the renal pelvis under the following conditions:

i) Low urine flow rates
ii) Diuresis
iii) Varying bladder pressures
iv) Renal autotransplantation
v) Acute ureteric obstruction
vi) Uretero-colic anastamosis

3. The results obtained have shown that:-

i) The pressures in the renal pelvis at low urine flow rates range from zero to a

few mm.Hg. Contraction complexes rarely occur and are of low amplitude.

ii) Diuresis is associated with an increase in the standing pelvis pressure which may exceed 20 mm.Hg. Contraction waves do occur and the amplitude of the waves may be 10 mm.Hg. In general highest urine flow rates are associated with highest pelvic pressures, but the increases in pelvic pressure could not be quantitated with narrow ranges of urine flow.

iii) Changes in bladder pressure during diuresis are directly reflected by changes in pressure in the renal pelvis.

iv) The renal pelvic pressures recorded in the auto-transplanted kidney are of the same order as those found in the normal kidney, and there is a similar response to diuresis and changes in bladder pressure.

v) In acute ureteric obstruction, after an initial increase in amplitude, there is a fall in height of the ureteric contraction waves, and this is associated at first with an increase in the standing pressure

in the ureter, then with an increase in the standing pressure in the pelvis. Within a varying period a common standing pressure of approximately 20 mm.Hg. is recorded from the pelvis and the ureter, and contraction waves are small and irregular.

vi) Immediately after uretero-colic anastamosis there is an increase in the standing pressure in the renal pelvis, ranging from 10 - 33 mm.Hg. at low urine flow rates. Contraction waves of amplitudes up to 9 mm.Hg. also occur. To begin with osmotic diuresis results in a further increase in the standing pressure and a slight increase in the amplitude of the contraction complexes. Thereafter a gradual fall occurs in the resting and diuretic pressures and at 14 weeks after the operation in the three surviving animals in this small series, the pelvic pressure lay within the normal range. At this period osmotic diuresis resulted only in a small increase in pelvic pressure.

CONCLUSIONS

From the above experiments it may be concluded that:

1. The pressure in the renal pelvis ranges from zero to a few mm.Hg. at low urine flow rates.

2. Pressures in the renal pelvis increase during diuresis, and contraction waves of moderate amplitude occur frequently.

3. Changes in bladder pressure are directly reflected by changes in pressure in the renal pelvis during diuresis.

4. Autotransplanting the kidney does not modify pelvic pressures.

5. Uretero-colic anastamosis is immediately followed by a high standing pressure in the renal pelvis with in most preparations large contraction waves. Further increases in pressure occur during diuresis. There is a gradual fall in these pressures which ultimately lie within the normal basal range in spite of the occurrence of stenosis at the uretero-colic junction.

These findings have made it possible to draw some conclusions on the part played by the renal pelvis in the transport of urine. At low urine flow rates the pelvis would appear to play a relatively passive role in urine transport, but in the physiological conditions of diuresis, and particularly when bladder pressure is high, the pelvis plays a more active role. These changes in role are not affected by depriving the renal pelvis of its extrinsic nerve supply. After uretero-colic anastamosis the pelvis also acts as a pump, but after a period of time there is a slow fall in pressure to the normal range, and this apparent reversion to a relatively passive role is associated with a loss of pelvic pressure response to osmotic diuresis.

This dual role of the renal pelvis may explain the apparently contradictory findings reported by previous workers who have studied pressures in the pelvis of the kidney.

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TABLE I - - NORMAL INTRAPELVIC PRESSURES

DOG NO:	MANO-METRIC TUBE	PERIOD OF STUDY (wks)	BASAL PELV. PRESS. RANGE (mm.Hg)	Diuretic Studies		
					Minimum & Maximum pressure (mm.Hg.)	
				No.	Bladder Empty	Bladder Full
No.1	Lt	13	0 - 2	2	10 - 15	-
2	Lt	26	0 - 1	3	7 - 10	28 - 38
3	Rt	7	0 - 1	1	19 - 24	-
5	Rt	4	0 - 6	3	16 - 24	-
6	Rt	6	0 - 1	1	20 - 23	-
	Lt	6	0 - 1	2	16 - 21	-
7	Rt	12	0 - 2	2	13 - 21	-
	Lt	16	0 - 7	4	8 - 18	-
8	Rt	5	0 - 3	3	12 - 17	-
	Lt	5	0	2	11 - 18	-
9	Rt	4	0	1	22 - 25	-
	Lt	4	0 - 3	1	26 - 29	-
11	Lt	5	0 - 1	2	5 - 8	-
14	Rt	7	0 - 1	1	12 - 16	-
17	Lt	15	0 - 1	3	12 - 20	-
19	Lt	8	0 - 5	2	10 - 14	-

TABLE I (Cont) - NORMAL INTRAPELVIC PRESSURES

DOG NO:	MANO-METRIC TUBE	PERIOD OF STUDY (wks)	BASAL PELV. PRESSURE RANGE (mm.Hg)	Diuretic Studies Minimum & Maximum pressure range		
				No.	Bladder Empty	Bladder Full
20	Lt	8	0 - 7	3	19 - 21	31 - 42
	Rt	23	0 - 5	3	19 - 22	19 - 26
21	Lt	6	3 - 4	1	-	12 - 18
22	Rt	16	0 - 7	2	-	35 - 47
23	Rt	8	0 - 10	4	-	33 - 49
41	Rt	5	0 - 1	2	17 - 23	-
43	Rt	9	0 - 1	2	20 - 27	26 - 32
45	Rt	3	0 - 2	1	11 - 13	-
	Lt	3	0	1	15 - 20	-
46	Rt	4	1	1	-	24 - 35
	Lt	4	0	1	-	18 - 27

TABLE II - RENAL AUTOTRANSPLANT SERIES

DOG NO:	AUTO NO:	AUTOTRANSPLANT KIDNEY	AUTOTRANSPLANT SITE	Opposite Kidney	COMPLICATIONS
10	A 1	Rt	Iliac	Control	Arterial thrombosis
17	A 2	Rt	Iliac	"	-
22	A 3	Lt	Iliac	"	-
23	A 4	Lt	Iliac	"	-
24	A 5	Lt	Iliac	"	Arterial thrombosis
25	A 6	Lt	Iliac	"	Gastric Dilatation
26	A 7	Lt	Iliac	Nephr.	Intussusception
27	A 8	Lt	Iliac	"	Intussusception
28	A 9	Lt	Iliac	"	Arterial thrombosis
29	A 10	Lt	Iliac	"	Intussusception
30	A 11	Lt	Iliac	"	Anaesthetic death
31	A 12	Lt	Iliac	"	-
32	A 13	Lt	Iliac	"	Intussusception
33	A 14	Lt	Iliac	"	Perinephric abscess
34	A 15	Lt	Iliac	"	Intussusception
36	A 16	Lt	Iliac	"	Arterial thrombosis
37	A 17	Lt	Iliac	"	" "
38	A 18	Lt	Renal	"	" "
39	A 19	Rt	Iliac	"	" "
40	A 20	Lt	Renal	"	Venous thrombosis
42	A 21	Lt	Renal	"	Arterial thrombosis
43	A 22	Lt	Renal	Control	-
44	A 23	Rt	Iliac	"	-

TABLE III - AUTOTRANSPLANT PRESSURE STUDIES

DOG No:	PERIOD OF STUDY (wks)	BASAL PELV. PRESS. RANGE (mm.Hg)	Diuretic Studies		
			No.	Minimum & Maximum Press. (mm Hg)	
				BLADDER Empty	BLADDER Full
17 (A 2)	14	0 - 7	3	17 - 19	-
22 (A 3)	13	0 - 9	4	-	22 - 28
23 (A 4)	8	18 - 21 (7)	4	-	44 - 50
31 (A12)	23	12 (4)	7	37 - 72	32 - 37
33 (A14)	4	33 - 40 (7-9)	4	18 - 21	50 - 55
34 (A15)	1	27 - 31	1	28 - 32	-
43 (A22)	19	0 - 7	3	19 - 27	40 - 49
44 (A23)	11	5 - 8	2	23 - 26	-

NOTE Figures in brackets are pressures recorded 4 weeks after transplant

TABLE IV - PRESSURES AFTER URETERO-COLIC ANASTAMOSIS

Dog No:	Coffey Ureteroc-colic Anast.	1 wk Post Anast.		14 wk Post Anast.	
		Resting Pressure (mm.Hg)	Diuretic Max.Press (mm.Hg)	Resting Pressure (mm.Hg)	Diuretic Max.Press (mm.Hg)
No. 1	Lt	17 - 23	30 - 45	5	5
No. 3	Rt	22 - 27	22 - 36	1 - 2	8 - 29
No. 5	Rt	25	33	-	-
	Lt	33	40	-	-
No. 7	Rt	23 - 32	31 - 40	-	-
No.11	Lt	21 - 23	36 - 38	-	-
No.14	Lt	10	18 - 25	-	-
No.15	Lt	15 - 17	18 - 20	-	-
No.20	Rt	22	55 - 62	5	11 - 13

TABLE V - URETERIC T-TUBE STUDIES

DOG NO:	TYPE OF TUBE	URETER	NO. OF DIURETIC STUDIES	COMPLICATIONS
3	T	L-MID	-	TUBE ROTATED
4	T	L-UPPER	3	TUBES BITTEN
9	L	L-MID	-	ABSCESS
10	T	L-MID	-	TUBE ROTATED
13	T	L-MID	-	TUBE BLOCKED
14	T	L-MID	4	TUBE ROTATED
15	T	L-MID	-	TUBE ROTATED
18	T	L-UPPER	2	TUBE ROTATED
19	T	R-MID	2	TUBE ROTATED
23	T	R-UPPER	-	TUBE BLOCKED

PROTOCOLS TO EXPERIMENTS

A ANIMAL PREPARATIONS

DOG No. 1

18. 2.60	Left nephrostomy: Badenoch technique
1. 4.60	Osmotic diuresis
8. 4.60	Water diuresis
21. 4.60	Descending pyelography; tube position satisfactory
19. 5.60	Left uretero-colic anastamosis
7. 6.60	Osmotic diuresis
14. 6.60	Descending pyelography; Hydronephrosis and hydroureter
21. 6.60	Osmotic diuresis
23. 8.60	Experiment terminated: Left kidney atrophic with hydronephrosis and hydroureter. Stenosis of uretero-colic anastamosis to pin-hole size.

DOG No. 2

25. 4.60	Left nephrostomy: Badenoch technique: tube to perirenal space: right nephrectomy.
2. 6.60	Osmotic diuresis: perinephric tube blocked.
30. 9.60	Descending pyelogram; tube position satisfactory.
6.10.60	Osmotic diuresis: bladder not catheterised.

18.10.60	Osmotic diuresis: bladder not catheterised.
26.10.60	Experiment terminated as tube bitten off. Position of manometric tube mid-pelvis.

DOG No.3

20.10.60	Left and right nephrostomies: Badenoch technique: Upper third ureter intubated with Teflon T-tube
24.10.60	Osmotic diuresis: left nephrostomy tube unsatisfactory. Left ureterogram - T-tube angulated.
1.11.60	Right uretero-colic anastomosis.
8.11.60	Osmotic diuresis.
12.11.60	Osmotic diuresis.
15.11.60	Osmotic diuresis.
27. 1.61	Osmotic diuresis.
10. 2.61	Osmotic diuresis.
14. 2.61	Experiment terminated: Marked stenosis of uretero-colic anastomosis.

DOG No.4

18.10.60	Left nephrostomy: Upper third ureter intubated with Teflon T-tube
24.10.60	Osmotic diuresis.
3.11.60	Osmotic diuresis. Urine volume
7.11.60	Osmotic diuresis.
5. 1.61	Experiment terminated as dog had bitten through all tubes, and had low-grade abscess of tracks.

DOG No.5

31.10.60	Left and right nephrostomies: Badenoch technique.
4.11.60	Osmotic diuresis
11.11.60	Osmotic diuresis.
17.11.60	Descending pyelogram; tip of left nephrostomy tube in pelvi-ureteral zone.
23.11.60	Osmotic diuresis
29.11.60	Bilateral ureteroileocolic anastamoses
5.12.60	Osmotic diuresis
13.12.60	Animal died: right pyonephrosis

DOG No.6

3.11.60	Left and right nephrostomies: Badenoch technique.
12.11.60	Osmotic diuresis
14.11.60	Osmotic diuresis: Left pelvic pressures - urine volumes. Right nephrostomy tube unsatisfactory.
2.12.60	Experiment terminated: Right nephrostomy tube not in pelvis, position left tube satisfactory, pressure sequence during acute ureteric obstruction.

DOG No.7

21.11.60	Left and right nephrostomies: Badenoch technique
2.12.60	Osmotic diuresis
9.12.60	Osmotic diuresis

16. 2.61	Right uretero-colic anastamosis, pressure sequence during acute ureteric obstruction.
10. 3.61	Osmotic diuresis
17. 3.61	Osmotic diuresis
17. 3.61	Experiment terminated as animal had a right pyonephrosis, position left nephrostomy tube satisfactory, stenosis right ureteral anastamosis

DOG No.8

30.12.60	Left and right nephrostomies. Soldinger technique.
6. 1.61	Osmotic diuresis: Left nephrostomy tube unsatisfactory.
16. 1.61	Osmotic diuresis
23. 1.61	Osmotic diuresis
6. 2.61	Experiment terminated as animal had pulled out both nephrostomy tubes.

DOG No.9

12.1.61	Left and right nephrostomies. Soldinger technique.
20.1.61	Osmotic diuresis
8.3.61	Middle third left ureter intubated with Teflon L tube
16.3.61	Experiment terminated as animal had developed retro-peritoneal abscess.

DOG No.10

6. 2.61	Right kidney autotransplanted to right iliac region. Left and right nephrostomies: Seldinger technique.
15. 2.61	Laparotomy: thrombosis right renal artery with infarction of kidney - nephrectomy. Middle third left ureter intubated with stainless steel T-tube.
21. 2.61	Experiment terminated, T-tube at angle to ureter

DOG No.11

14.2.61	Left and right nephrostomies: Seldinger technique.
3.3.61	Descending pyelogram: right tube not in pelvis
13.3.61	Osmotic diuresis
14.3.61	Osmotic diuresis
20.3.61	Left uretero-colic anastamosis, pressure sequence during acute ureteric obstruction.
21.3.61	Excretion urogram; left hydronephrosis.
24.3.61	Osmotic diuresis
7.4.61	Experiment terminated, left pyonephrosis. Patent uretero-colic anastamosis.

DOG No.12

15. 3.61	Left nephrostomy: Seldinger technique: right nephrectomy
6. 4.61	Experiment terminated as animal had pulled out tube.

DOG No.13

10. 4.61	Left nephrostomy: Seldinger technique. Middle third left ureter intubated with stainless steel T-tube coated with Teflon.
24. 4.61	Experiment terminated as T-tube blocked with calcareous debris.

DOG No.14

13. 4.61	Left and right nephrostomies: Seldinger technique. Middle third left ureter intubated with Teflon coated stainless steel T-tube.
24. 4.61	Osmotic diuresis
25. 4.61	Descending pyelogram, T-tube satisfactory.
28. 4.61	Osmotic diuresis - failure of recording equipment.
1. 5.61	Osmotic diuresis.
12. 5.61	Osmotic diuresis
1. 6.61	Osmotic diuresis
2. 6.61	Experiment terminated, pressure sequence during acute ureteric obstruction.

DOG No.15

4. 5.61	Left and right nephrostomies. Seldinger technique. Middle third right ureter intubated with Teflon coated stainless steel T-channel.
16. 5.61	Excretion urogram - position of T-channel satisfactory and no proximal dilatation.
20. 5.61	Osmotic diuresis: neither nephrostomy tube satisfactory.

26. 5.61	Osmotic diuresis: left nephrostomy tube satisfactory.
30. 5.61	Excretion urogram - mild hydronephrosis, T-channel apparently not in ureter.
8. 6.61	Left uretero-colic anastamosis, pressure sequence during acute ureteric obstruction.
16. 6.61	Osmotic diuresis
18. 7 61	Experiment terminated as tubes bitten off. Top of right nephrostomy tube in kidney tissue, considerable fibrosis around T-channel. Left hydronephrosis with stenosis of uretero-colic anastamosis

DOG No.16

15. 5.61	Left and right nephrostomies: Seldinger technique.
25. 5.61	Experiment terminated as dog had bitten off tubes.

DOG No.17

25. 5.61	Right and left nephrostomies: Seldinger technique. Right renal autotransplant to right iliac vessels.
30. 5.61	Excretion urogram: good function both kidneys.
9. 6. 61	Osmotic diuresis
16. 6.61	Osmotic diuresis

28. 6.61	Osmotic diuresis
4. 8.61	Osmotic diuresis. Recording unsatisfactory.
30. 8.61	Osmotic diuresis. Part of recording accidentally destroyed.
8. 9.61	Experiment terminated. Position of nephrostomy tubes satisfactory.

DOG No.18

5.6. 61	Right and left nephrostomies: Seldinger technique. Upper third left ureter intubated with Teflon-coated stainless steel T-tube.
19. 6.61	Osmotic diuresis: Right nephrostomy tube unsatisfactory.
19. 6.61	Descending pyelogram: T-tube rotated partly obstructing ureter.
24. 6.61	Osmotic diuresis: Right nephrostomy tube unsatisfactory.
26. 6.61	Experiment terminated as dog had bitten tubes

DOG No.19

29. 6.61	Right and left nephrostomies: Seldinger technique. Middle third right ureter intubated with a Teflon coated stainless steel T-tube
31. 7.61	Excretion urogram: slight rotation of T-tube
3. 8. 61	Osmotic diuresis

27. 8.61	Osmotic diuresis
30. 8.61	Experiment terminated: Slight fibrosis around T-tube with minimal hydronephrosis. Position of nephrostomy tubes satisfactory.

DOG No.20

24. 8.61	Right and left nephrostomies: Seldinger technique.
1. 9.61	Osmotic diuresis
7. 9.61	Osmotic diuresis - left tube functioning intermittently
14. 9.61	Osmotic diuresis
20.10.61	Right uretero-colic anastamosis, pressure sequence during acute obstruction.
3.11.61	Osmotic diuresis - left tube faulty.
20.11.61	Osmotic diuresis - left tube faulty.
27.11.61	Osmotic diuresis - left tube faulty.
25. 1.62	Osmotic diuresis.
1. 2.62	Experiment terminated. Right hydronephrosis. Position of nephrostomy tubes satisfactory.

DOG No.21

25. 8.61	Two nephrostomy tubes inserted left kidney, one tube right kidney: Seldinger technique.
15. 9.61	Descending pyelography.
18. 9.61	Direct infusion of fluid into pelvis. Osmotic diuresis.
9.10.61	Experiment terminated because of tube tracks. Position of tubes satisfactory.

DOG No.22

31. 8.61	Right and left nephrostomies. Seldinger technique. Left renal autotransplant to left iliac vessels.
14. 9.61	Osmotic diuresis
15. 9.61	Excretion urogram: good function.
22. 9 61	Osmotic diuresis
13.10.61	Osmotic diuresis, right tube faulty.
9.11.61	Right ureter divided and implanted in bladder by double flap technique.
17.11.61	Osmotic diuresis
1.12.61	Osmotic diuresis: Pressure record damaged in camera.
1.12.61	Experiment terminated. Left uretero-vesical anastamosis satisfactory, slight stenosis of right anastamosis. Position of nephrostomy tubes satisfactory.

DOG No.23

8. 9.61	Right and left nephrostomies: Seldinger technique. Left renal autotransplant to left iliac vessels.
15. 9.61	Excretion urogram: Dilatation of left pelvis and ureter.
15. 9.61	Osmotic diuresis.
25. 9.61	Osmotic diuresis.
9.10.61	Osmotic diuresis.
16.10.61	Osmotic diuresis.

26.10.61	Upper third right ureter intubated with Teflon coated stainless steel T-tube
3.11.61	T-tube blocked.
7.11.61	Experiment terminated. Left ureter stenosed at vesical anastemosis. Nephrostomy tubes satisfactory. T-tube blocked with calcareous debris.

DOG No.24

12.10.61	Right and left nephrostomies; Seldinger technique. Left renal autotransplant to left iliac vessels.
16.10.61	Left kidney not functioning.
17.10.61	Experiment terminated. Left kidney infected - arterial thrombosis.

DOG No.25

2.11.61	Right and left nephrostomies: Seldinger technique. Left renal autotransplant to left iliac vessels.
3.11.61	Dog dead; gastric dilatation.

DOG No.26

6.11.61	Left nephrostomy: Seldinger technique. Left renal autotransplant to right iliac vessels. Right nephrectomy.
10.11.61	Dog vomited once, but clinically well.
11.11.61	Dog dead. Ileo-ileal intussusception.

DOG No.27

13.11.61	Left nephrostomy: Seldinger technique. Left renal autotransplant to right iliac vessels. Right nephrectomy.
16.11.61	Dog vomited. Laparatomy - intussusception, easily reduced.
20.11.61	Dog died from pneumonia.

DOG No.28

4.12.61	Left nephrostomy: Seldinger technique. Left renal autotransplant to right iliac vessels - arterial spasm. Right nephrectomy.
9.12.61	Experiment terminated as dog anuric. Arterial thrombosis with renal infarction.

DOG No.29

7.12.61	Left nephrostomy: Seldinger technique. Left renal autotransplant to left iliac vessels. Right nephrectomy.
11.12.61	Laparotomy: Ileo-ileal intussusception reduced.
14.12.61	Laparotomy: Ileo-ileal intussusception. Gut resected.
18.12.61	Experiment terminated: partial disruption of anastamosis.

DOG No. 30

11.12.61	Left nephrectomy: Seldinger technique. Left renal autotransplant to right iliac vessels. Right nephrectomy.
12.12.61	Dog having colic and passing blood per rectum. Laparotomy - spastic colon. Anaesthetic death.

DOG No. 31

1. 1.62	Left nephrostomy: Seldinger technique. Left autotransplant to right iliac vessels.
5. 1.62	Excretion urogram - slight hydronephrosis of kidney.
5. 1.62	Osmotic diuresis
8. 1.62	Right nephrectomy
11. 1.62	Osmotic diuresis
18. 1.62	Osmotic diuresis
29. 1.62	Descending pyelogram: no dilatation.
9. 2.62	Osmotic diuresis
15. 2.62	Osmotic diuresis
25. 5.62	Osmotic diuresis
31. 5.62	Descending pyelogram - hydronephrosis.
15. 6.62	Water diuresis. Blood urea 28 mgm %
27. 6.62	Experiment terminated. 48 ml of urine in the pelvis. Position of tube satisfactory.

DOG No.32

4. 1.62	Left nephrostomy: Seldinger technique. Left autotranoplant to right iliac vessels.
8. 1.62	Right nephrectomy.
11. 1.62	Laparotomy. Ileo-colic intussusception. Resected.
12. 1.62	Dog dead. Had inhaled vomitus.

DOG No.33

15. 1.62	Left nephrostomy: Seldinger technique Left autotranoplant to right iliac vessels.
19.1.62	Osmotic diuresis
22.1.62	Excretion urogram: moderate left hydronephrosis
22.1.62	Right nephrectomy.
26.1.62	Osmotic diuresis
2.2.62	Osmotic diuresis
9.2.62	Osmotic diuresis
14.2.62	Experiment terminated because of escape of pus alongside nephrostomy tube - perinephric abscess.

DOG No.34

29. 1.62	Right nephrectomy
13. 2.62	Left nephrectomy: Seldinger technique
15. 2.62	Descending pyelogram: hydronephrosis

	Ileal
16. 2.62	Osmotic diuresis
19. 2.62	Dog dead. Silent ileo-ileal intussusception. Stenosis at uretero-vesical anastamosis.

DOG No. 35

5.2. 62	Two tubes inserted in right kidney, one to pelvis, the second to pelvi-ureteral zone: Seldinger technique. Left nephrectomy.
8. 2.62	Osmotic diuresis
22. 2.62	Descending pyelogram to determine position of tubes.
22. 2.62	Osmotic diuresis
1. 3.62	Osmotic diuresis
22. 3.62	Direct infusion experiment
29. 3.62	Teflon tube removed from pyelo-ureteral tube. Direct infusion experiment.
30. 3.62	Direct infusion experiment.
5. 4.62	Direct infusion experiment.
12. 4.62	Experiment terminated.

DOG No. 36

19. 2.62	Right nephrectomy
27. 2.62	Left nephrostomy. Seldinger technique. Left autotransplant to both internal iliac arteries as two renal arteries.
2. 3.62	Experiment terminated - arterial thrombosis with renal infarction.

DOG No. 37

9. 3.62	Right nephrectomy
19. 3.62	Left nephrostomy: Seldinger technique. Left auto-transplant to right pelvis.
23. 3.62	Experiment terminated. Arterial thrombosis with renal infarction.

DOG No.38

3. 4.62	Right nephrectomy.
9. 4.62	Left nephrostomy: Seldinger technique Left autotransplant to right pelvis.
12. 4.62	Experiment terminated. Arterial thrombosis with renal infarction.

DOG No.39

6. 4.62	Left nephrectomy
13. 4.62	Right nephrostomy: Seldinger technique. Right autotransplant to right pelvis.
17.4.62	Experiment terminated. Arterial thrombosis with renal infarction.

DOG No.40

24. 4.62	Left nephrostomy: Seldinger technique. Left autotransplant to renal vessels, ureter mobilised to bladder.
30. 4.62	Right nephrectomy
7. 5.62	Experiment terminated. Renal vein thrombosis.

DOG No.41

8. 5.62	Right nephrostomy: Seldinger technique. Left nephrectomy.
14.5.62	Osmotic diuresis
18.5.62	Water diuresis
14.6.62	Experiment terminated as dog had bitten tube.

DOG No.42

10. 5.62	Left nephrostomy: Seldinger technique. Left autotransplant to renal vessels, ureter mobilised to bladder.
17. 5.62	Right nephrectomy
19. 5.62	Experiment terminated. Arterial thrombosis, with renal infarction.

DOG No.43

17. 5.62	Right and left nephrostomies: Seldinger technique. Left autotransplant to renal vessels, ureter mobilised to bladder.
25. 5.62	Osmotic diuresis
31. 5.62	Osmotic diuresis. Right tube faulty.
25. 6.62	Osmotic diuresis
12. 7.62	Right and left uretero-vesical anastamoses
19. 7.62	Osmotic diuresis
16. 8.62	Osmotic diuresis

20. 8.62	Osmotic diuresis
2.10.62	Experiment terminated as dog had bitten tubes. Both tubes in satisfactory position.

DOG No.44

17. 5.62	Left nephrectomy.
24. 5.62	Right nephrostomy: Seldinger technique. Right autotransplant to right pelvis.
31. 5.62	Osmotic diuresis - nephrostomy tube faulty.
18. 6.62	Osmotic diuresis
3. 7.62	Osmotic diuresis
4. 7.62	Experiment terminated. Position of tubes satisfactory.

DOG No.45

12. 7.62	Two nephrostomy tubes inserted left kidney, one tube right kidney: Seldinger technique.
19. 7.62	Osmotic diuresis
24. 7.62	Direct infusion experiment: Pyelograms made.
30. 7.62	Experiment terminated as dog had developed abscess around the left tubes.

DOG No.46

18.10.62	Two nephrostomy tubes inserted left kidney, one tube right: Seldinger technique.
25.10.62	Osmotic diuresis: Cineradiography.
29.10.62	Osmotic diuresis: pressure record accidentally destroyed.
31.10.62	Osmotic diuresis
9.11.62	Experiment terminated. Position of tubes satisfactory.

PROTOCOL TO EXPERIMENTS

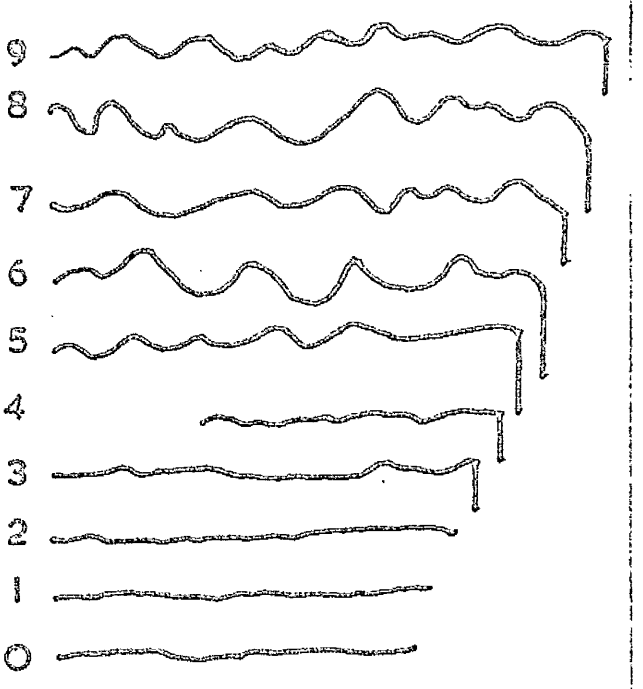
B. PRESSURE RECORDINGS

1) Normal Kidneys

No.1 1.4.60 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 (0)
5	37	0 (1)
10		0 (2)
15	75	5 (3)
20		5 (4)
25	143	8 - 11 (5)
30		10 - 15 (6)
35	184	5 - 9 (7)
40		7 - 14 (8)
45	77	5 (9)

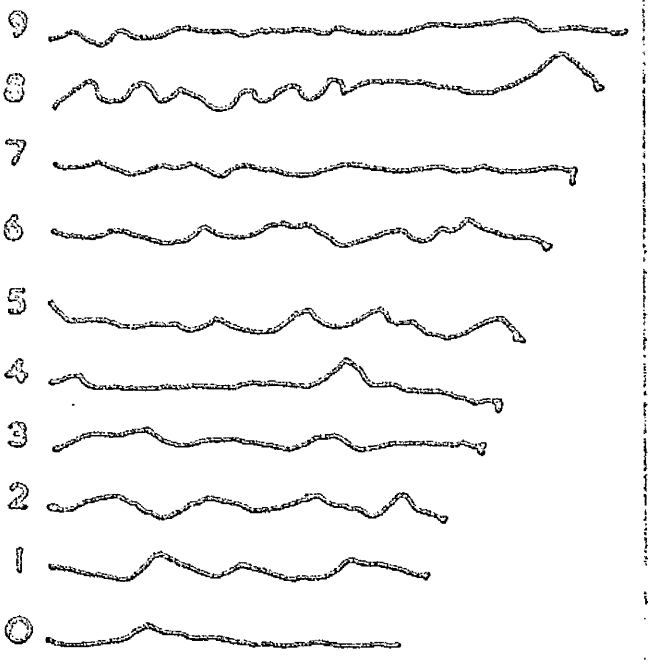
Pressure Tracing — 20 sec



No.1 8.4.60 Left Kidney Water Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 2 (0)
5	5	0 - 3 (1)
10	26	0 - 3 (2)
15		0 - 3 (3)
20	70	0 - 5 (4)
25		0 - 5 (5)
30	51	0 - 4 (6)
35		1 - 3 (7)
40	90	0 - 5 (8)
45		0 - 2 (9)

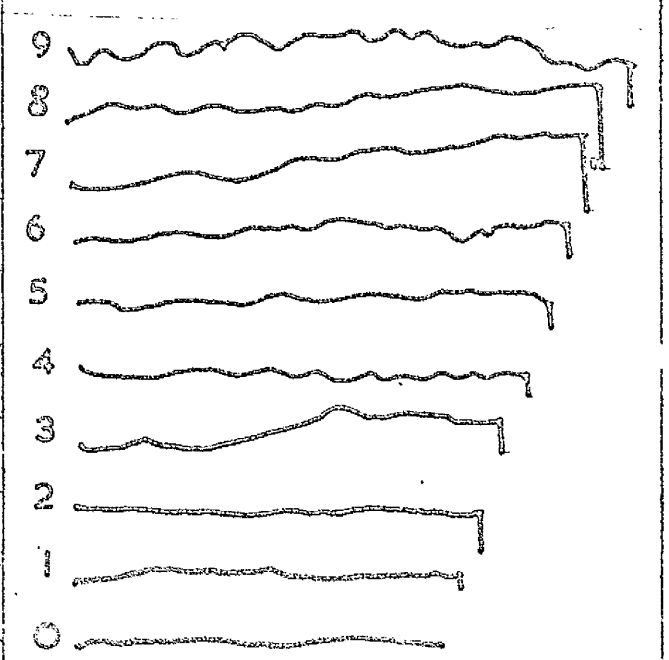
Pressure Tracing — 20 sec



No.2 2.6.60 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/10 min L. Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	9	0 - 2 (1)
10		3 - 4 (2)
15	17	1 - 6 (3)
20		2 - 4 (4)
25	34	2 - 5 (5)
30		3 - 5 (6)
35	48	4 - 9 (7)
40		7 - 10 (8)
45	-	5 - 10 (9)

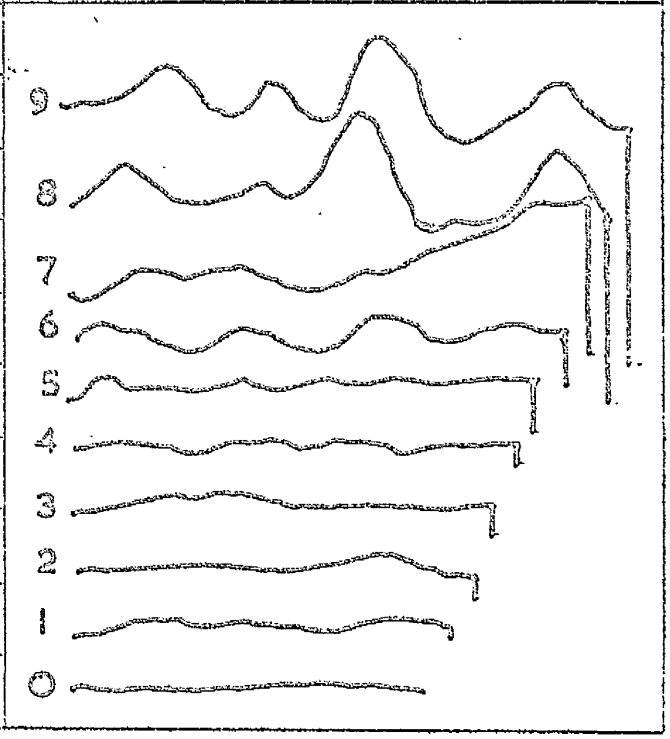
Pressure Tracing ——— 20 sec ———



No.2 6.10.60 Left Kidney Osmotic Diuresis

Time (Mins)	Bladder not drained	Pelv. Press mm Hg. (Tr.No)
Basal	-	0 (0)
5	-	1 - 2 (1)
10	-	3 - 5 (2)
15	-	3 - 5 (3)
20	-	3 - 5 (4)
25	-	6 - 8 (5)
30	-	5 - 9 (6)
35	-	8 - 18 (7)
40	-	20 - 35 (8)
45	-	28 - 38 (9)

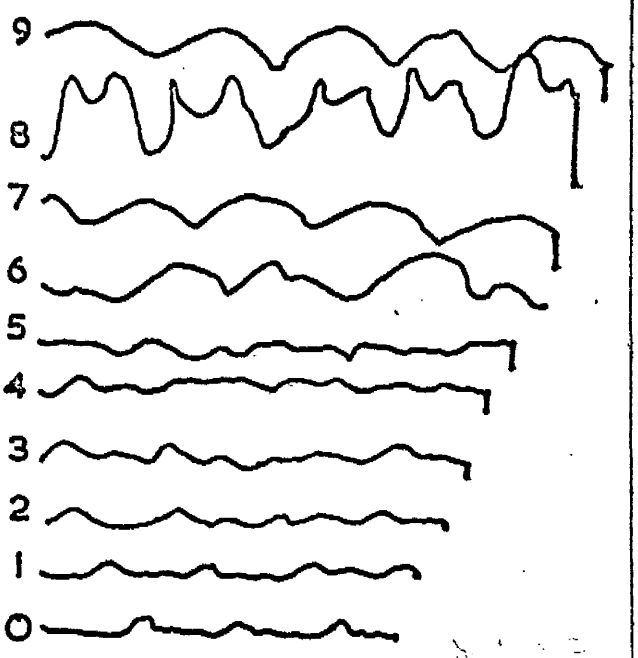
Pressure Tracing ——— 20 sec ———



No.2 18.10.60 Left Kidney Osmotic Diuresis

Time (Mins)	Bladder not drained	Pelv. Press mm Hg. (Tr.No)
Basal	-	0 - 1 (0)
5	-	0 - 1 (1)
10	-	0 - 2 (2)
15	-	2 - 5 (3)
20	-	1 - 3 (4)
25	-	2 - 5 (5)
30	-	0 - 7 (6)
35	-	3 - 8 (7)
40	-	4 - 15 (8)
45	-	3 - 8 (9)

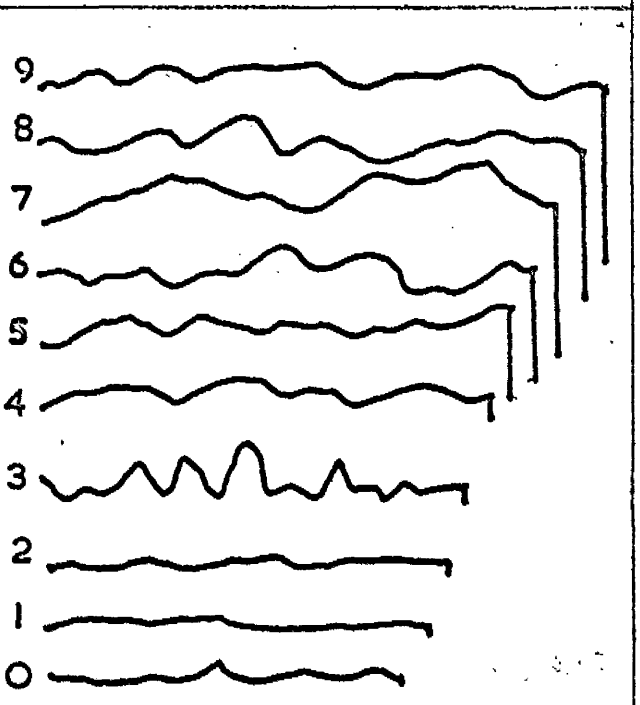
Pressure Tracing — 20 sec



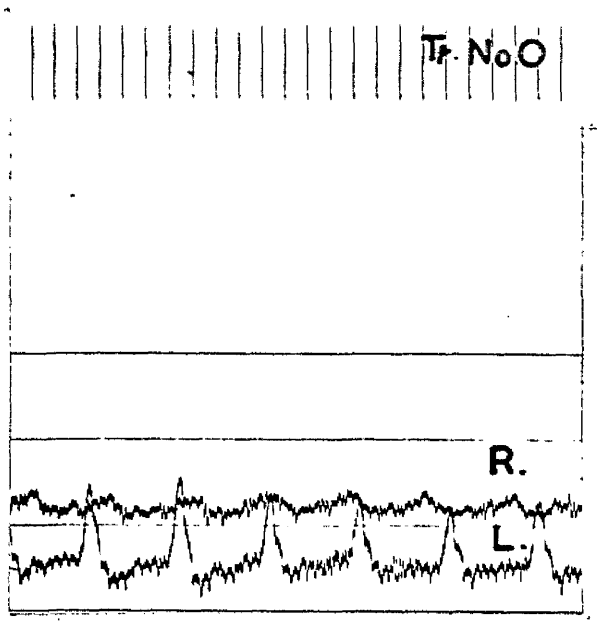
No.3 24.10.60 Right Kidney Osmotic Diuresis

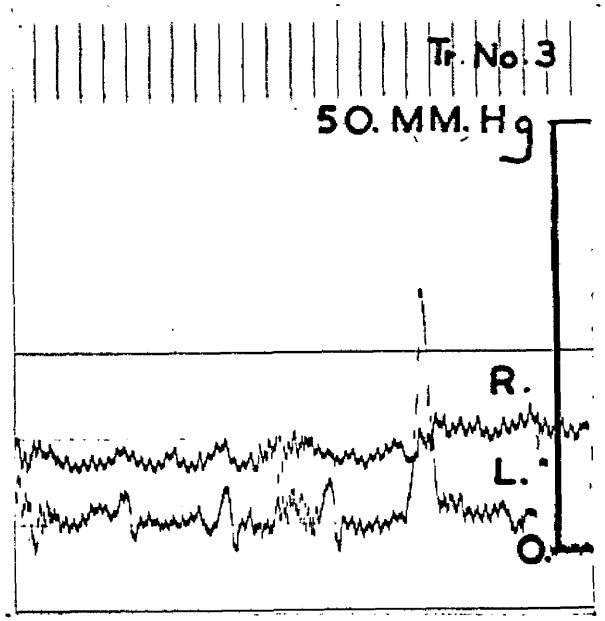
Time (Mins)	Urine/15 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	-	0 - 1 (0)
5	)	0 - 1 (1)
10	5))	0 - 1 (2)
15		1 - 7 (3)
20	)	2 - 5 (4)
25	15))	7 - 11 (5)
30		11 - 15 (6)
35		15 - 21 (7)
40	) 40)	17 - 21 (8)
45	)	19 - 24 (9)

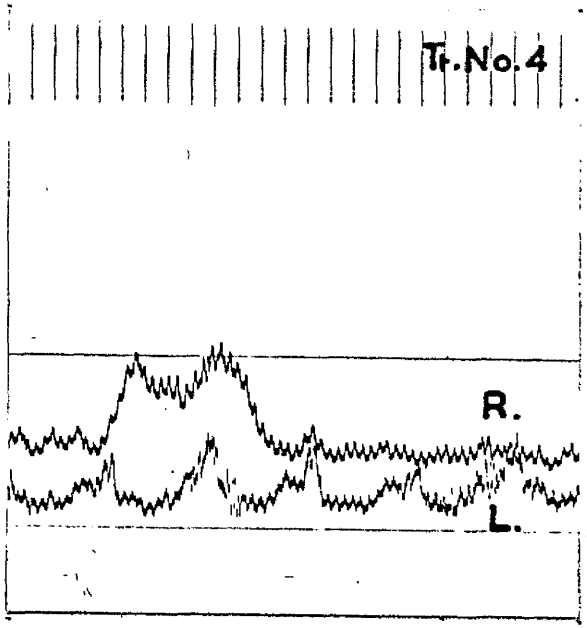
Pressure Tracing — 20 sec



No. 5 4/11/60







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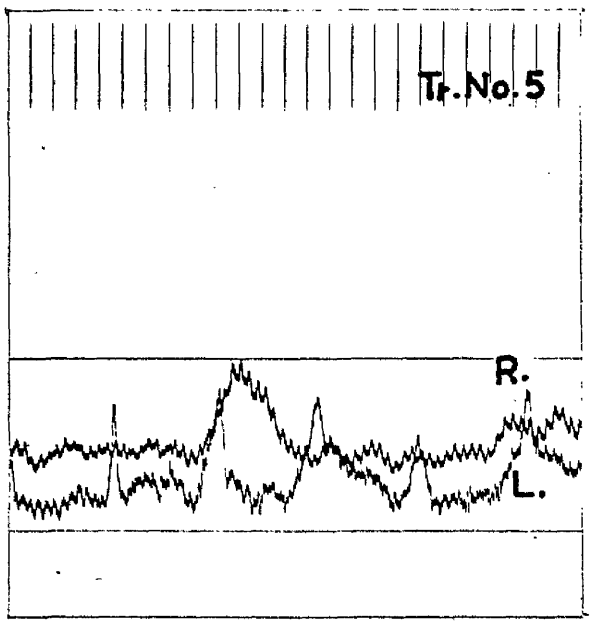
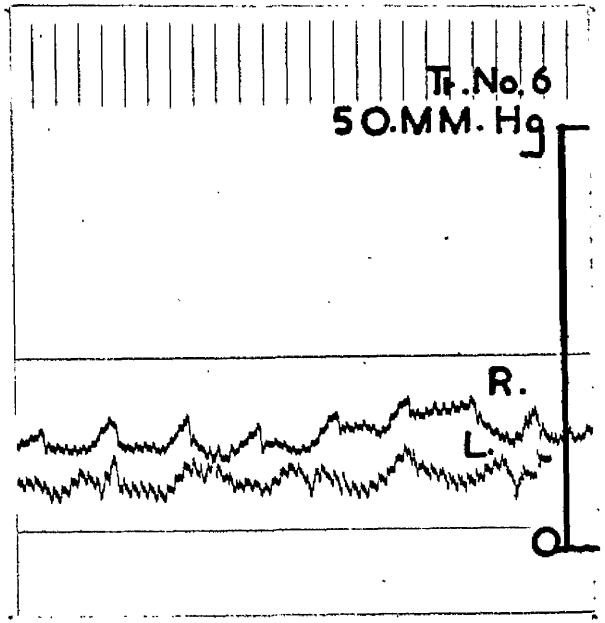
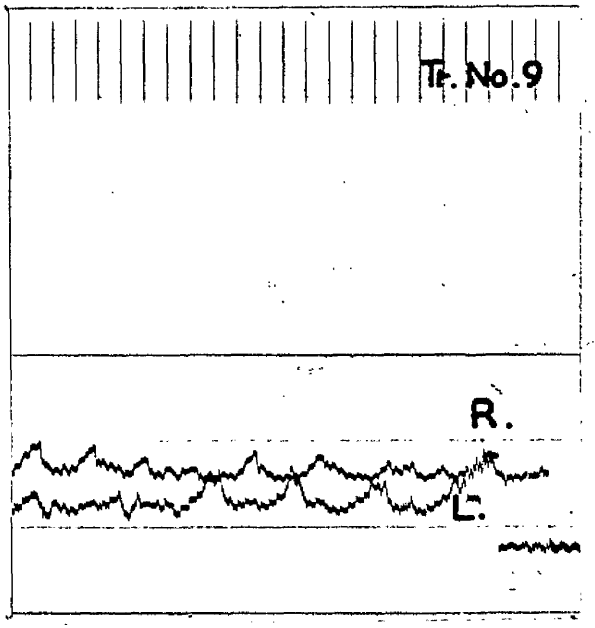


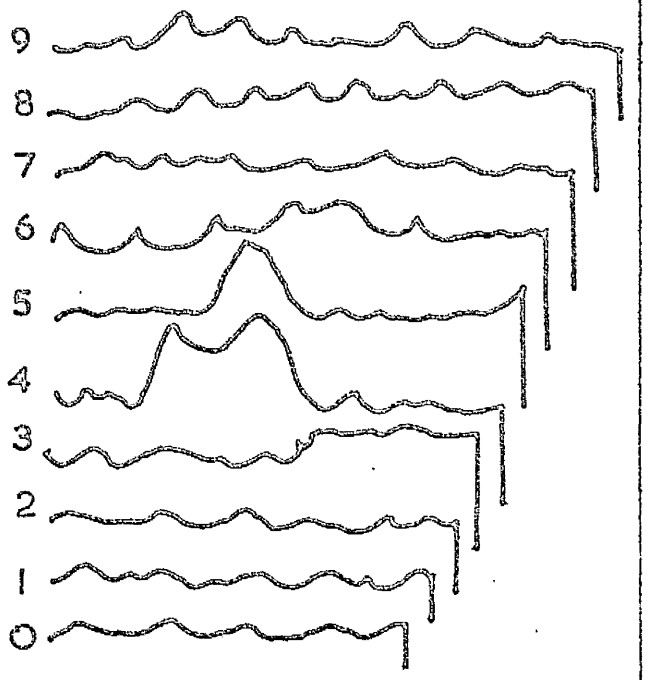




No.5 4.11.60 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	4 - 6 (0)
5	11	4 - 6 (1)
10	13	7 - 9 (2)
15	15	9 - 13 (3)
20	24	10 - 22 (4)
25	33	11 - 20 (5)
30	38	13 - 17 (6)
35	50	13 - 15 (7)
40	45	8 - 12 (8)
45	30	8 - 12 (9)

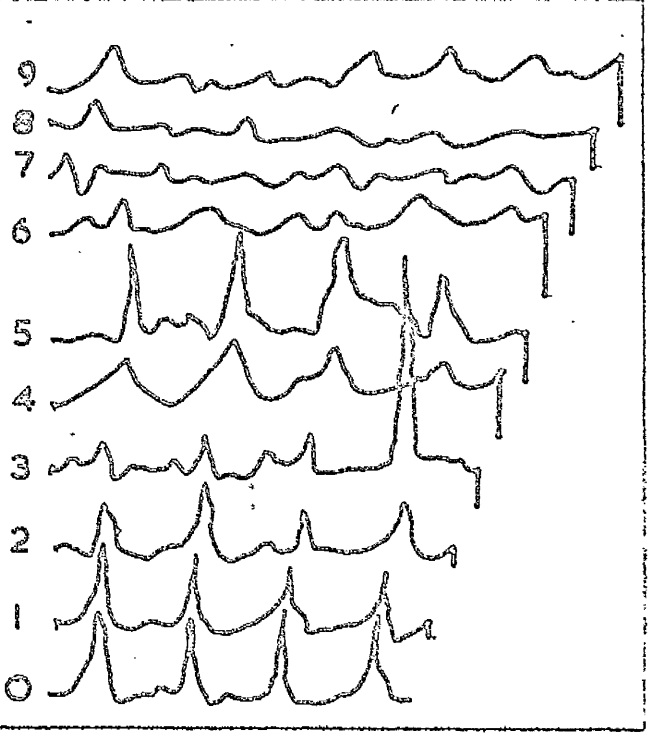
Pressure Tracing — 20 sec



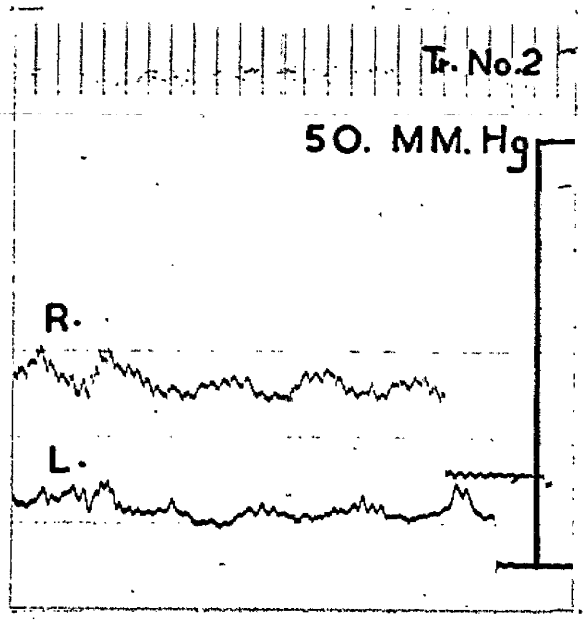
No.5 4.11.60 Left Kidney Osmotic Diuresis

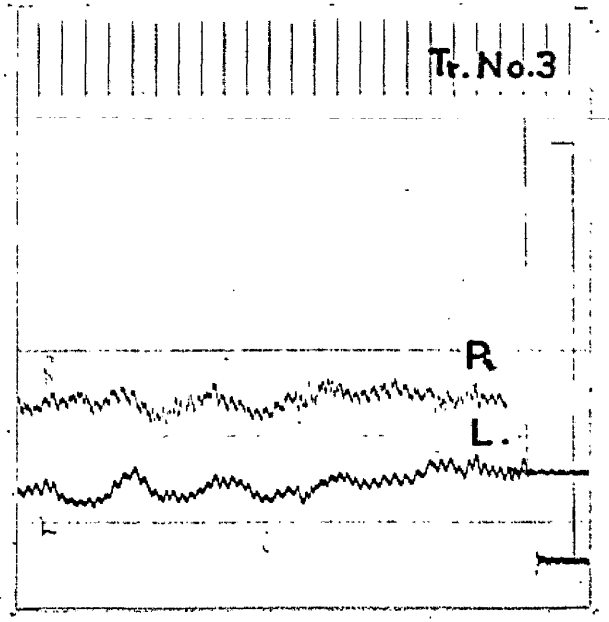
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 11 (0)
5	11	0 - 12 (1)
10	13	1 - 10 (2)
15	15	4 - 30 (3)
20	24	5 - 11 (4)
25	33	5 - 18 (5)
30	38	8 - 12 (6)
35	50	5 - 10 (7)
40	45	3 - 9 (8)
45	30	5 - 10 (9)

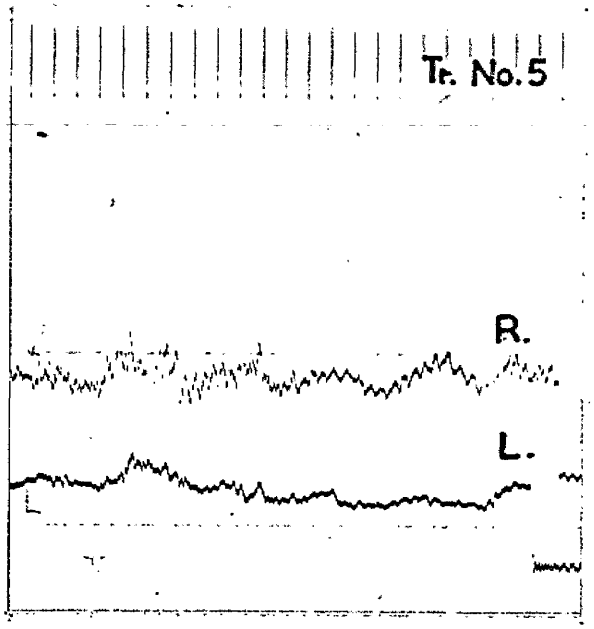
Pressure Tracing — 20 sec



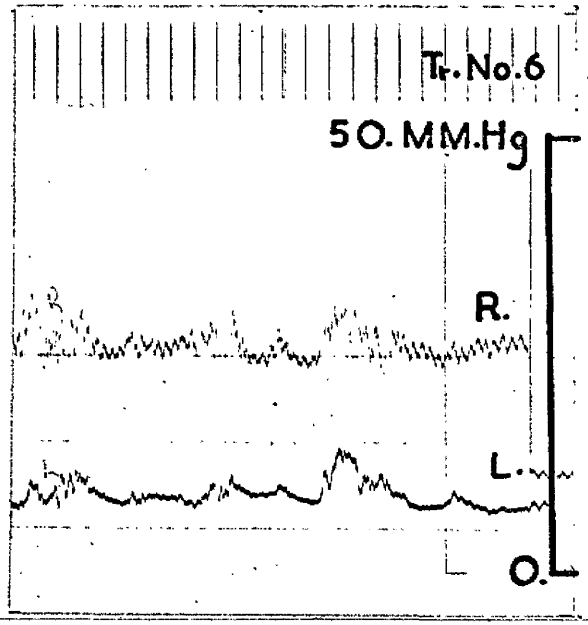
No.5 11/11/60

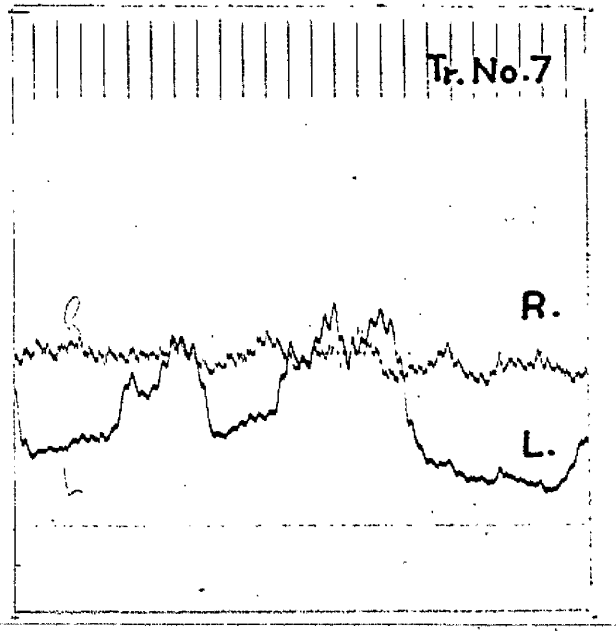


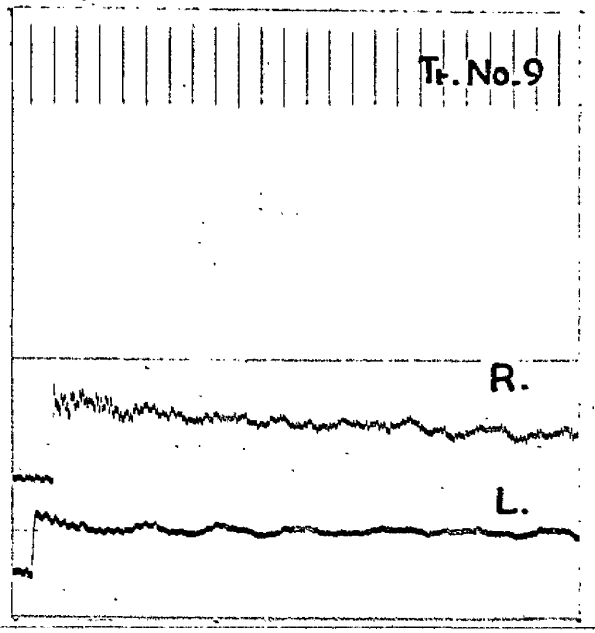




No.5 11/11/60



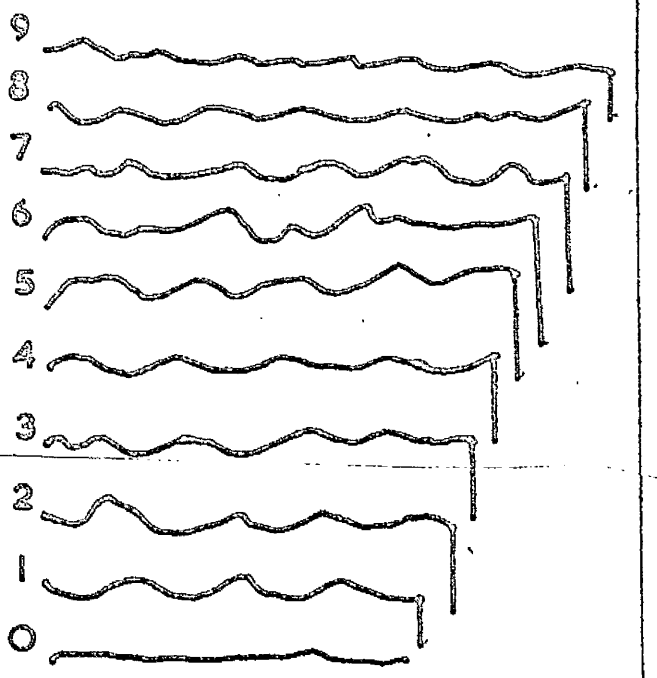




No.5 11.11.60 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	-	0 - 1 (0)
5	4	5 - 10 (1)
10	6	9 - 14 (2)
15	7	8 - 11 (3)
20	8	9 - 11 (4)
25	30	9 - 14 (5)
30	29	13 - 17 (6)
35	29	13 - 16 (7)
40	60	9 - 11 (8)
45	10	5 - 11 (9)

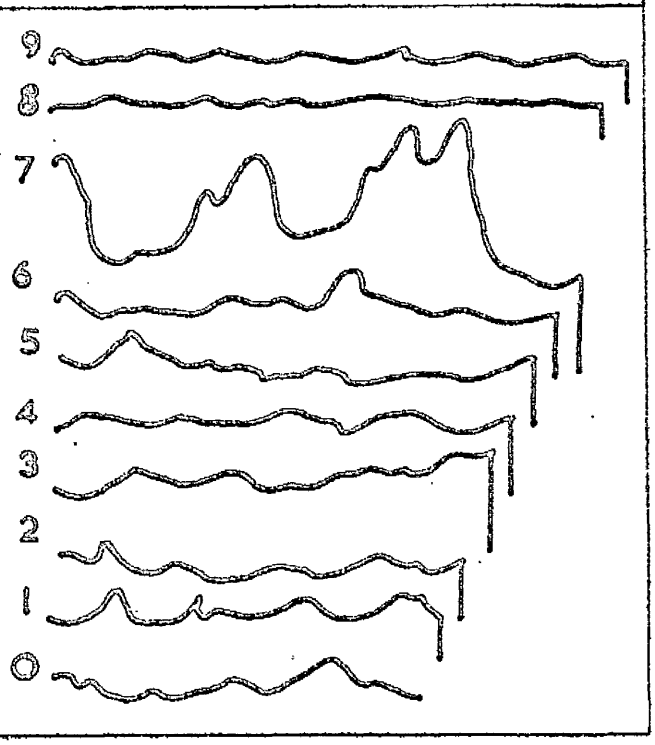
Pressure Tracing — 20 sec



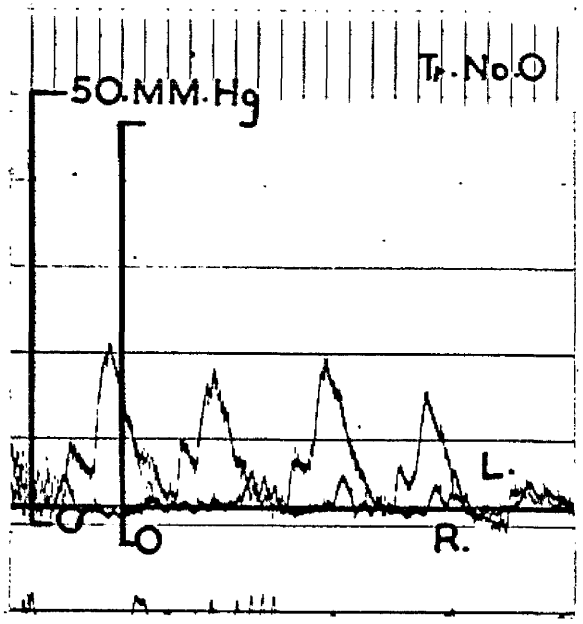
No.5 11.11.60 Left Kidney Osmotic Diuresis

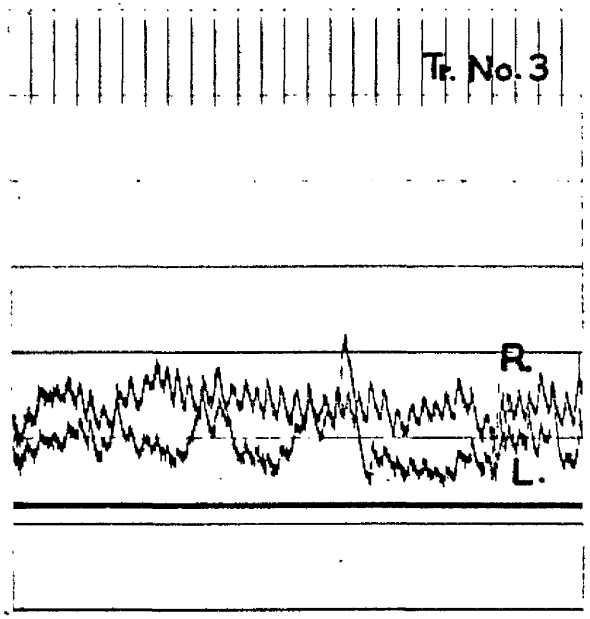
Time (Mins)	Urine/5 min R. & L.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	-	0 - 5 (0)
5	4	5 - 8 (1)
10	6	5 - 10 (2)
15	7	7 - 12 (3)
20	8	7 - 11 (4)
25	30	5 - 12 (5)
30	29	7 - 13 (6)
35	29	10 - 30 (7)
40	60	4 - 6 (8)
45	10	5 - 8 (9)

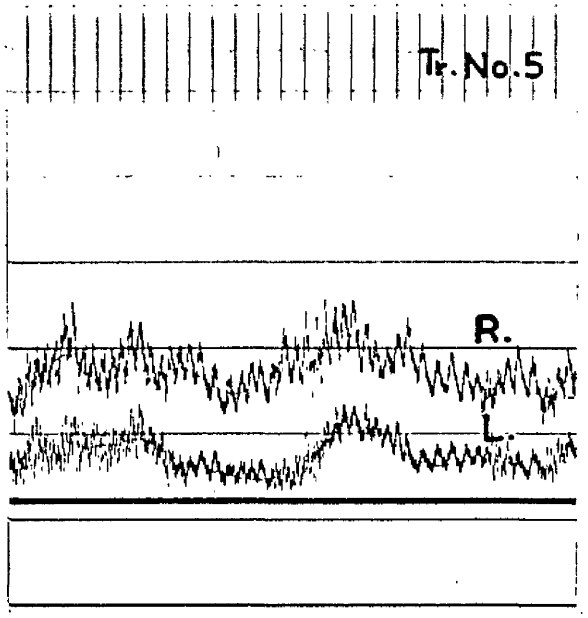
Pressure Tracing — 20 sec



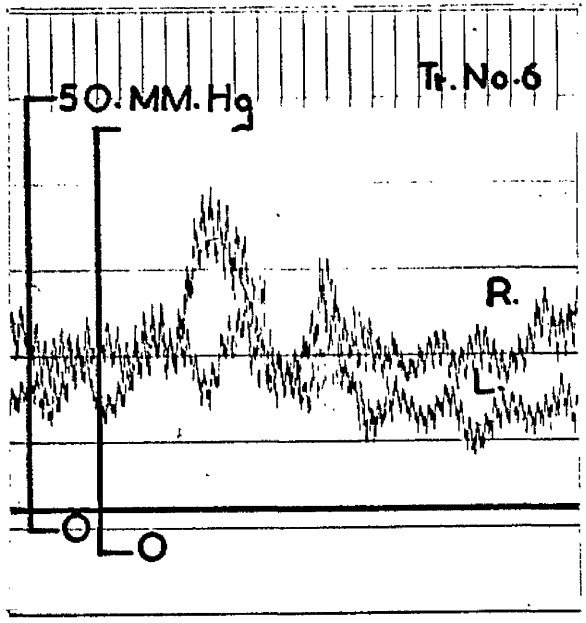
No. 5 22/11/60

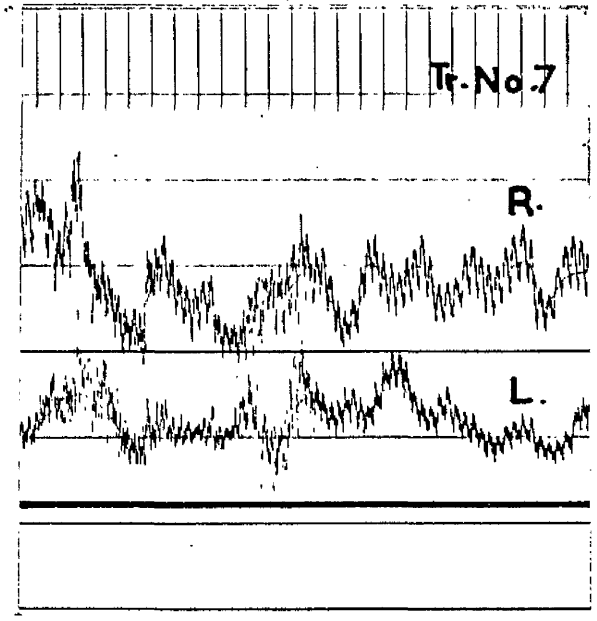


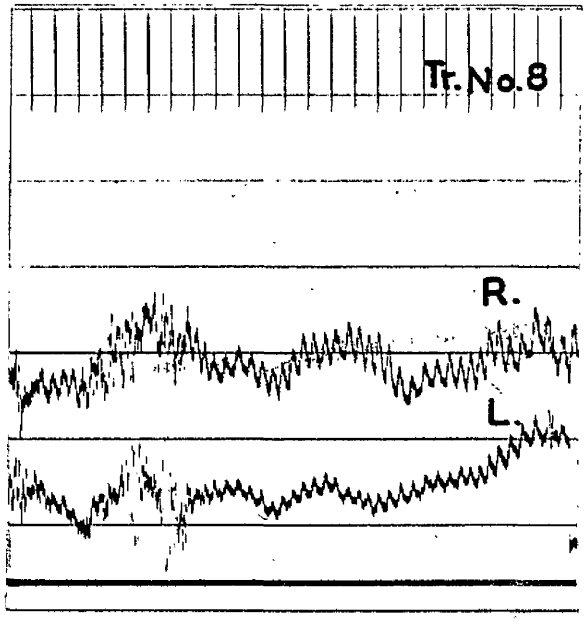




No. 5 22/11/60



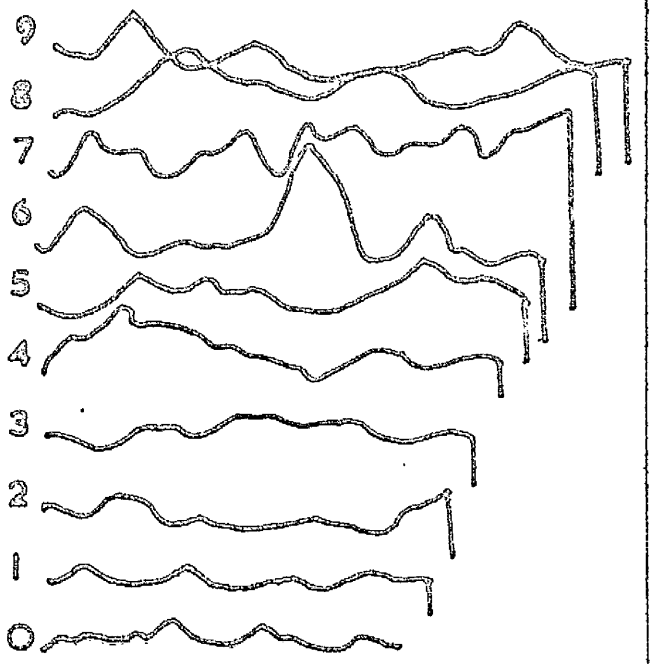




No.5 22.11.60 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 4 (0)
5	1	4 - 7 (1)
10	4	3 - 7 (2)
15	23	5 - 9 (3)
20	23	3 - 12 (4)
25	41	6 - 12 (5)
30	44	10 - 24 (6)
35	56	16 - 24 (7)
40	85	8 - 15 (8)
45	27	11 - 18 (9)

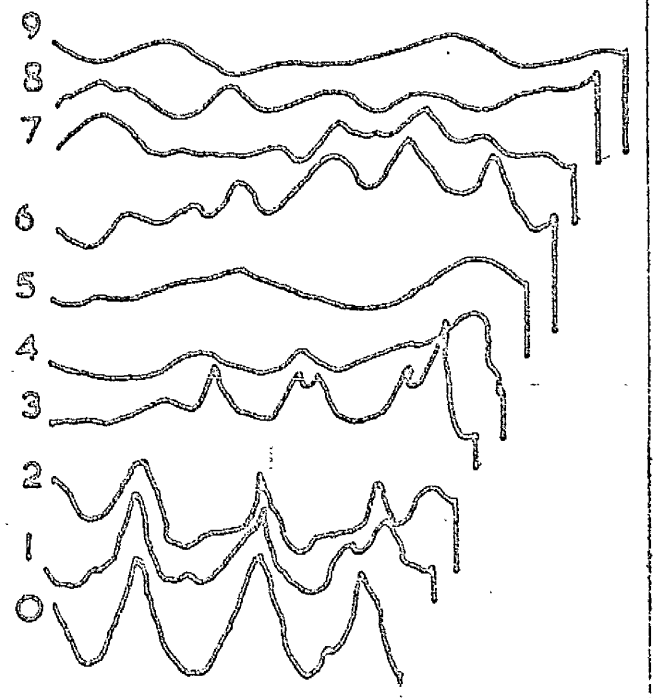
Pressure Tracing
20 sec



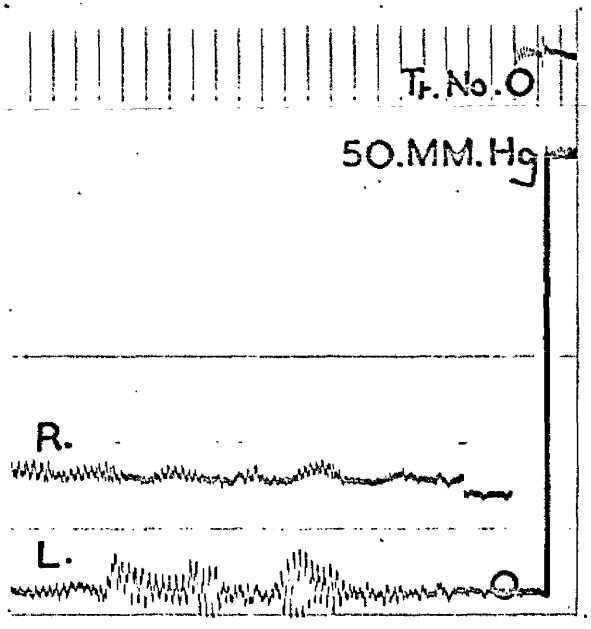
No.5 22.11.60 Left Kidney Osmotic Diuresis

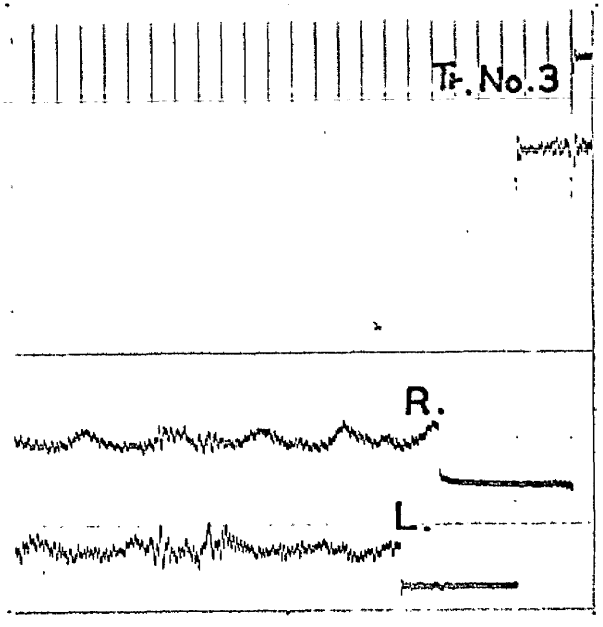
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 15 (0)
5	1	1 - 13 (1)
10	4	2 - 13 (2)
15	23	5 - 17 (3)
20	23	5 - 15 (4)
25	41	5 - 12 (5)
30	44	11 - 24 (6)
35	56	7 - 14 (7)
40	85	6 - 11 (8)
45	27	10 - 15 (9)

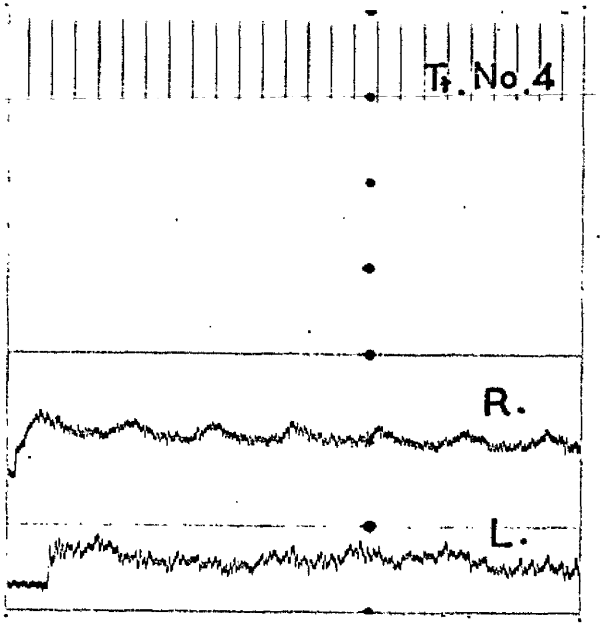
Pressure Tracing
20 sec



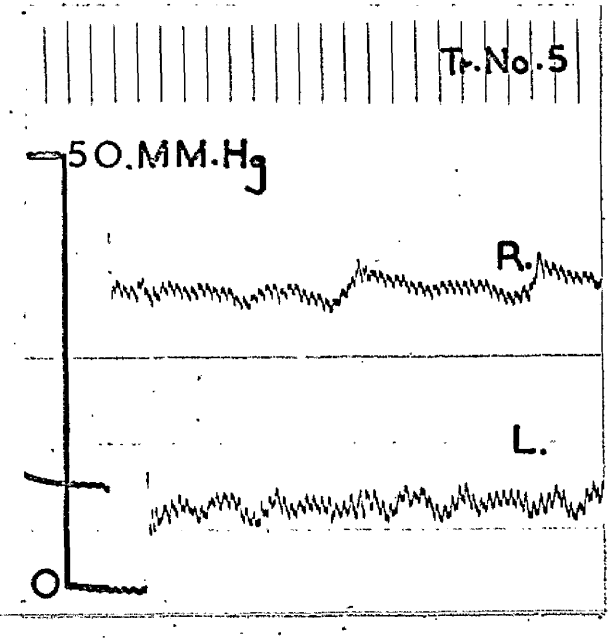
No.6 12/11/60

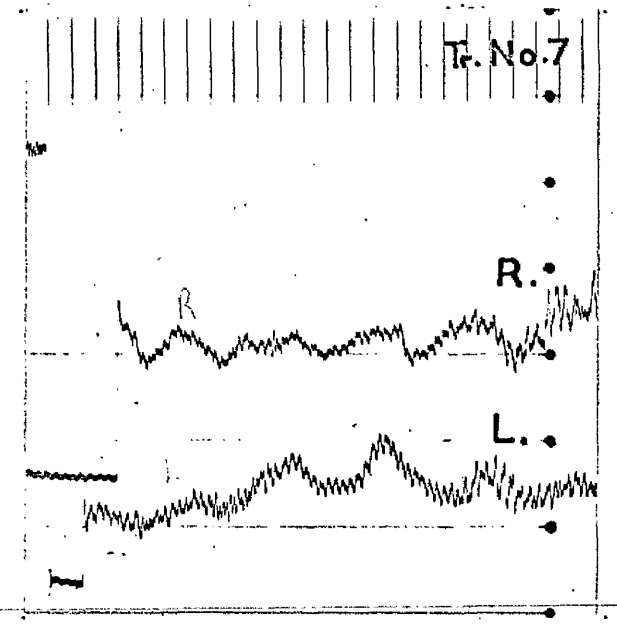


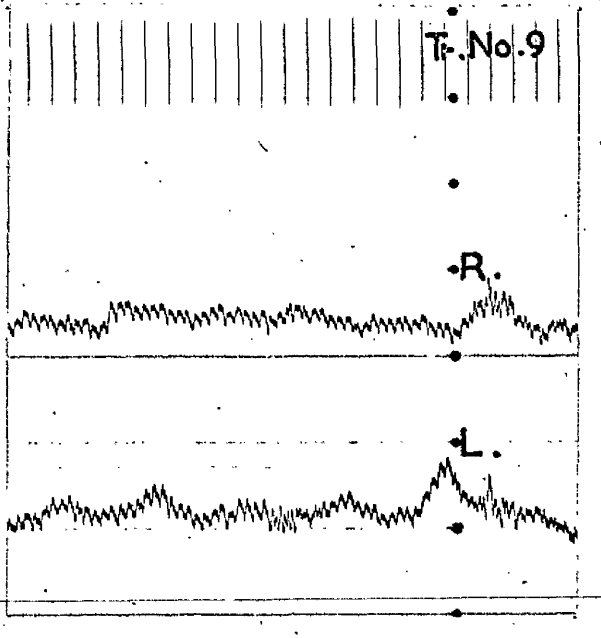




No.6 12/11/60



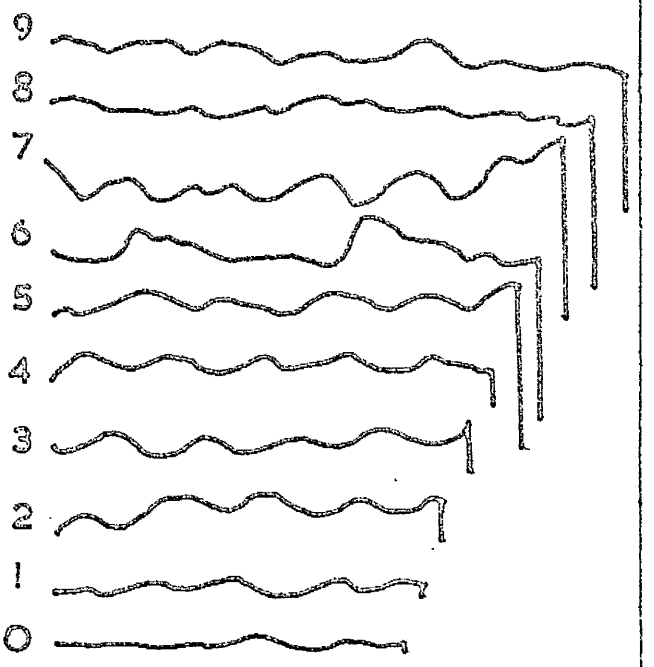




No.6 12.11.60 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	3	1 - 2 (1)
10	6	3 - 7 (2)
15	37	4 - 6 (3)
20	35	5 - 7 (4)
25	60	16 - 20 (5)
30	74	18 - 24 (6)
35	75	14 - 21 (7)
40	105	20 - 23 (8)
45	71	15 - 21 (9)

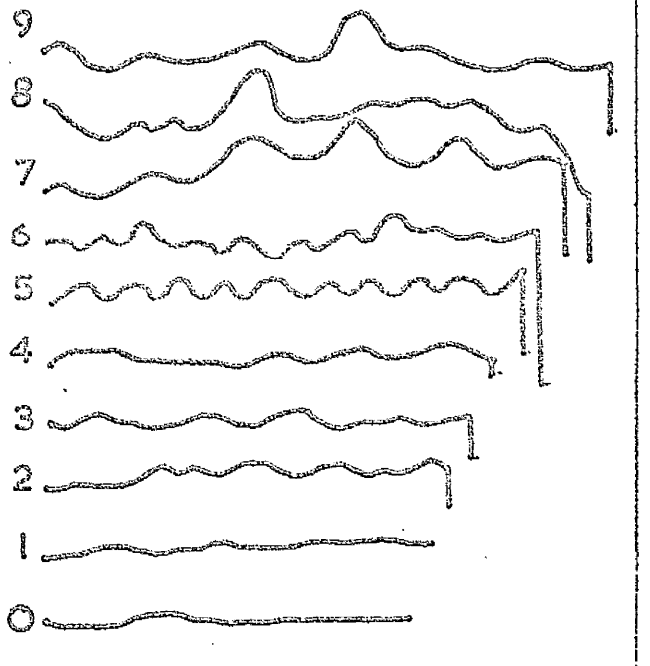
Pressure Tracing — 20 sec



No.6 12.11.60 Left Kidney Osmotic Diuresis

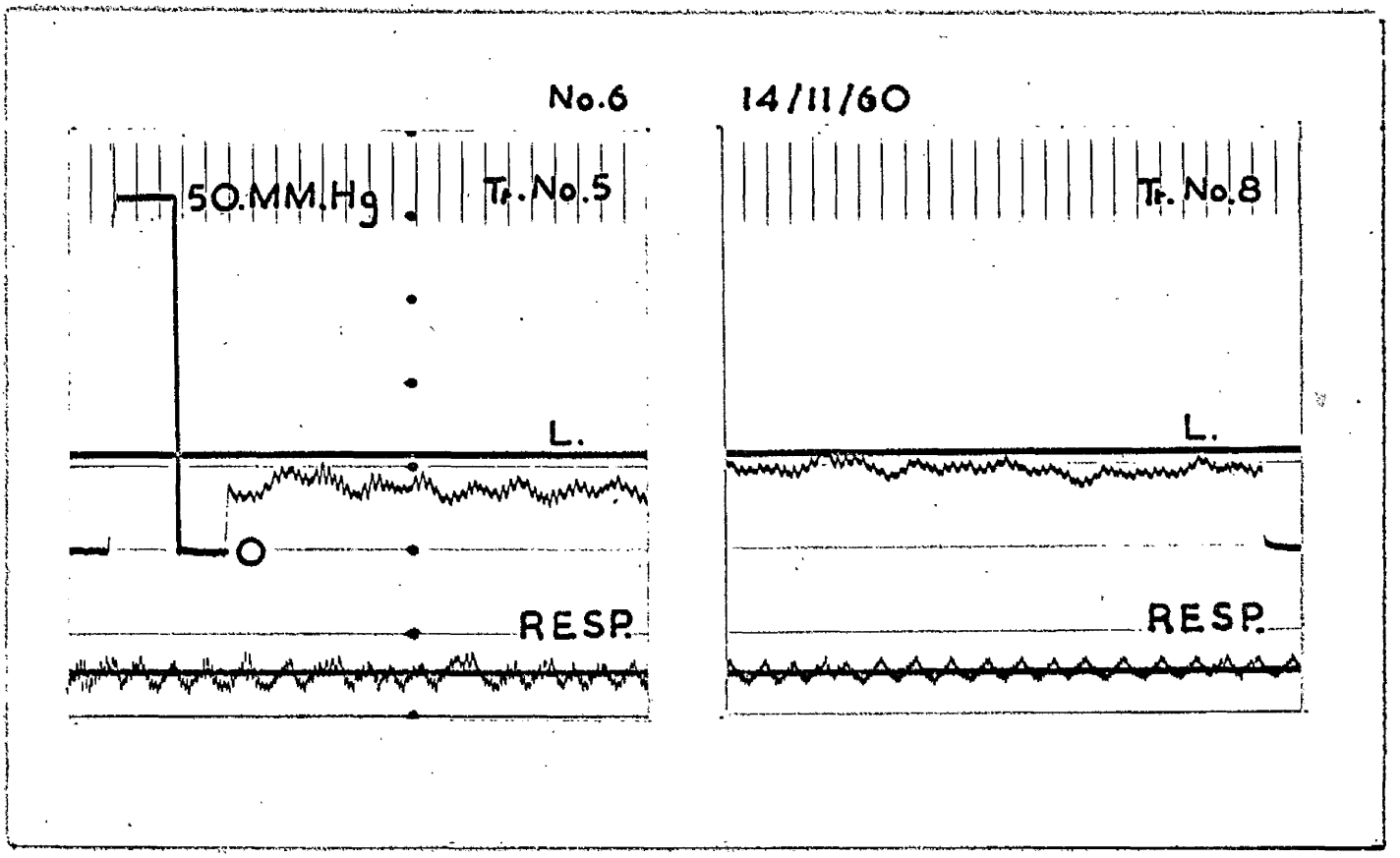
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	3	0 - 1 (1)
10	6	3 - 6 (2)
15	37	4 - 6 (3)
20	35	2 - 5 (4)
25	60	7 - 10 (5)
30	74	16 - 21 (6)
35	75	8 - 16 (7)
40	105	8 - 24 (8)
45	71	7 - 15 (9)

Pressure Tracing — 20 sec

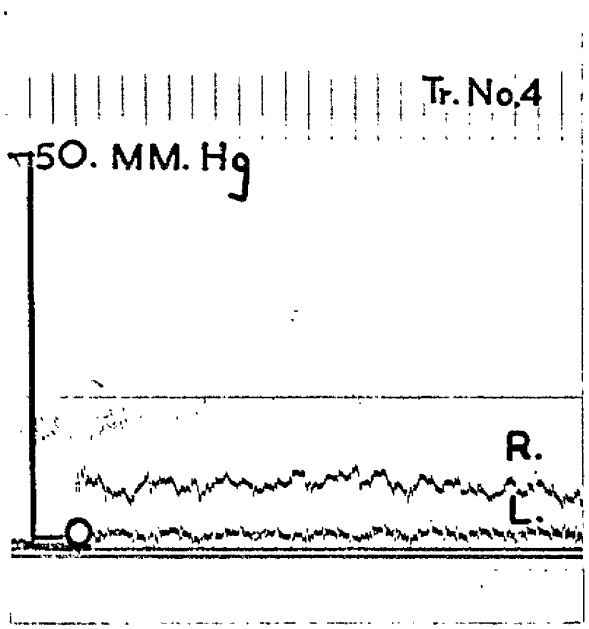


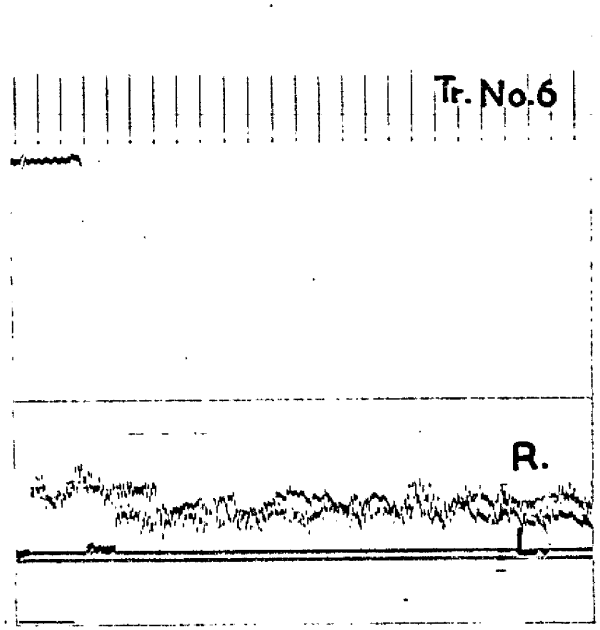
No.6 14.11.60 Left Kidney Osmotic Diuresis

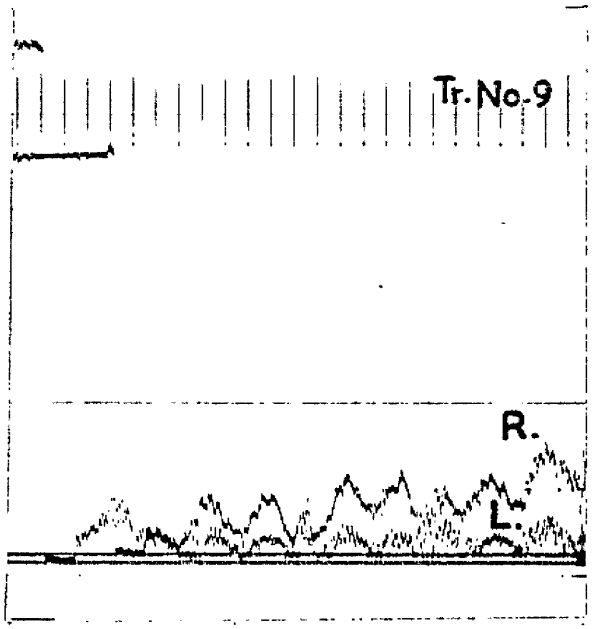
Time (Mins)	Urine/5 min L & R Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec
Basal	- mls	0 (0)	
5	5	0 (1)	
10	7	0 - 3 (2)	
15	14	0 - 1 (3)	
20	27	2 - 4 (4)	
25	39	6 - 10 (5)	
30	48	5 - 10 (6)	
35	43	5 - 10 (7)	
40	69	7 - 11 (8)	
45	52	5 - 8 (9)	



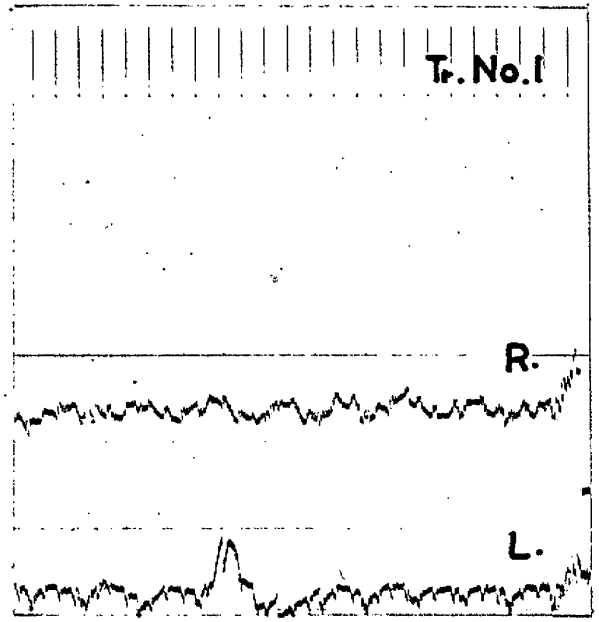
No.7 2/12/60

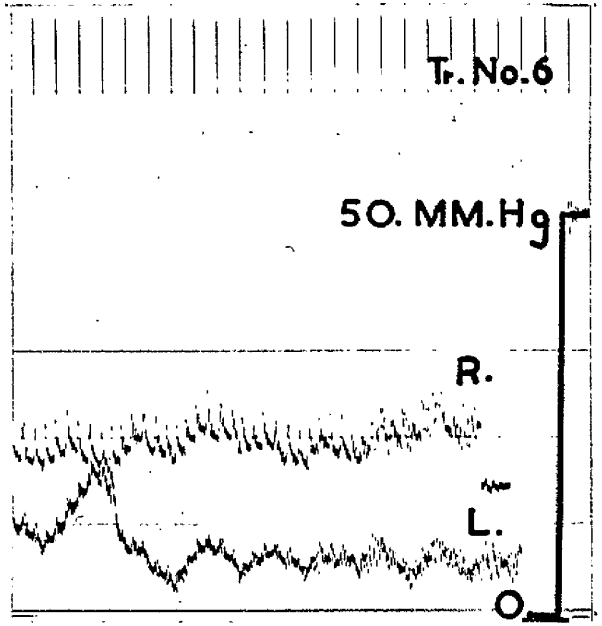


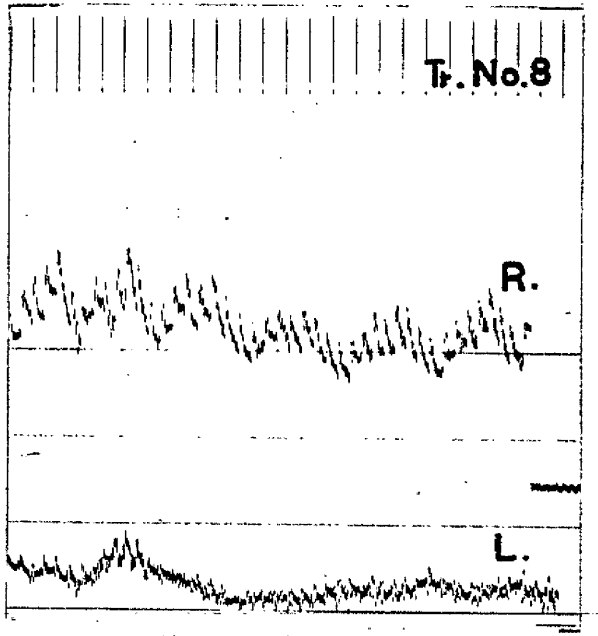




No.7 9/12/60

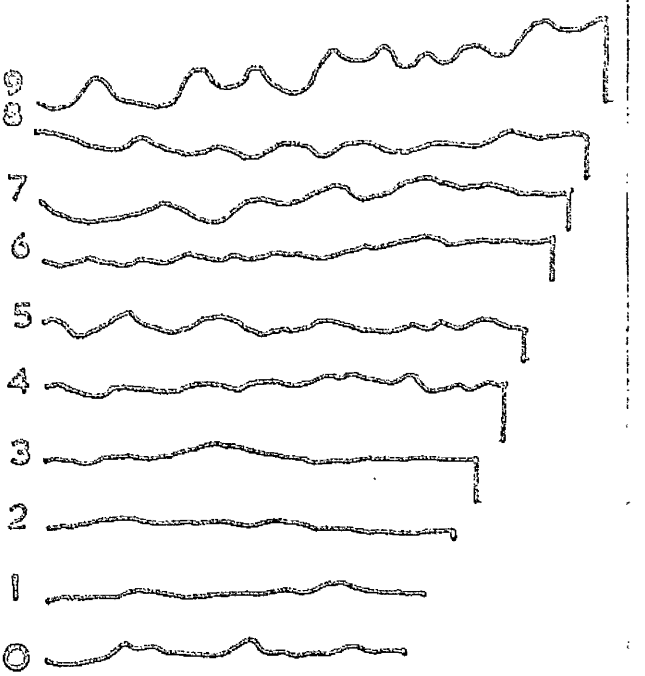






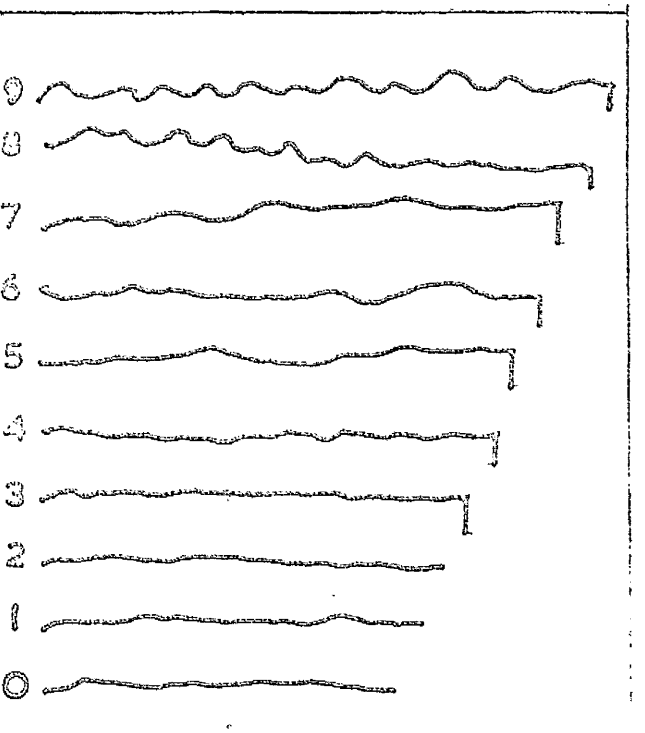
No.7 2.12.60 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 - 2 (0)	
5	4	0 - 1 (1)	
10	8	1 - 2 (2)	
15	13	4 - 7 (3)	
20	23	6 - 8 (4)	
25	31	3 - 5 (5)	
30	35	4 - 6 (6)	
35	40	1 - 7 (7)	
40	45	3 - 7 (8)	
45	42	0 - 10 (9)	



No.7 2.12.60 Left Kidney Osmotic Diuresis

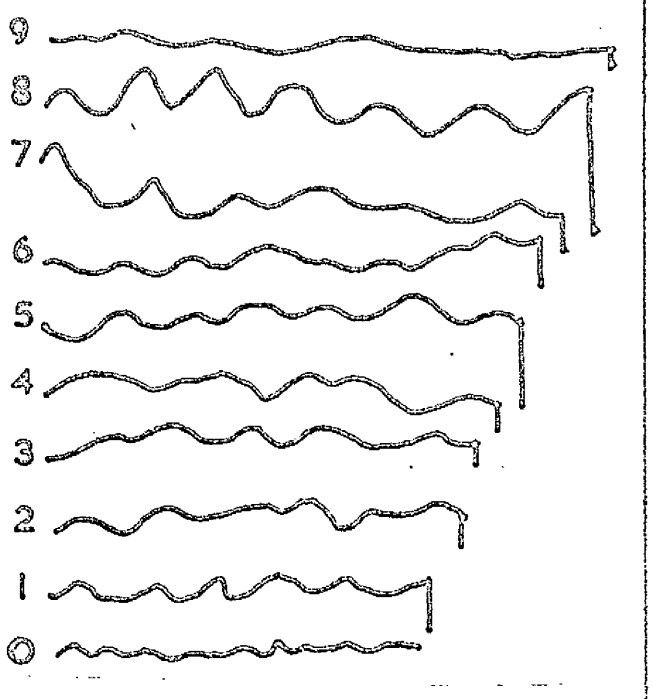
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 - 1 (0)	
5	4	0 - 1 (1)	
10	8	0 - 1 (2)	
15	13	5 (3)	
20	23	3 - 4 (4)	
25	31	4 - 5 (5)	
30	35	3 - 6 (6)	
35	40	3 - 6 (7)	
40	45	2 - 7 (8)	
45	42	2 - 4 (9)	



No.7 9.12.60 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	2	4 - 7 (1)
10	16	2 - 6 (2)
15	39	2 - 6 (3)
20	32	2 - 7 (4)
25	28	9 - 14 (5)
30	42	2 - 7 (6)
35	45	4 - 14 (7)
40	83	13 - 21 (8)
45	49	2 - 5 (9)

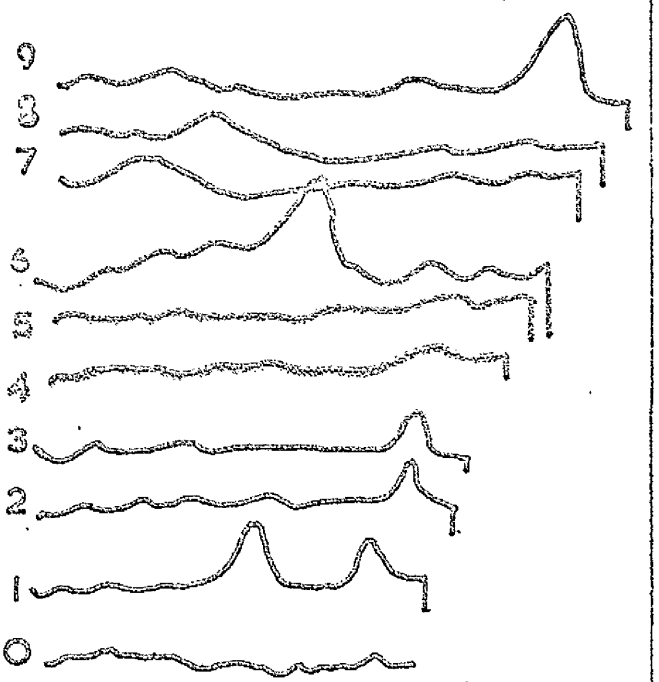
Pressure Tracing ——— 20 sec ———



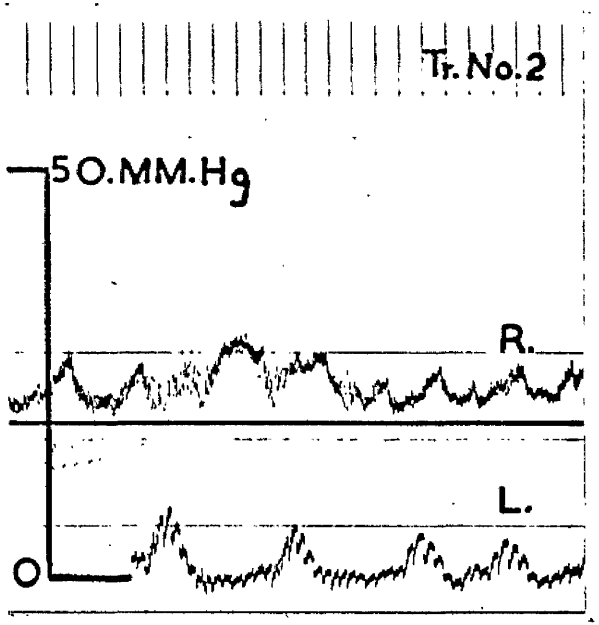
No.7 9.12.60 Left Kidney Osmotic Diuresis

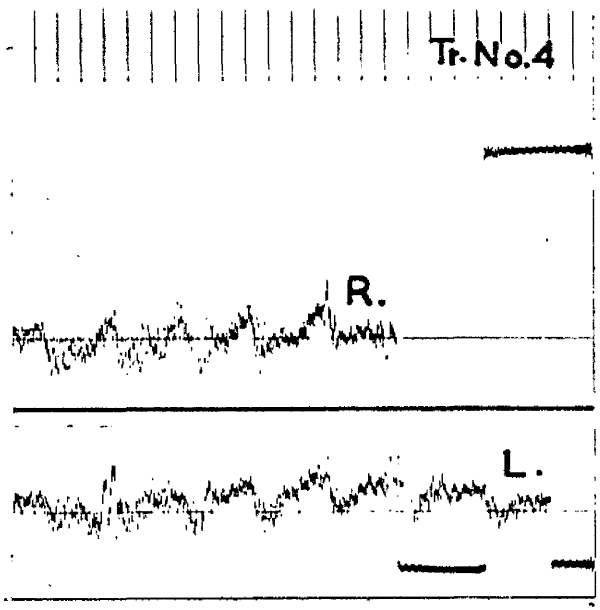
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 3 (0)
5	2	2 - 11 (1)
10	16	2 - 9 (2)
15	39	0 - 6 (3)
20	32	0 - 4 (4)
25	28	2 - 5 (5)
30	42	7 - 17 (6)
35	45	2 - 7 (7)
40	83	3 - 9 (8)
45	49	4 - 14 (9)

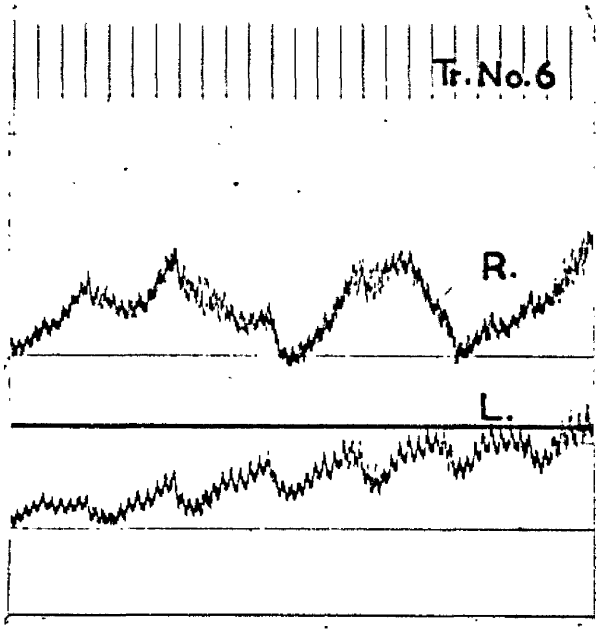
Pressure Tracing ——— 20 sec ———



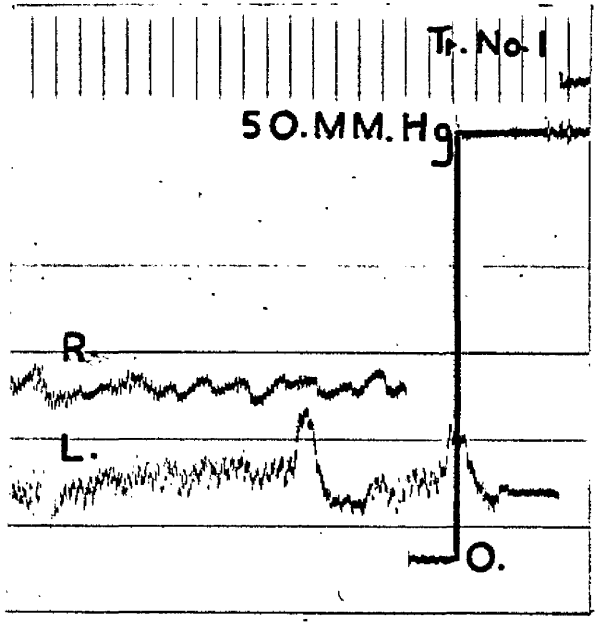
No.7 10/3/61

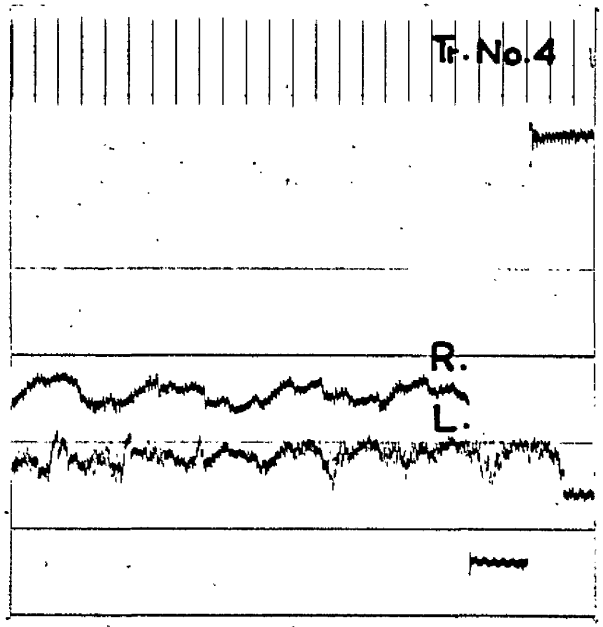


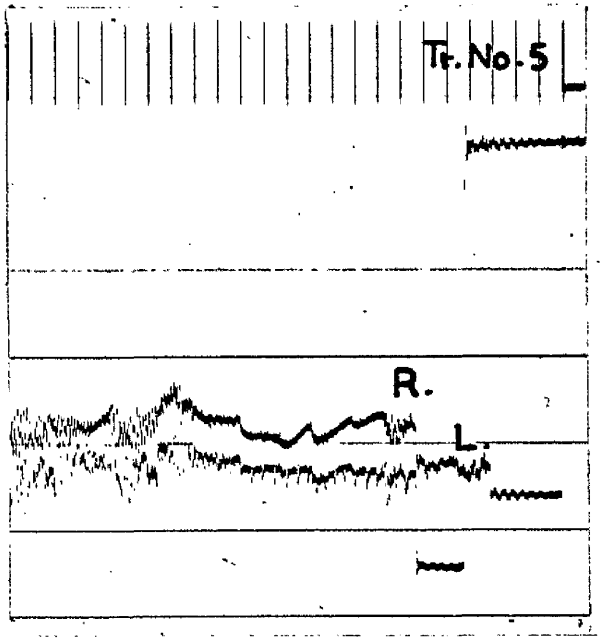




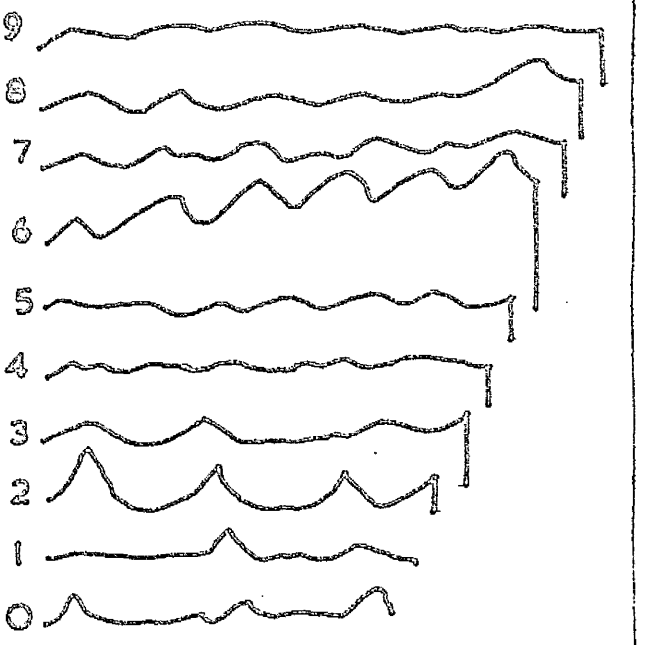
No.7 17/3/61



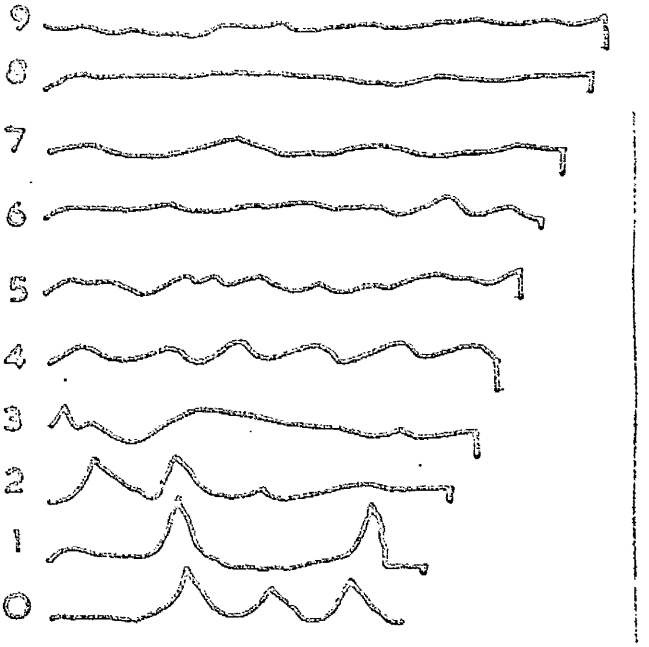




No.7 10.3.61 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 - 4 (0)	
5	2	0 - 5 (1)	
10	5	0 - 7 (2)	
15	10	5 - 10 (3)	
20	18	4 - 6 (4)	
25	28	4 - 6 (5)	
30	35	8 - 18 (6)	
35	74	4 - 8 (7)	
40	50	5 - 10 (8)	
45	46	5 - 8 (9)	

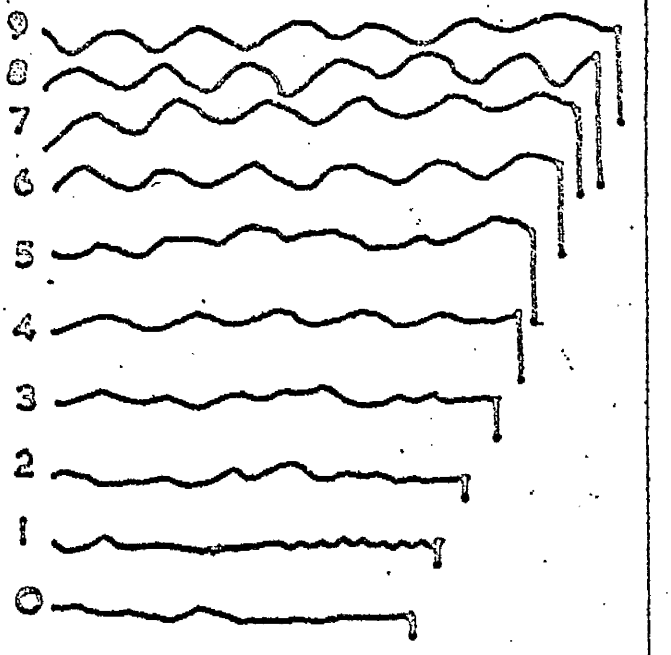
No.7 17.3.61 Left Kidney Osmotic Diuresis

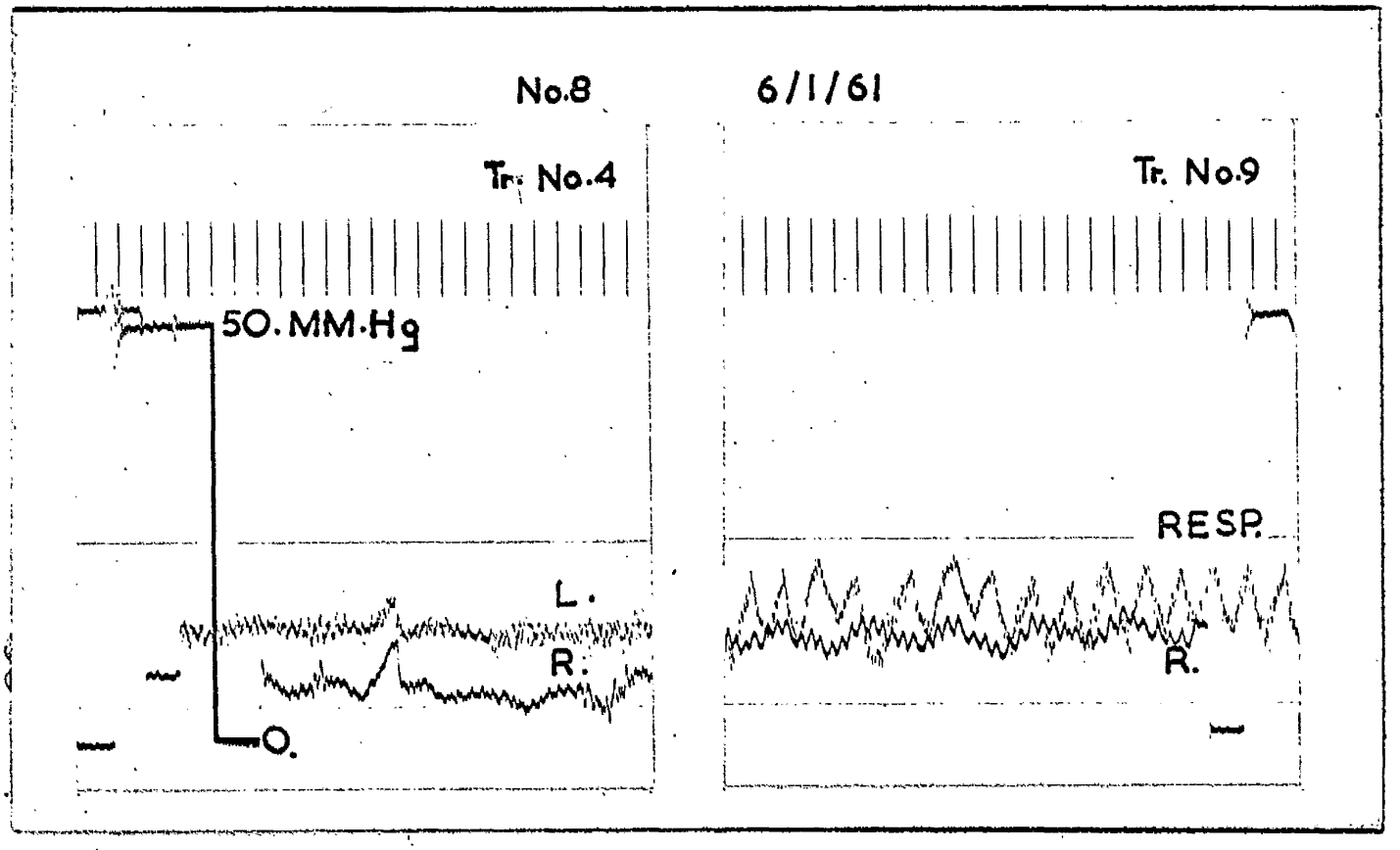
Time (Mins)	Urine/5 min L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 - 7 (0)	
5	6	0 - 10 (1)	
10	13	0 - 6 (2)	
15	15	2 - 7 (3)	
20	22	4 - 6 (4)	
25	26	2 - 5 (5)	
30	26	1 - 5 (6)	
35	42	2 - 5 (7)	
40	62	2 - 4 (8)	
45	57	3 - 5 (9)	

No.8 6.1.61 Right Kidney Osmotic Diuresis

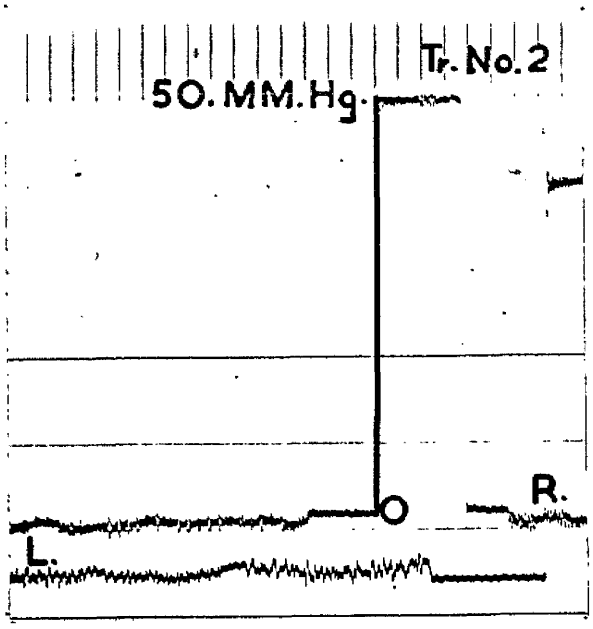
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	1 - 3 (0)
5	37	2 - 4 (1)
10.		2 - 5 (2)
15	38	4 - 7 (3)
20	65	7 - 9 (4)
25	60	9 - 13 (5)
30	75	9 - 12 (6)
35	108	9 - 13 (7)
40	60	12 - 17 (8)
45	74	10 - 14 (9)

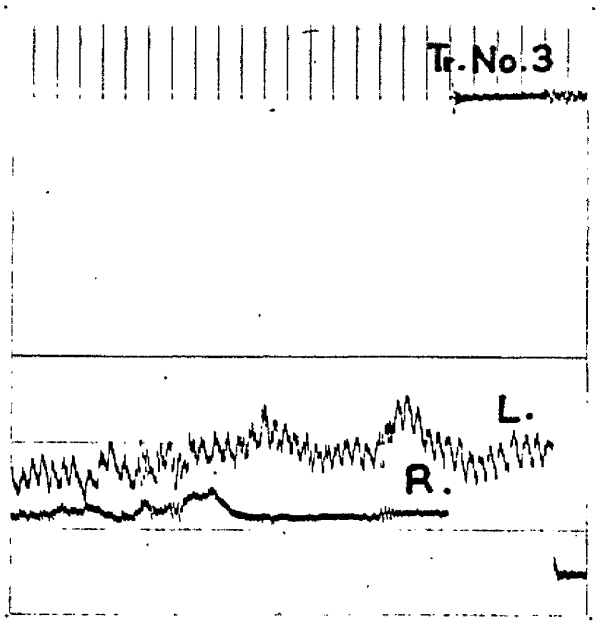
Pressure Tracing — 20 sec

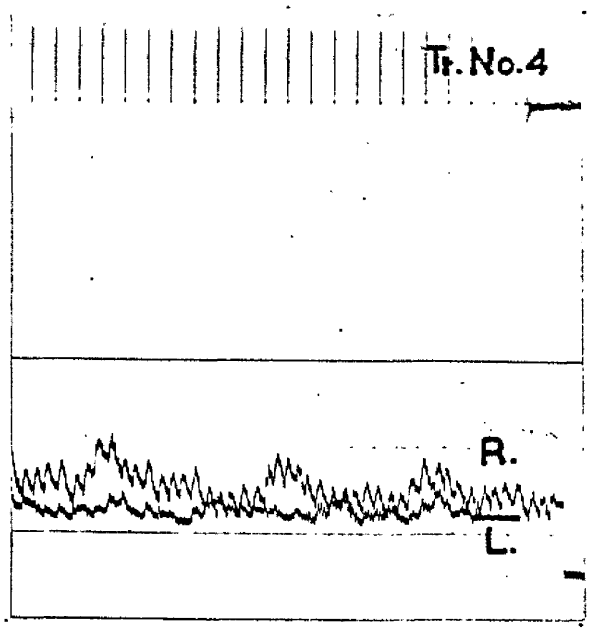




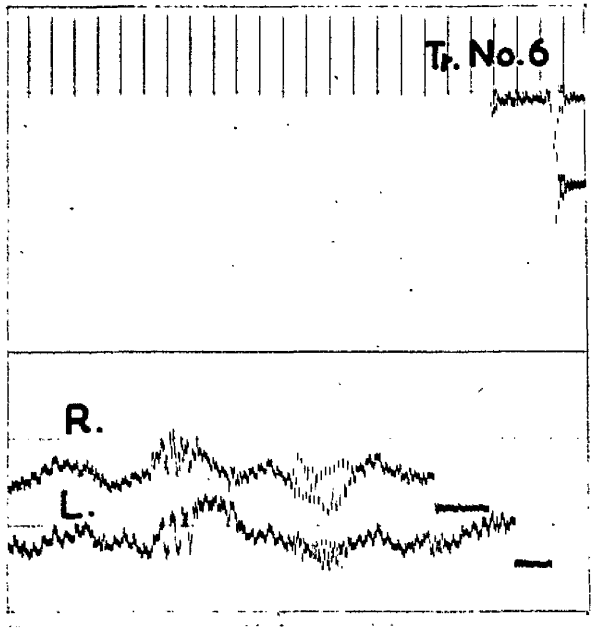
No.8 16/1/61

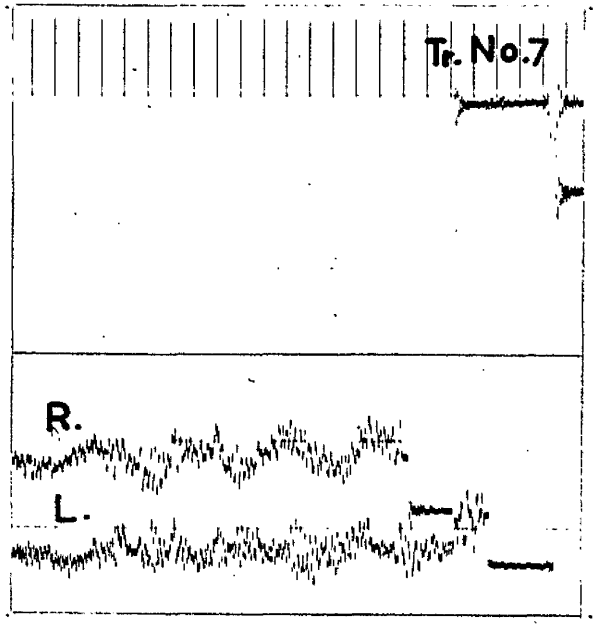


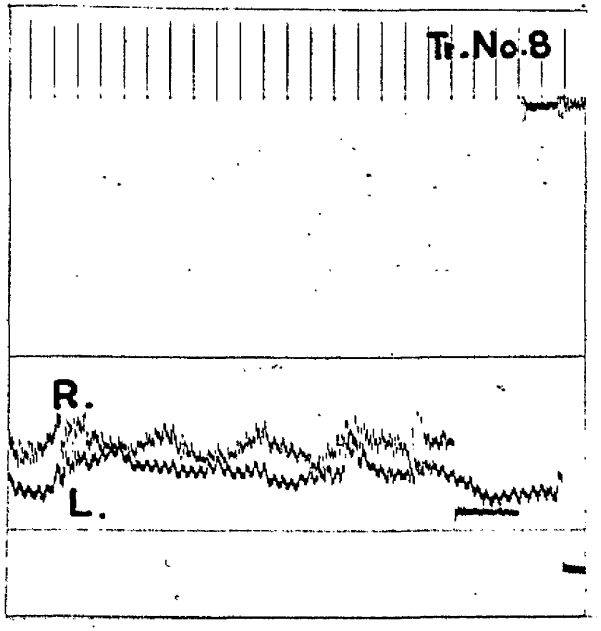




No. 8 16/1/61

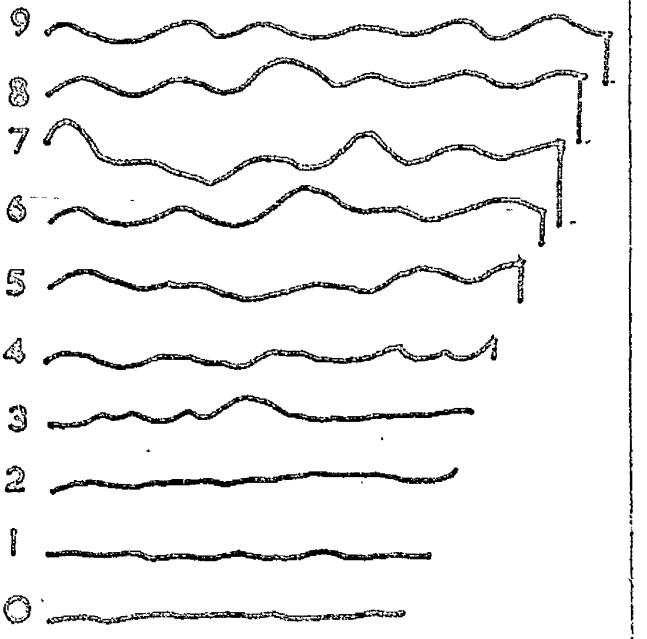






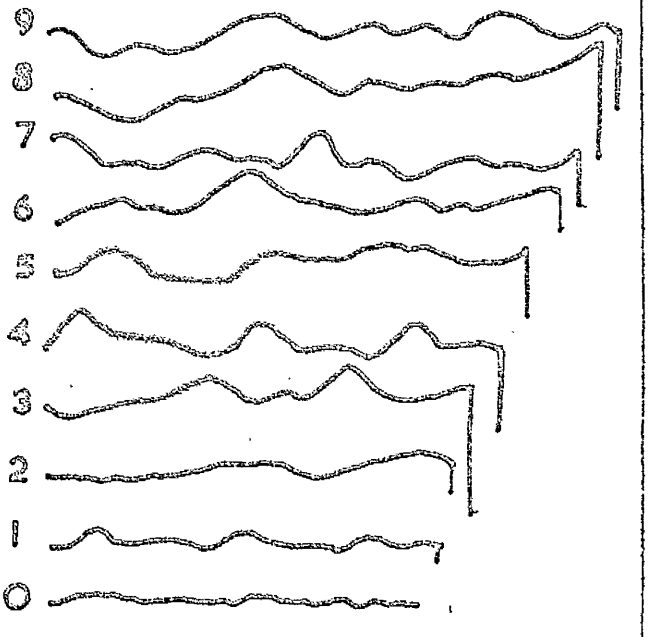
No.8 16.1.61 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/ 5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 (0)
5	11	0 (1)
10	20	0 (2)
15	39	0 - 4 (3)
20	37	0 - 3 (4)
25	70	1 - 5 (5)
30	67	3 - 7 (6)
35	80	6 - 13 (7)
40	100	6 - 10 (8)
45	54	6 - 9 (9)



No.8 16.1.61 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 (0)
5	11	0 - 3 (1)
10	20	1 - 4 (2)
15	39	11 - 18 (3)
20	37	8 - 14 (4)
25	70	5 - 9 (5)
30	67	2 - 7 (6)
35	80	4 - 9 (7)
40	100	5 - 13 (8)
45	54	7 - 11 (9)



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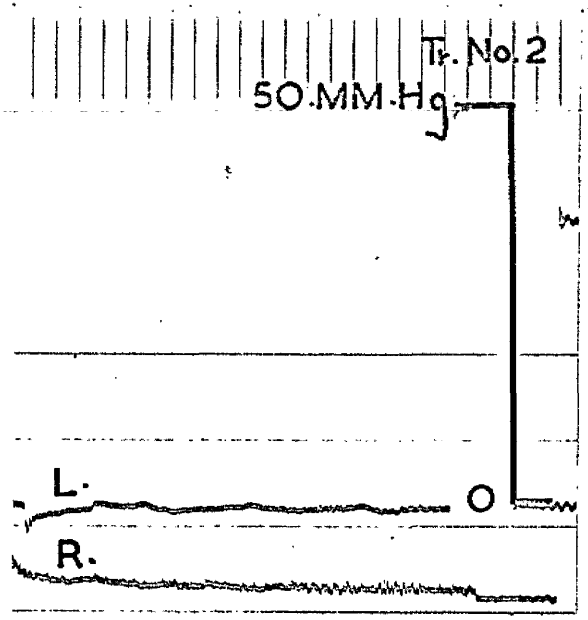
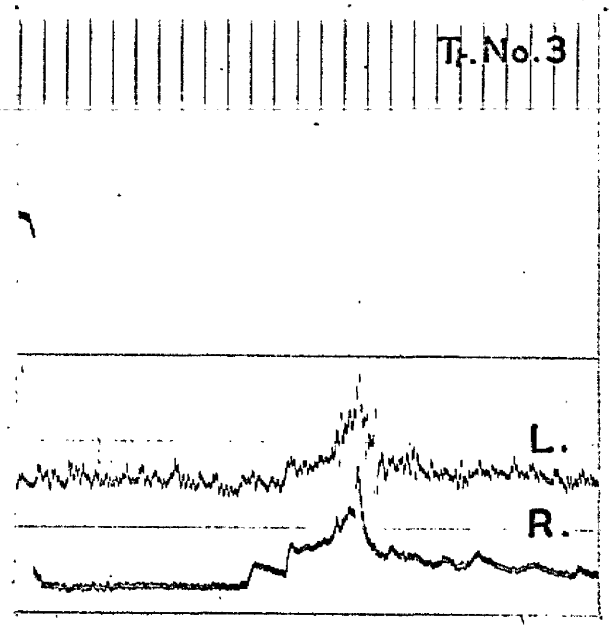
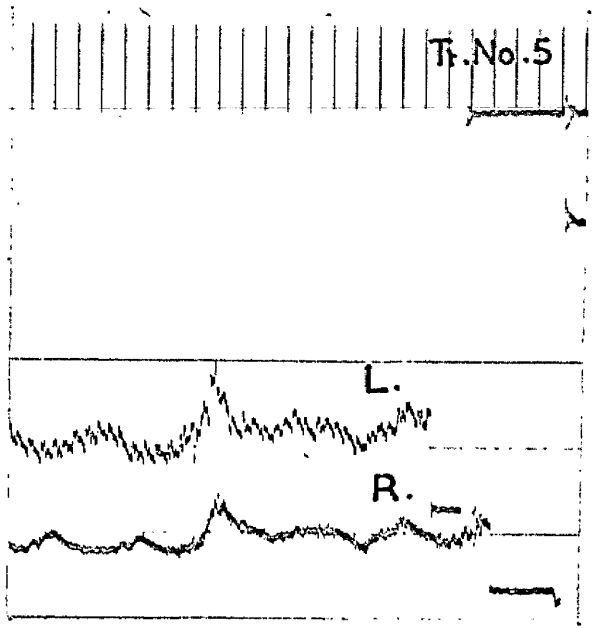
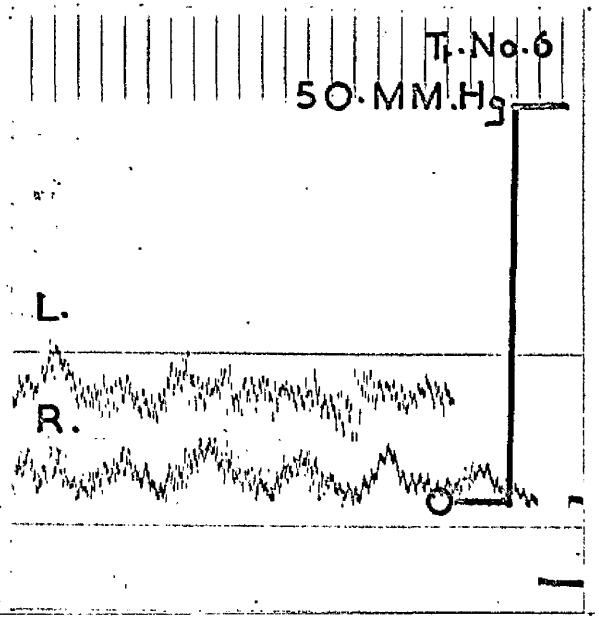
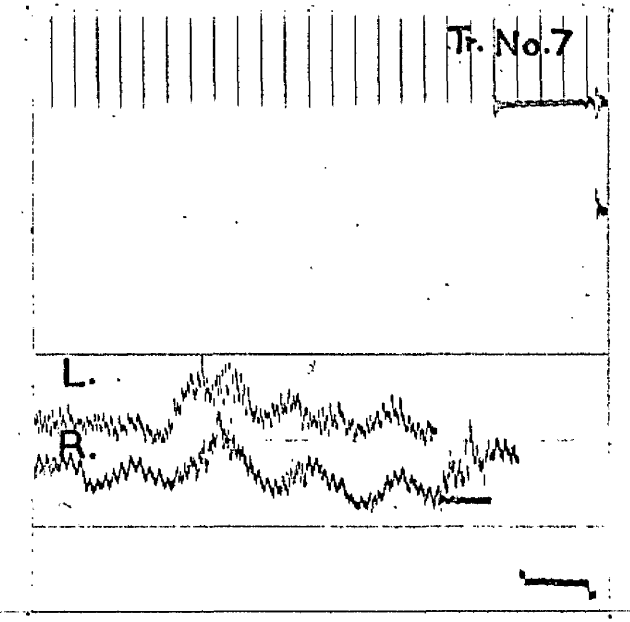


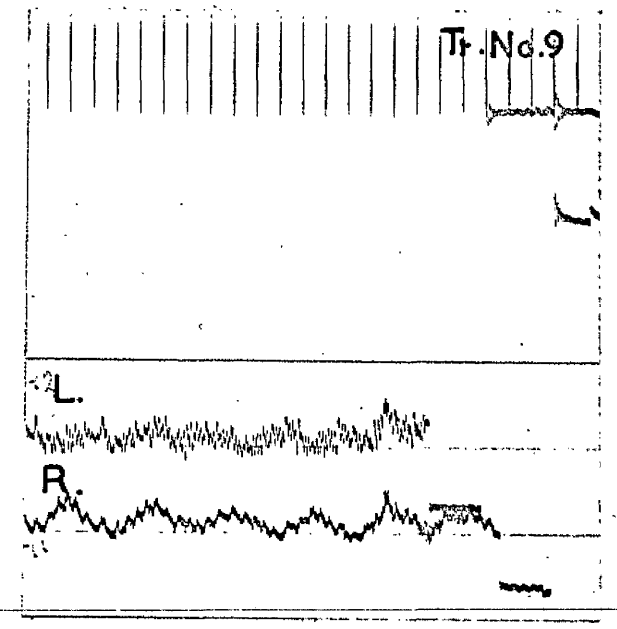




No 8 23/1/61

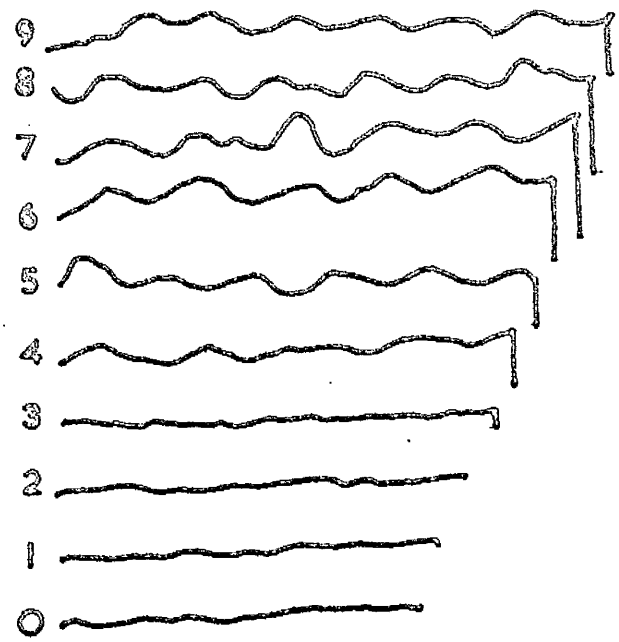






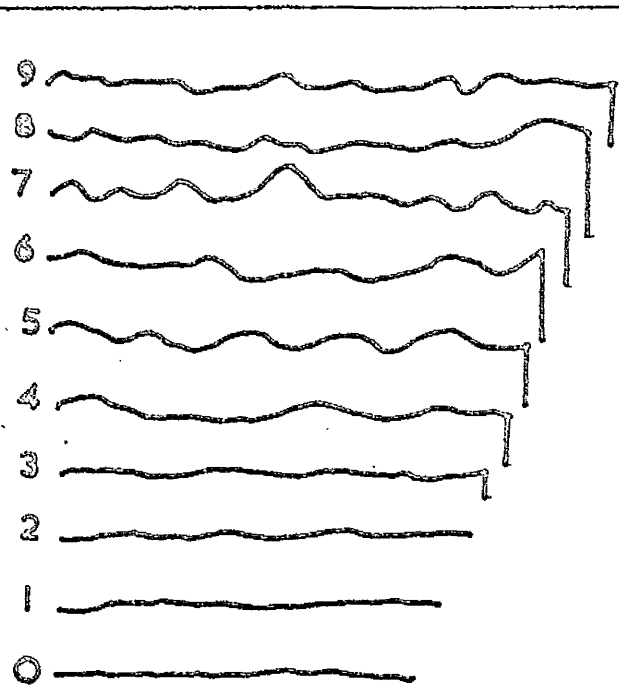
No.8 23.1.61 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 (0)	
5	14	0 (1)	
10	20	0 (2)	
15	39	2 - 3 (3)	
20	40	4 - 7 (4)	
25	75	6 - 11 (5)	
30	75	8 - 12 (6)	
35	64	11 - 16 (7)	
40	102	10 - 14 (8)	
45	80	5 - 9 (9)	

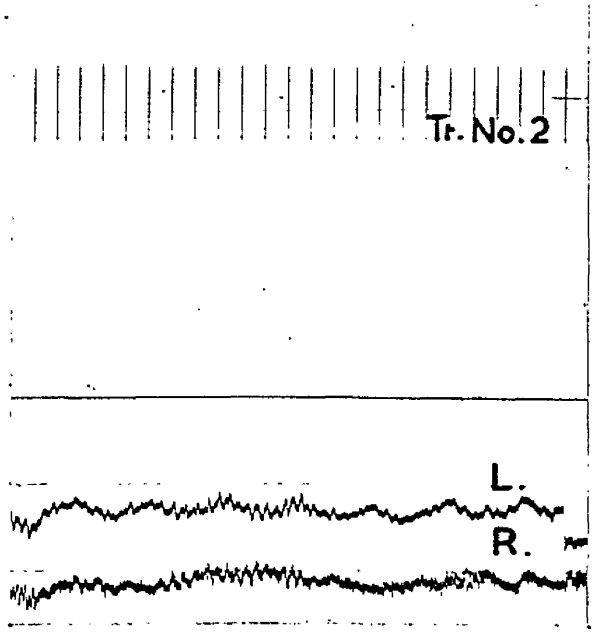


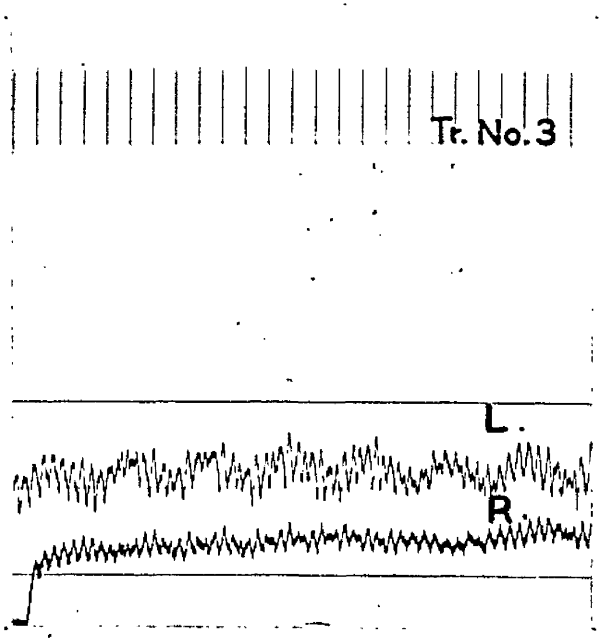
No.8 23.1.61 Left Kidney Osmotic Diuresis

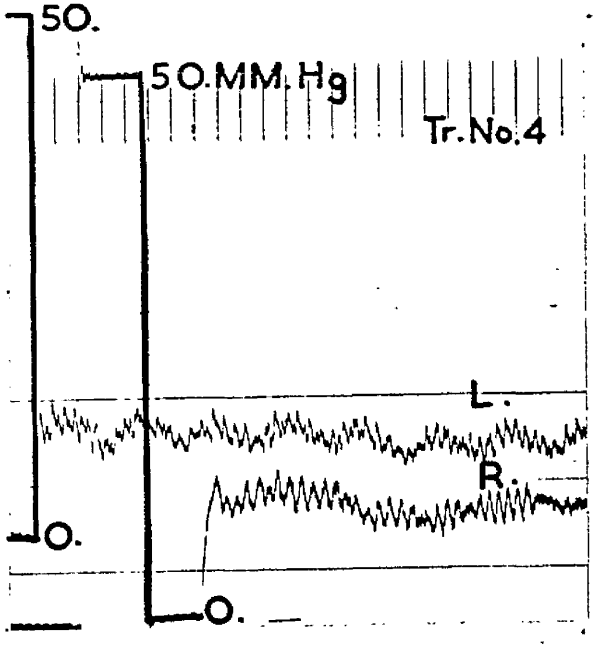
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 (0)	
5	14	0 (1)	
10	20	0 (2)	
15	39	3 - 5 (3)	
20	40	6 - 9 (4)	
25	75	6 - 9 (5)	
30	75	7 - 11 (6)	
35	64	9 - 14 (7)	
40	102	10 - 14 (8)	
45	80	7 - 9 (9)	



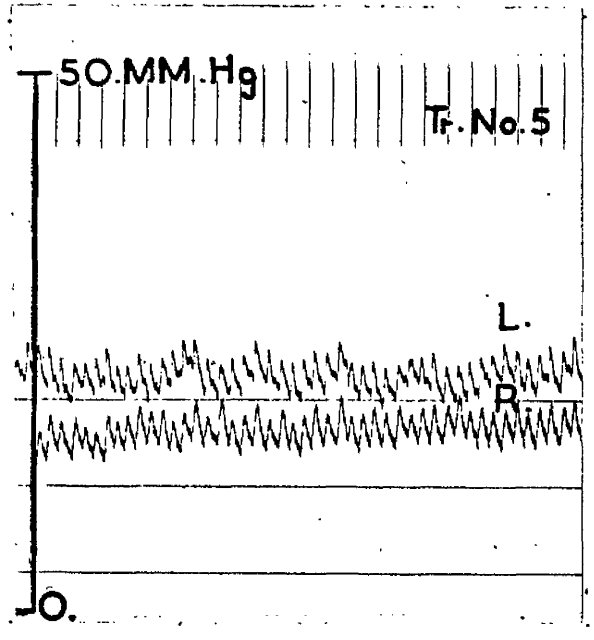
No 9 20/1/61

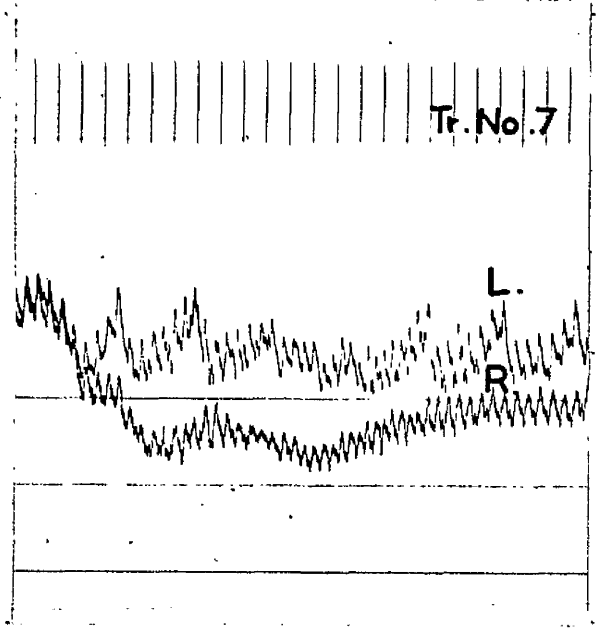


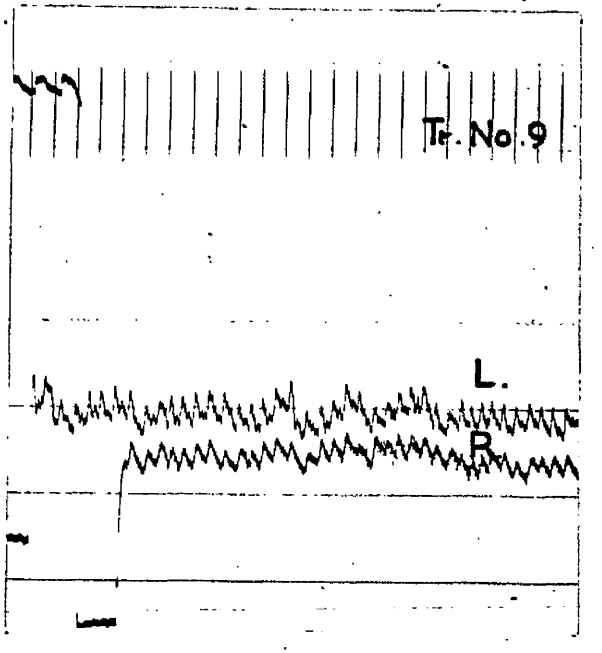




No 9 20/1/61

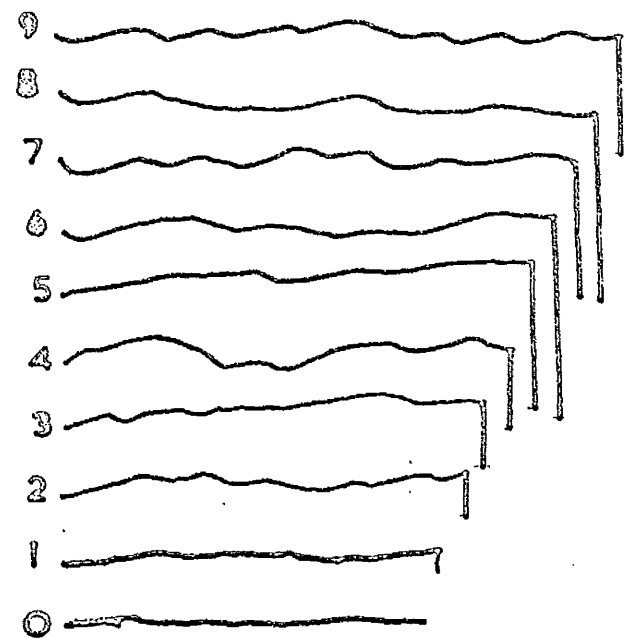






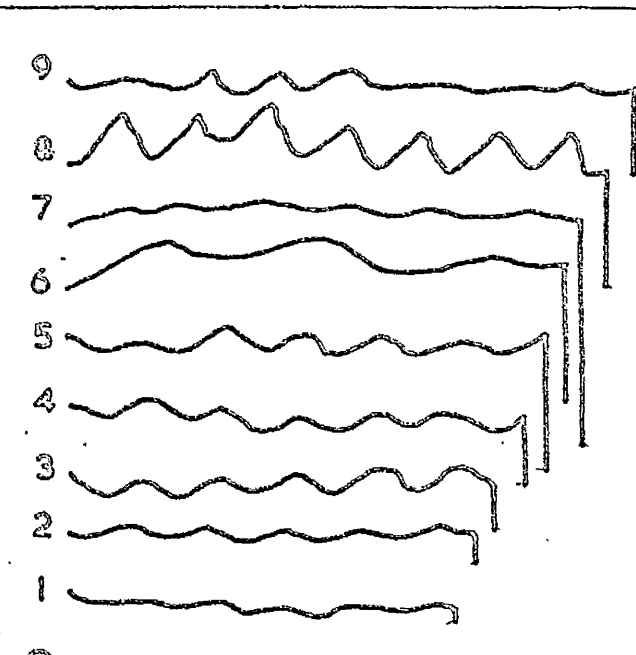
No.9 20.1.61 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec —
Basal	- mls	0 (0)	
5	15	3 (1)	
10	26	3 - 6 (2)	
15	20	6 - 9 (3)	
20	37	7 - 11 (4)	
25	50	15 - 18 (5)	
30	71	22 - 25 (6)	
35	50	16 - 19 (7)	
40	40	22 - 25 (8)	
45	40	13 - 16 (9)	

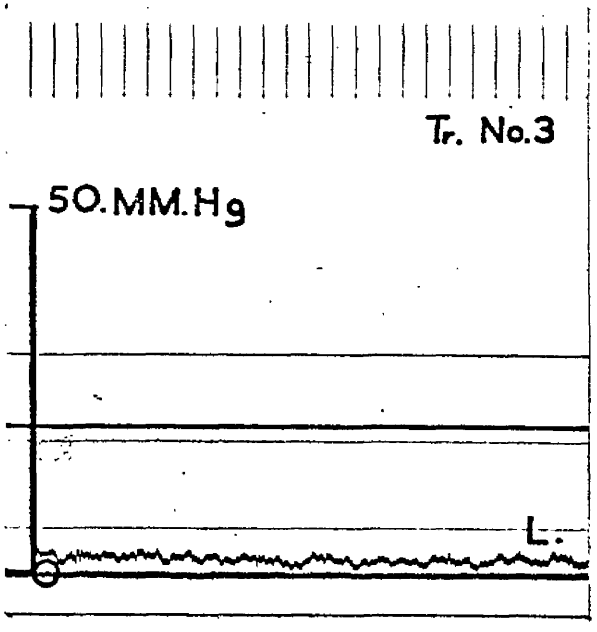


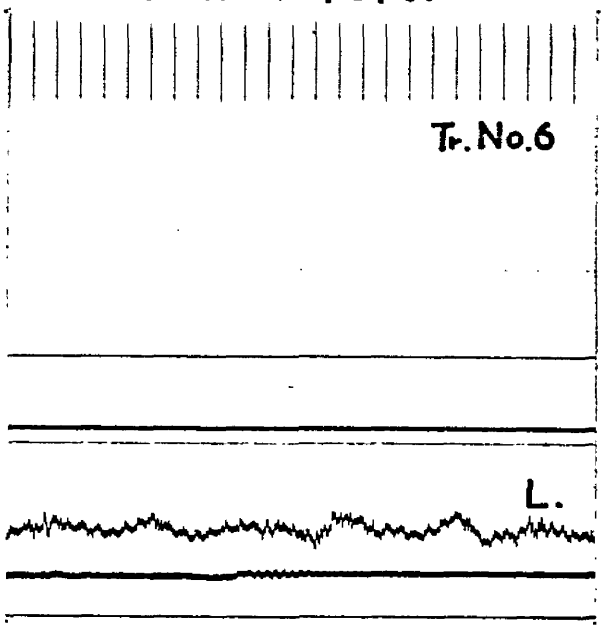
No.9 20.1.61 Left Kidney Osmotic Diuresis

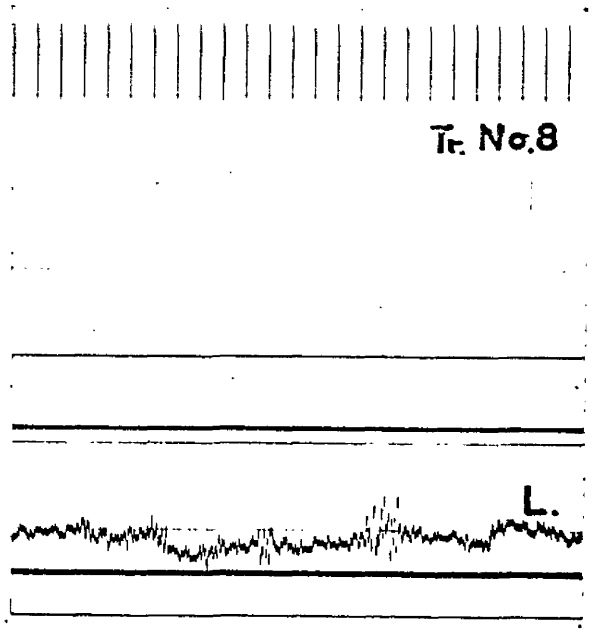
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec —
Basal	- mls	0 - 3 (0)	
5	15	1 - 4 (1)	
10	26	3 - 5 (2)	
15	20	5 - 7 (3)	
20	37	7 - 10 (4)	
25	50	14 - 18 (5)	
30	71	14 - 19 (6)	
35	50	26 - 29 (7)	
40	40	13 - 22 (8)	
45	40	10 - 13 (9)	



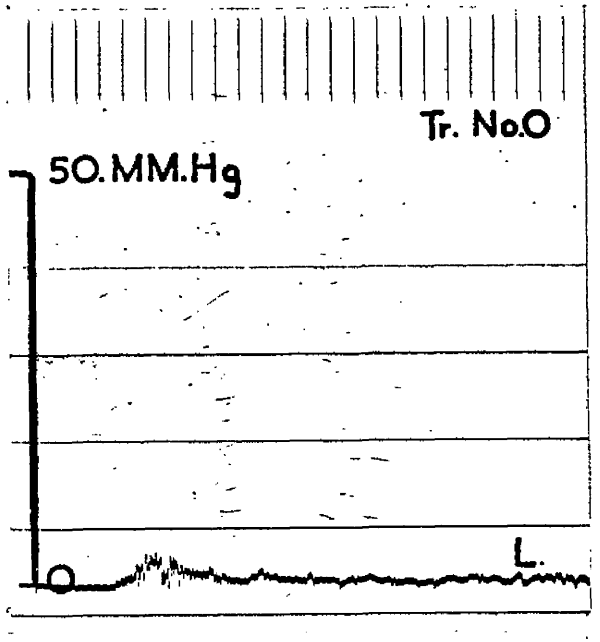
No II 13/3/61

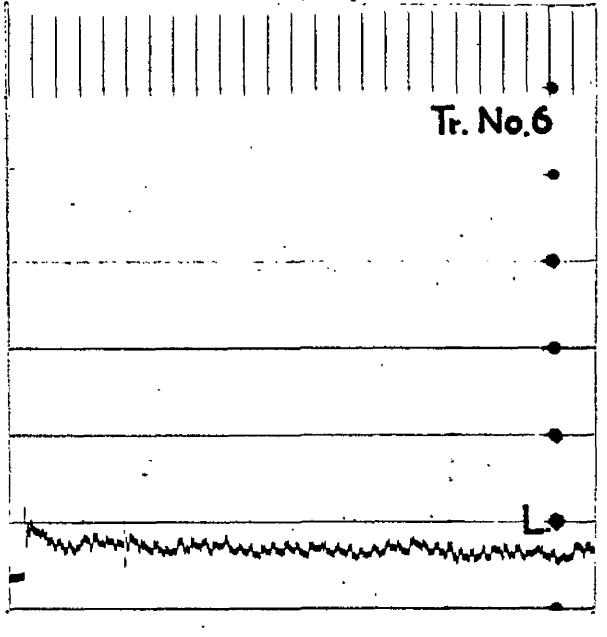


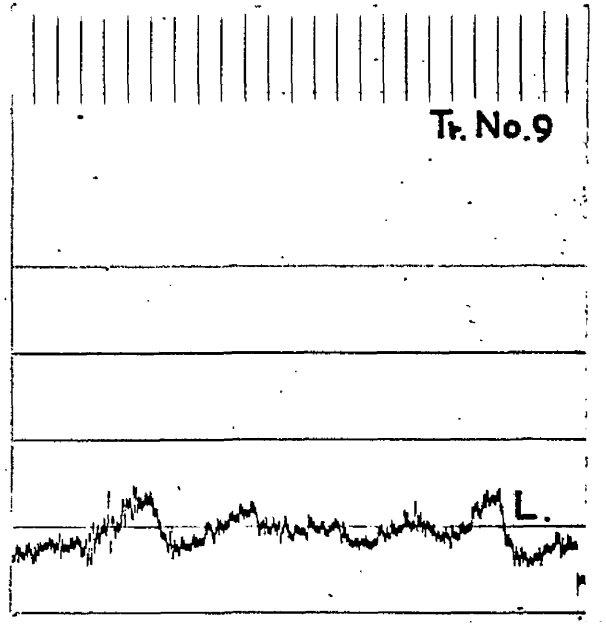




No II 14/3/61



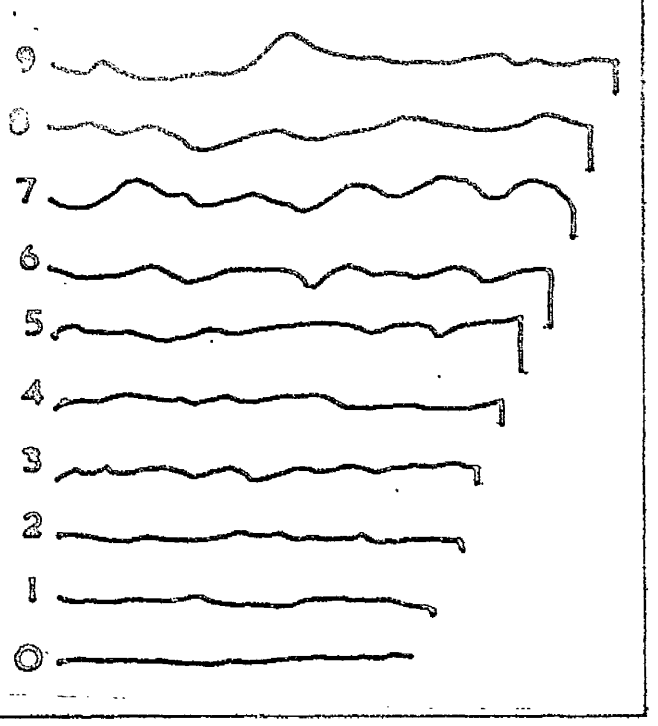




No.11 13.3.61 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid'	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 (0)
5	11	0 - 1 (1)
10	25	1 - 3 (2)
15	28	1 - 3 (3)
20	33	2 - 4 (4)
25	48	4 - 7 (5)
30	64	5 - 8 (6)
35	67	4 - 8 (7)
40	94	4 - 7 (8)
45	99	3 - 8 (9)

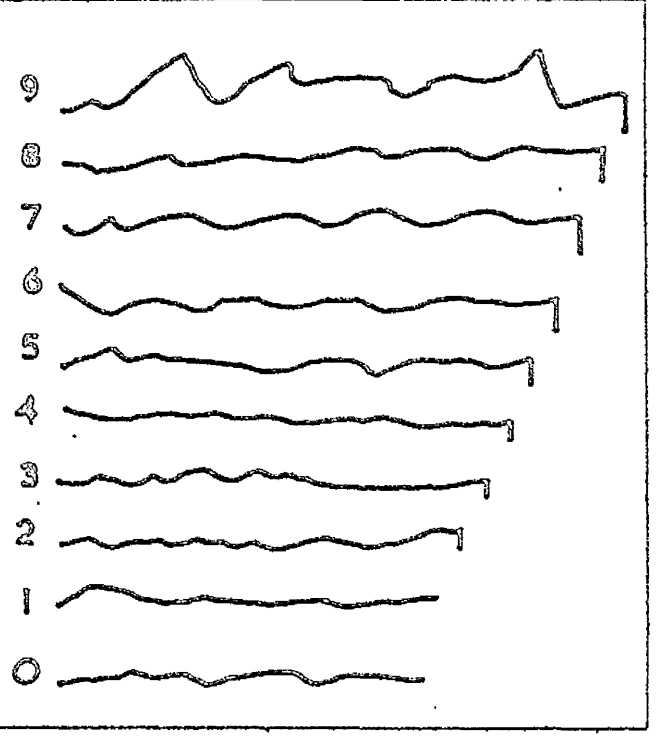
Pressure Tracing — 20 sec

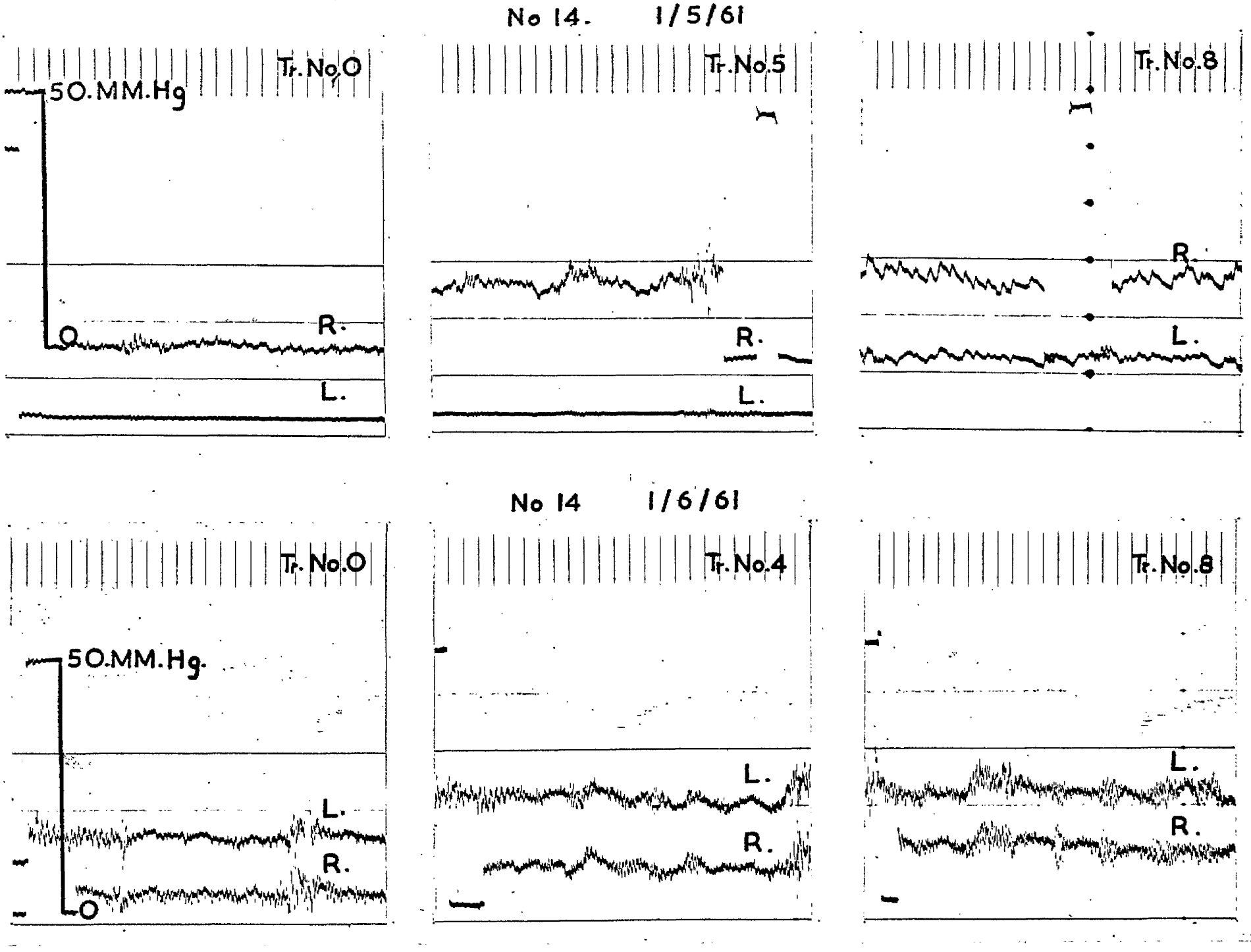


No.11 14.3.61 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	11	0 - 2 (1)
10	15	0 - 3 (2)
15	30	1 - 3 (3)
20	29	1 - 3 (4)
25	41	2 - 4 (5)
30	52	2 - 5 (6)
35	84	3 - 6 (7)
40	99	2 - 4 (8)
45	74	2 - 9 (9)

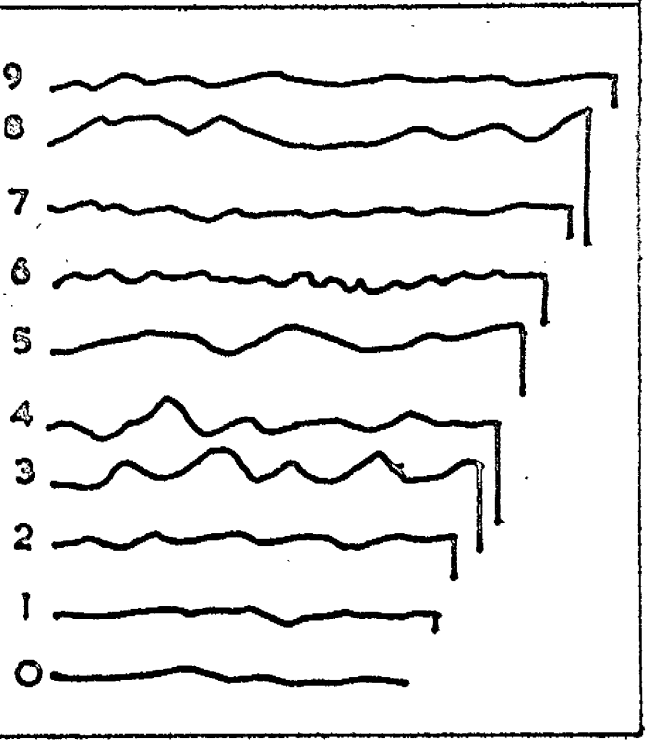
Pressure Tracing — 20 sec




No 14. 1/5/61
Tr. No. O
50. MM. Hg
R.
L.
Tr. No. 5
R.
L.
Tr. No. 8
R.
L.
No 14 1/6/61
Tr. No. O
50. MM. Hg.
L.
R.
Tr. No. 4
L.
R.
Tr. No. 8
L.
R.

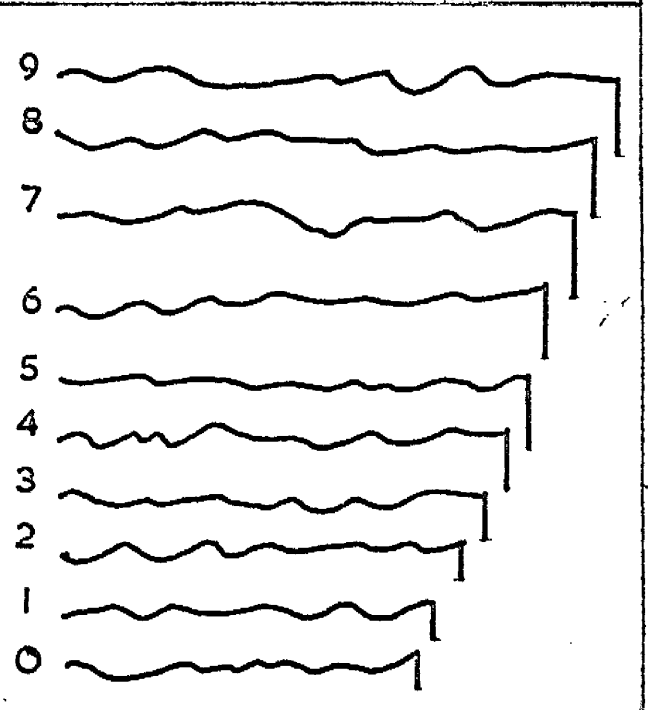
No.14 1.5.61 Right Kidney Osmotic Diuresis

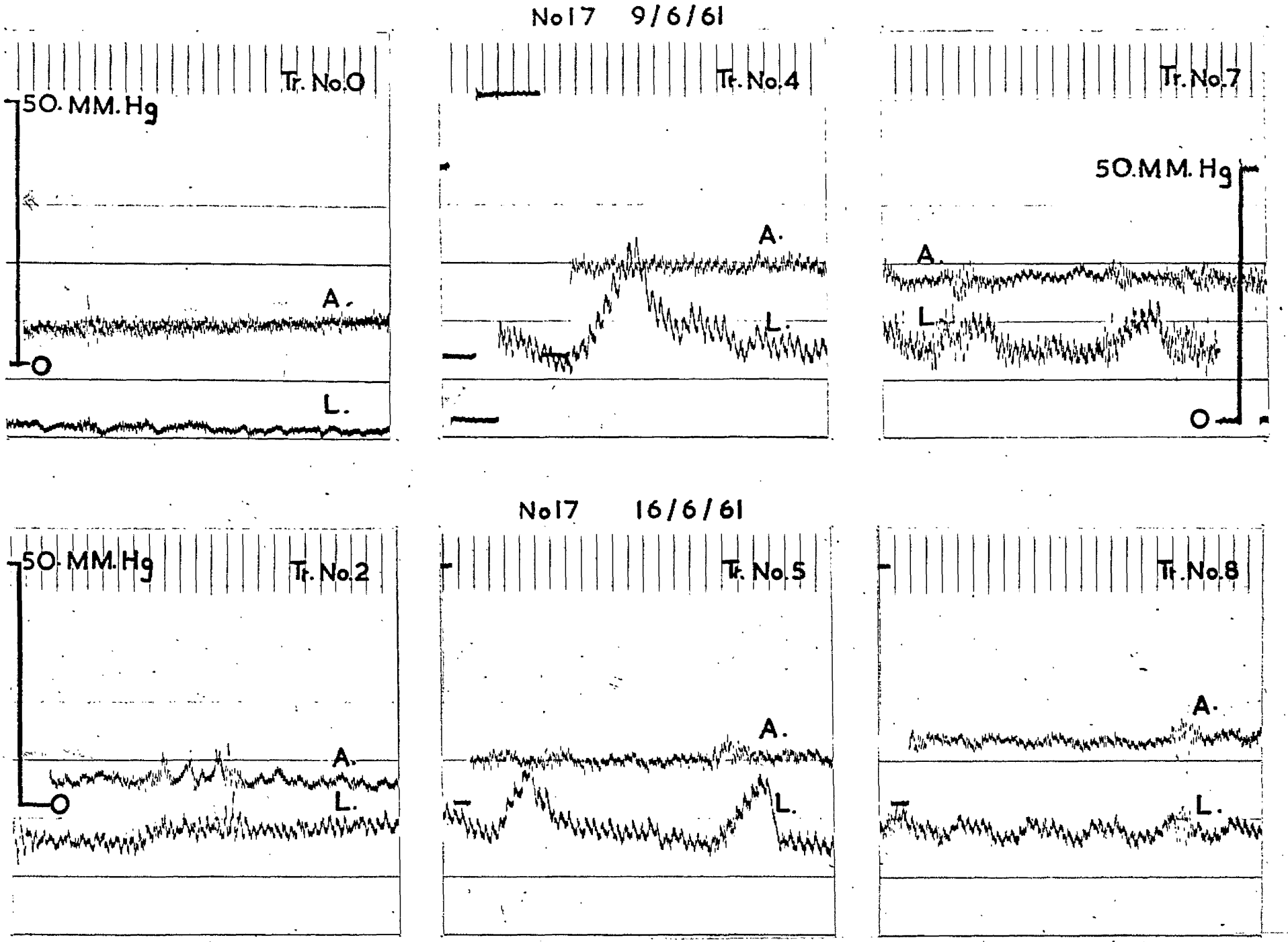
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	14	1 - 4 (1)
10	30	4 - 6 (2)
15	30	8 - 12 (3)
20	67	11 - 15 (4)
25	69	12 - 16 (5)
30	62	4 - 6 (6)
35	32	3 - 5 (7)
40	46	12 - 16 (8)
45	40	3 - 6 (9)



No.14 1.6.61 Right Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	1 - 4 (0)
5	1	2 - 5 (1)
10	3	3 - 6 (2)
15	18	5 - 8 (3)
20	34	6 - 9 (4)
25	36	8 - 10 (5)
30	32	6 - 8 (6)
35	20	8 - 12 (7)
40	44	8 - 11 (8)
45	30	8 - 11 (9)

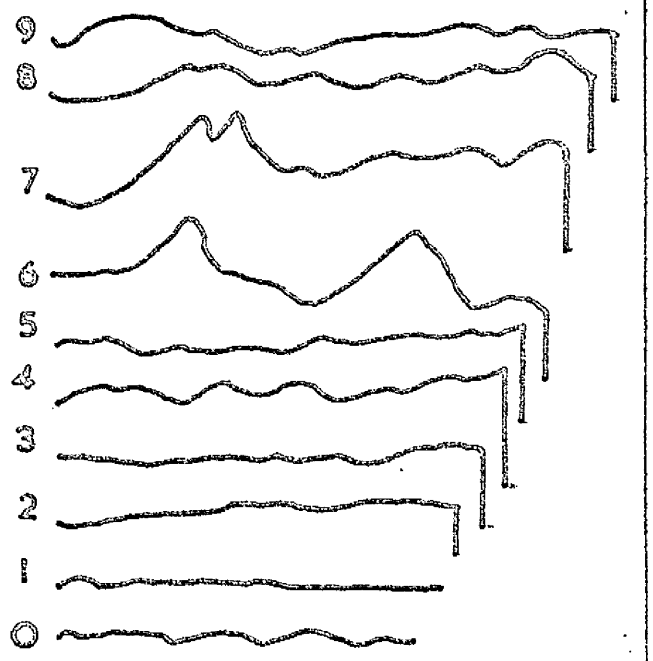



No17 9/6/61
Tr. No.0
50. MM. Hg
A.
O
L.
Tr. No.4
A.
L.
Tr. No.7
50.MM. Hg
A.
L.
O
No17 16/6/61
50. MM. Hg
Tr. No.2
A.
O
L.
Tr. No.5
A.
L.
Tr. No.8
A.
L.

No.17 9.6.61 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	23	0 - 1 (1)
10	19	3 - 6 (2)
15	32	7 - 10 (3)
20	33	11 - 14 (4)
25	44	7 - 11 (5)
30	46	7 - 20 (6)
35	54	6 - 16 (7)
40	72	6 - 12 (8)
45	70	6 - 10 (9)

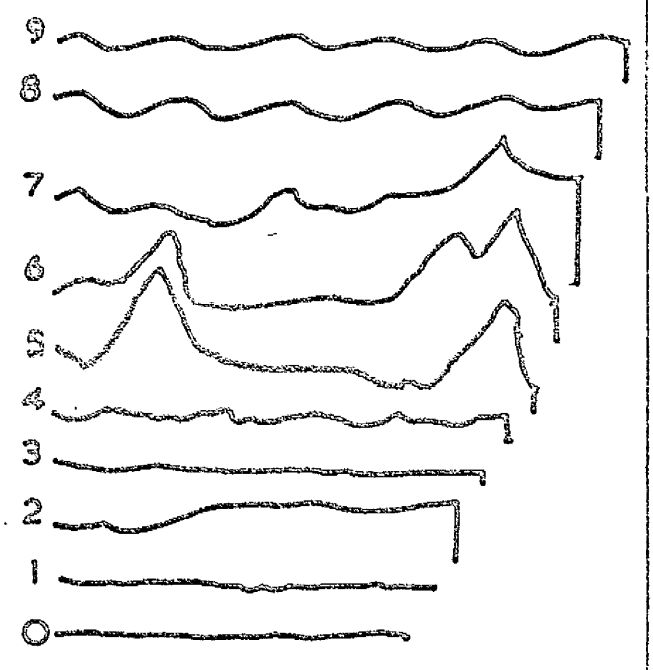
Pressure Tracing
20 sec



No.17 16.6.61 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto & L.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 (0)
5	14	0 (1)
10	21	3 - 8 (2)
15	31	0 - 1 (3)
20	40	3 - 4 (4)
25	49	3 - 18 (5)
30	59	4 - 16 (6)
35	64	8 - 18 (7)
40	76	5 - 9 (8)
45	60	3 - 5 (9)

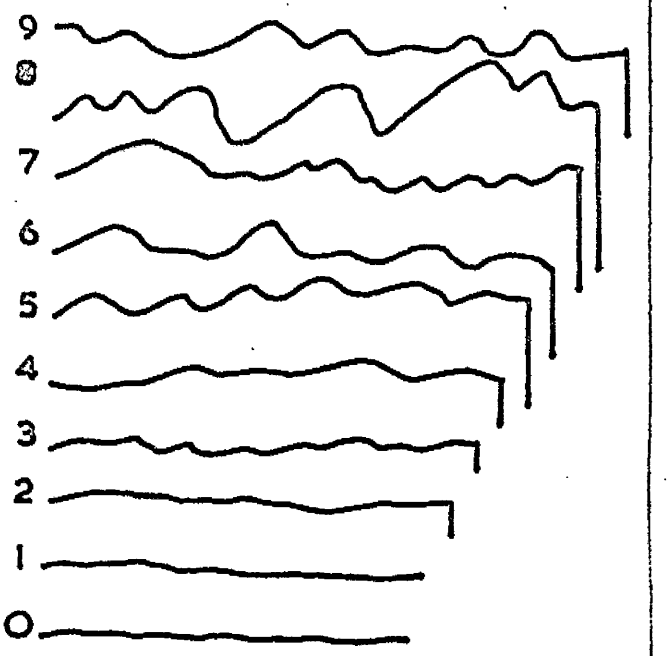
Pressure Tracing
20 sec

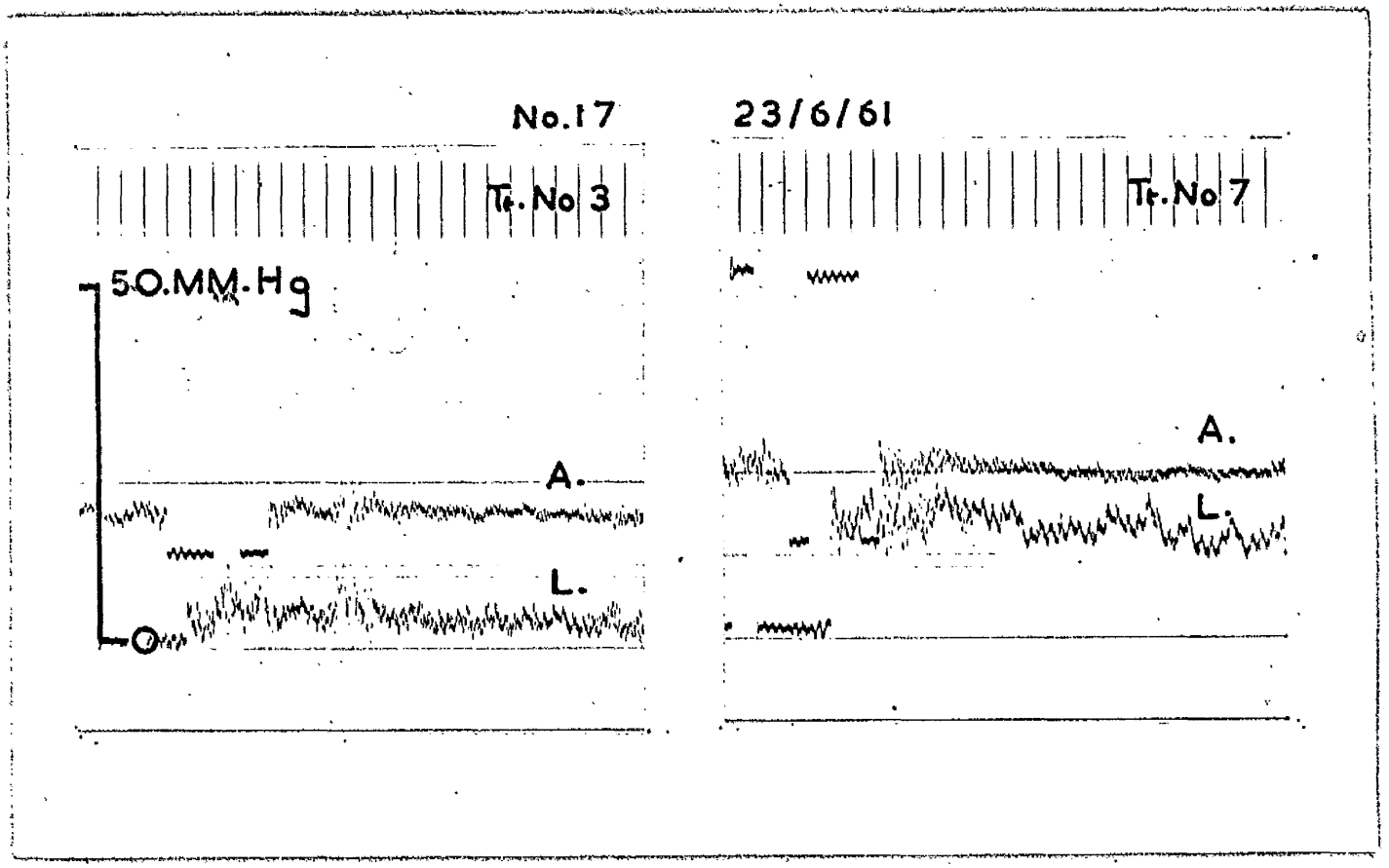


No.17 23.6.61 Left Kidney Osmotic Diuresis

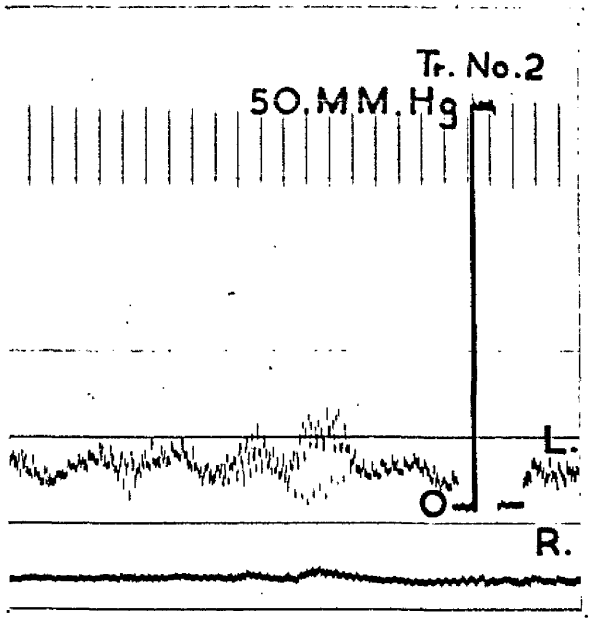
Time (Mins)	Urine/5 min Auto & L.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	2	0 - 1 (1)
10	12	4 - 6 (2)
15	20	3 - 5 (3)
20	45	5 - 7 (4)
25	37	11 - 17 (5)
30	62	10 - 16 (6)
35	70	12 - 16 (7)
40	105	12 - 20 (8)
45	73	10 - 13 (9)

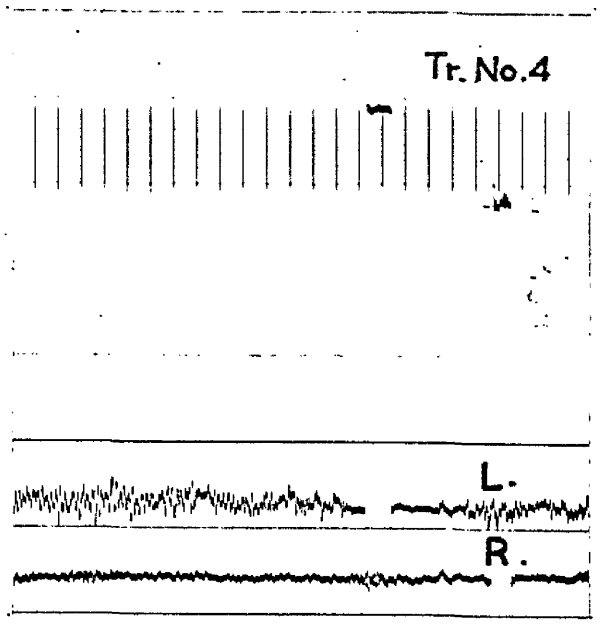
Pressure Tracing
20 sec

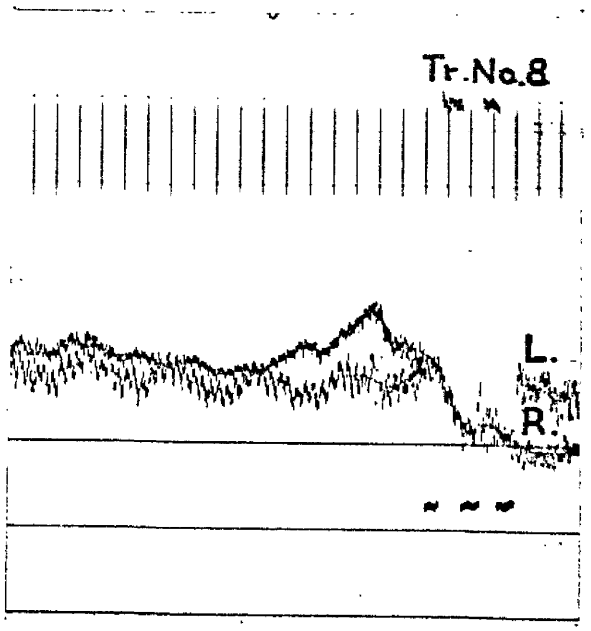




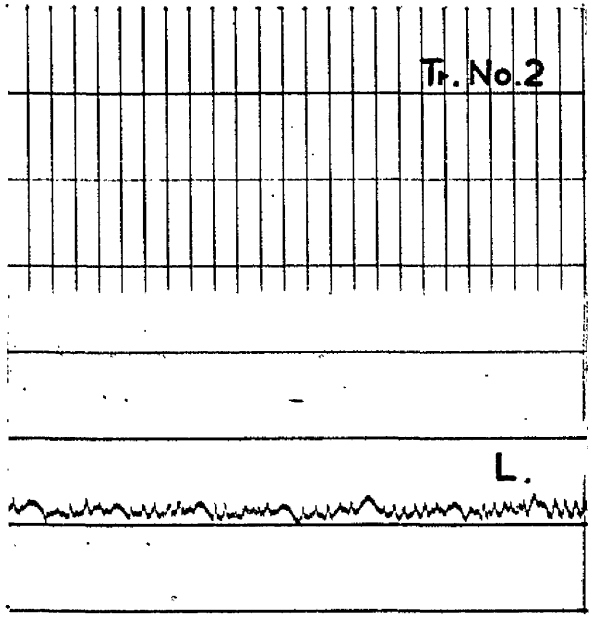
No 19 3/8/61

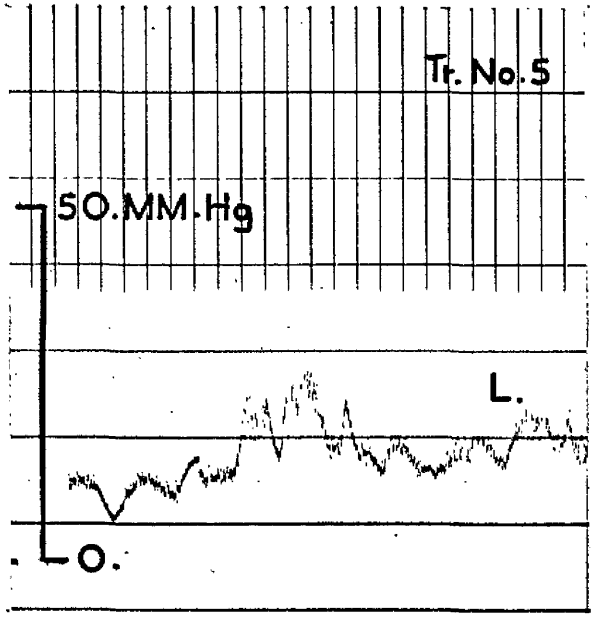


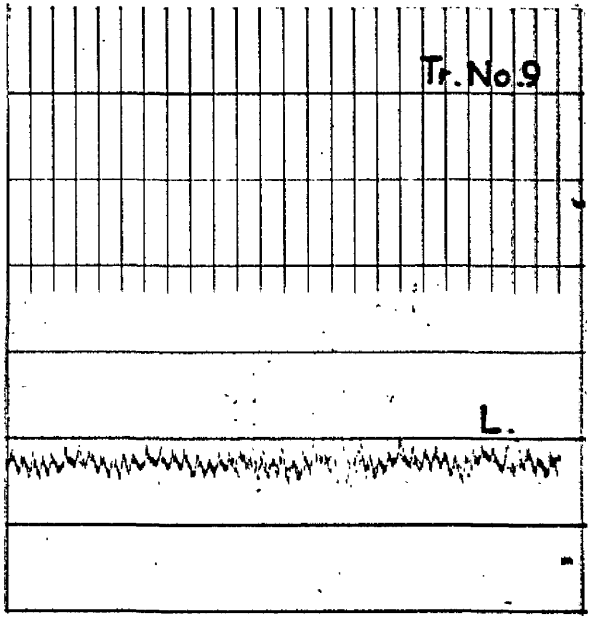




No 19 27/8/61

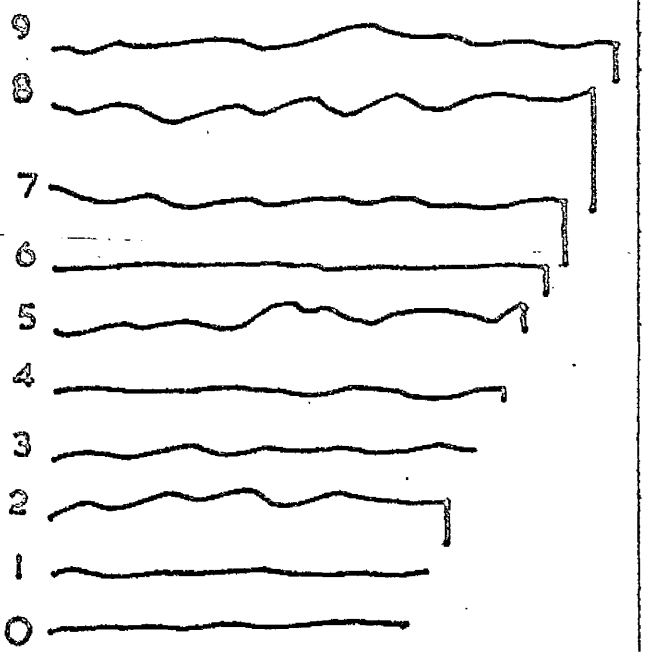






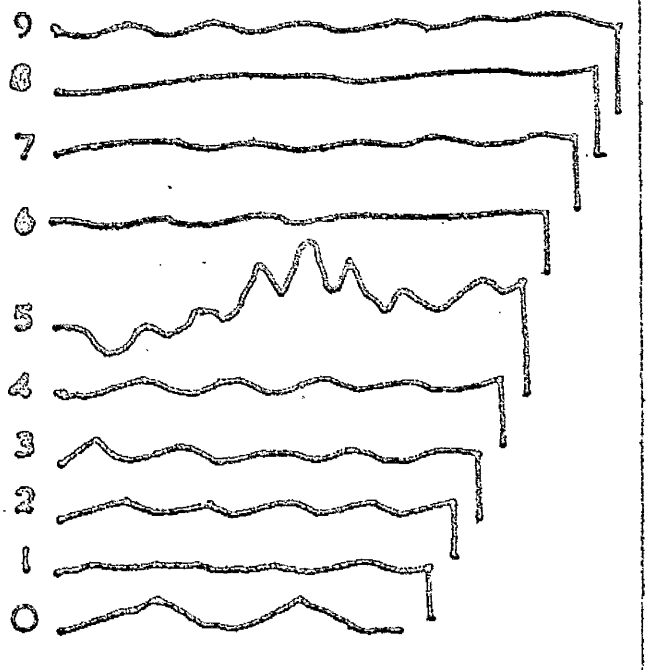
No.19 3.8.61 Left Kidney Osmotic Diuresis

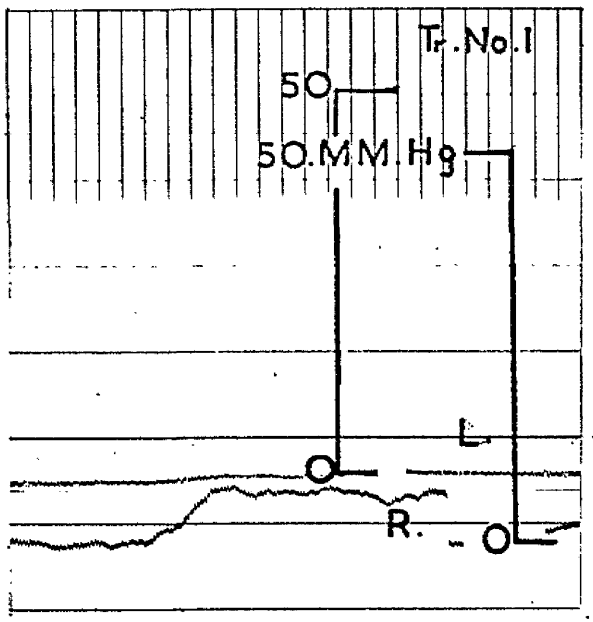
Time (Mins)	Urine/5 min R & L Kid.	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 (0)	
5	2	0 (1)	
10	7	4 - 7 (2)	
15	17	0 - 2 (3)	
20	10	0 - 2 (4)	
25	76	1 - 5 (5)	
30	30	3 (6)	
35	79	6 - 9 (7)	
40	87	10 - 14 (8)	
45	62	4 - 7 (9)	



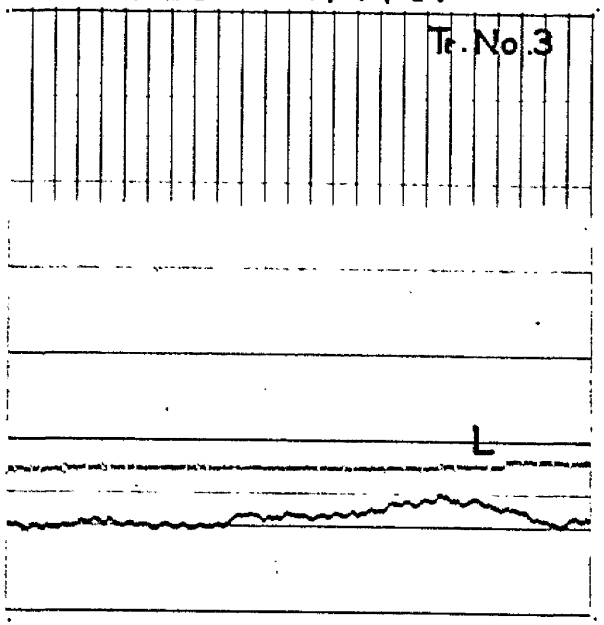
No.19 27.8.61 Left Kidney Osmotic Diuresis

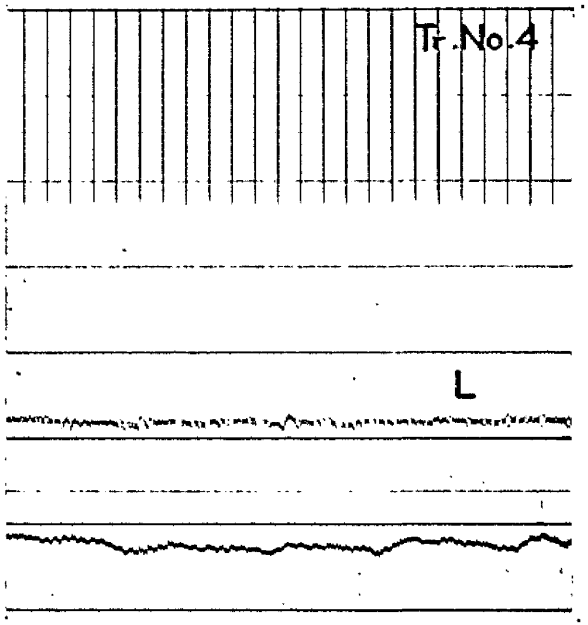
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 - 5 (0)	
5	12	6 - 7 (1)	
10	20	5 - 8 (2)	
15	19	7 - 10 (3)	
20	26	6 - 10 (4)	
25	49	7 - 20 (5)	
30	41	5 - 7 (6)	
35	29	8 - 9 (7)	
40	72	7 - 11 (8)	
45	70	10 - 13 (9)	

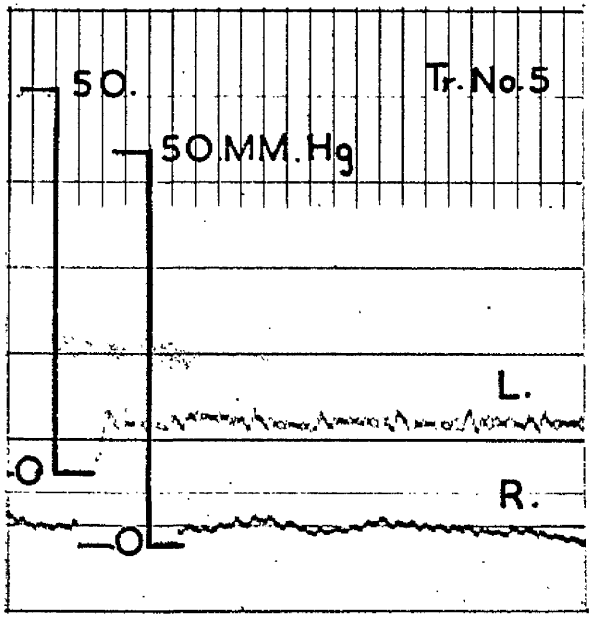




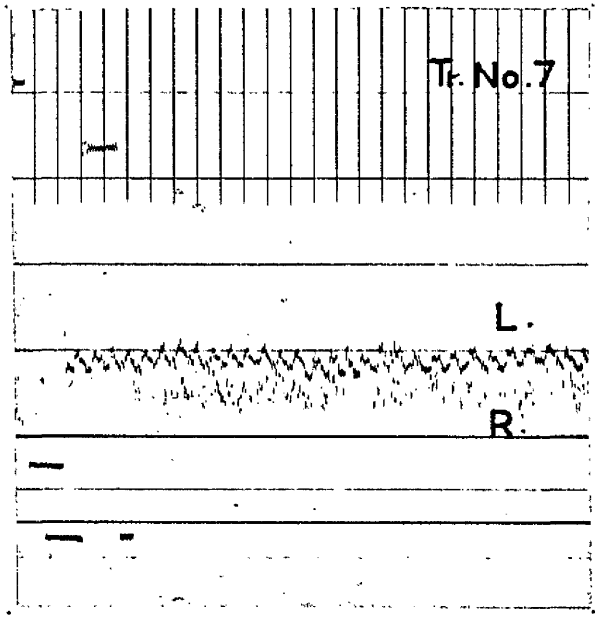
No 20 1/9/61

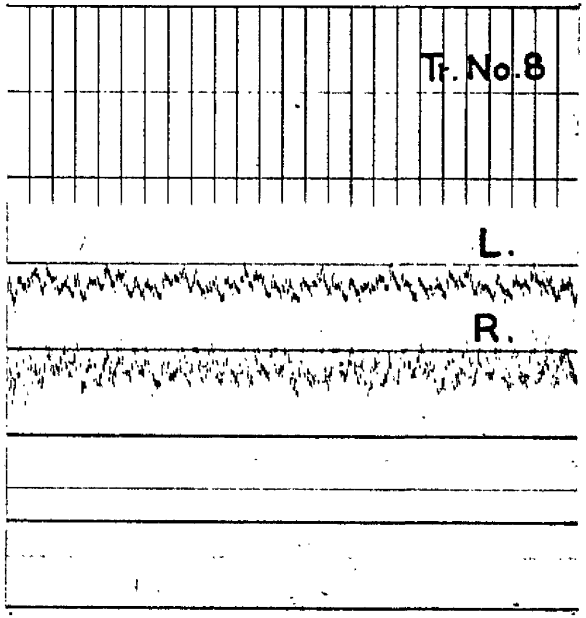






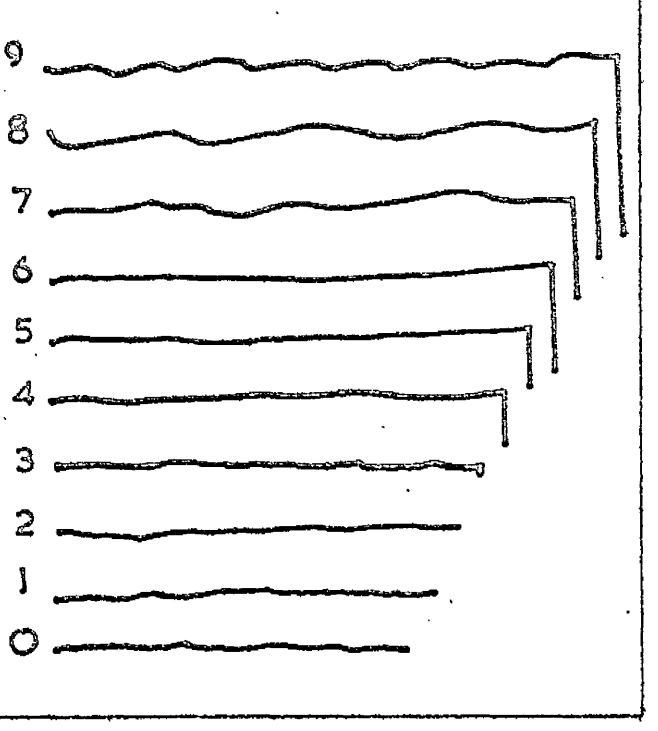
No 20 1/9/61





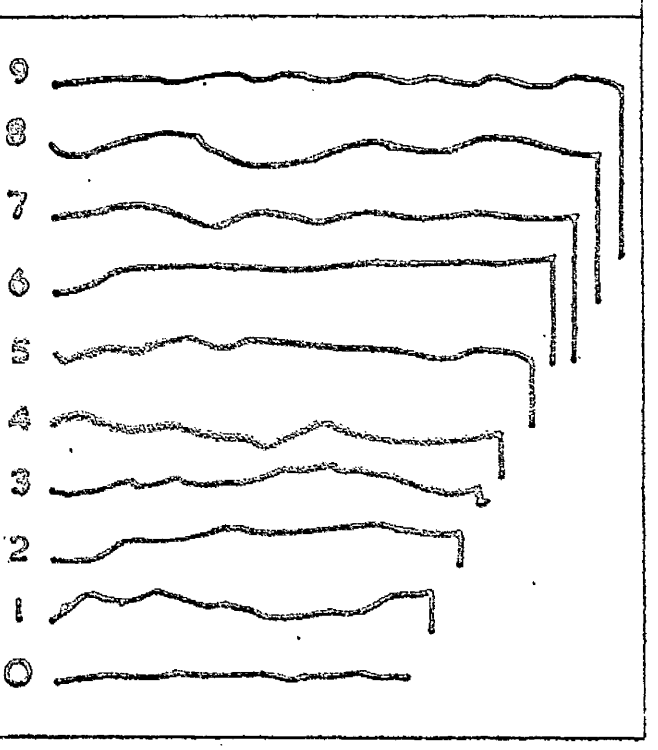
No.20 1.9.61 Left Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L Kid.	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 (0)
5	5	0 (1)
10	13	0 (2)
15	30	1 (3)
20	49	6 (4)
25	47	7 (5)
30	49	12 (6)
35	52	11 - 13 (7)
40	61	13 - 15 (8)
45	89	19 - 21 (9)

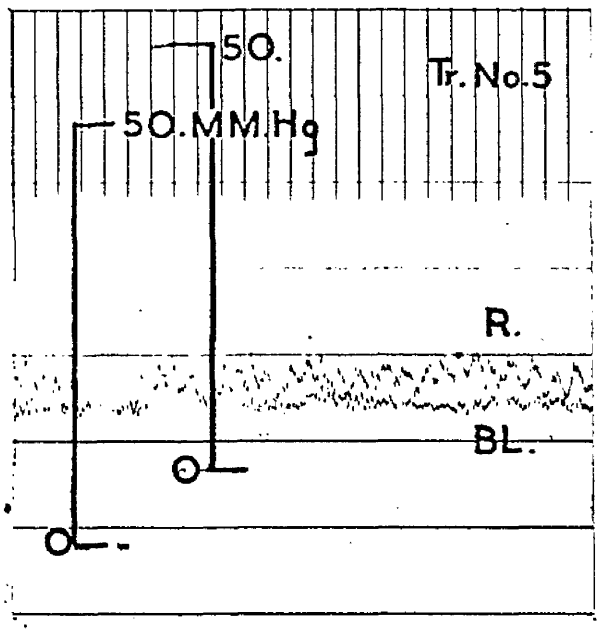


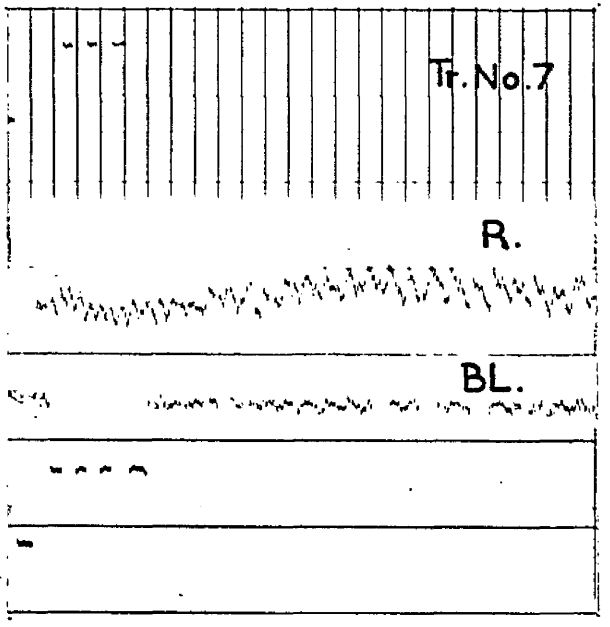
No.20 1.9.61 Right Kidney Osmotic Diuresis

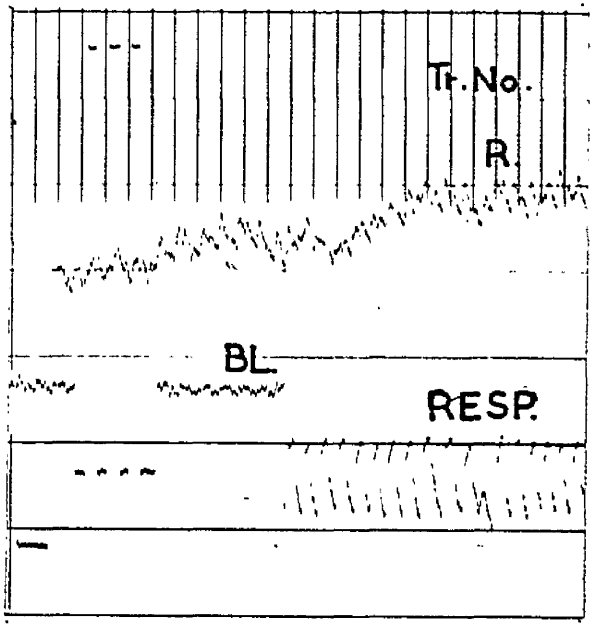
Time (Mins)	Urine/5-min R & L Kid.	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 1 (0)
5	5	1 - 5 (1)
10	13	0 - 5 (2)
15	30	2 - 6 (3)
20	49	0 - 6 (4)
25	47	8 - 10 (5)
30	49	10 - 12 (6)
35	52	15 - 17 (7)
40	61	17 - 20 (8)
45	89	19 - 22 (9)



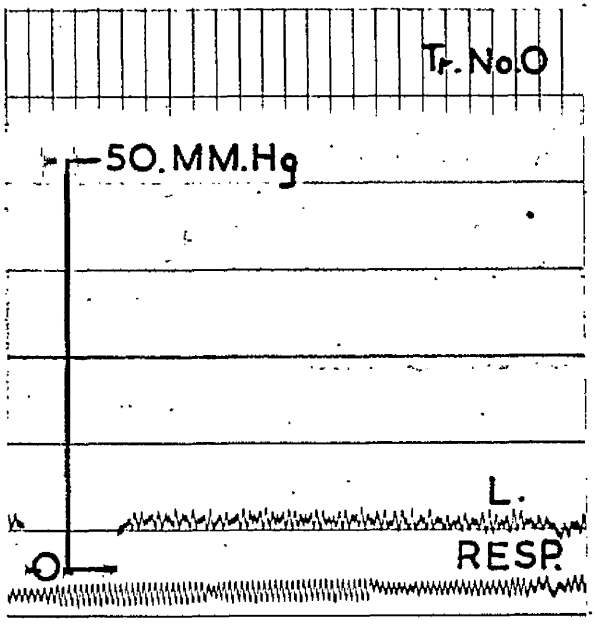
No 20 7/9/61

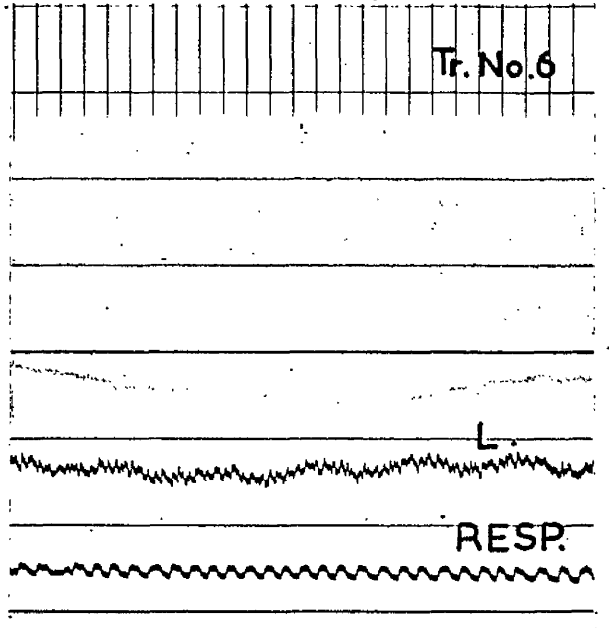


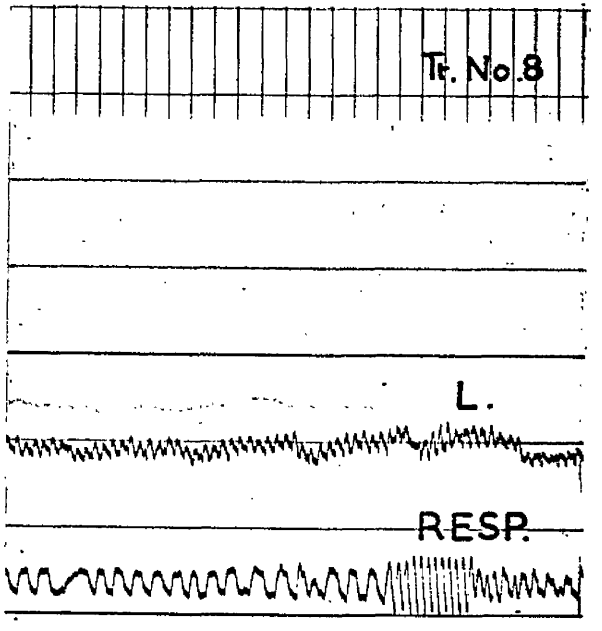




No 20 25/1/62



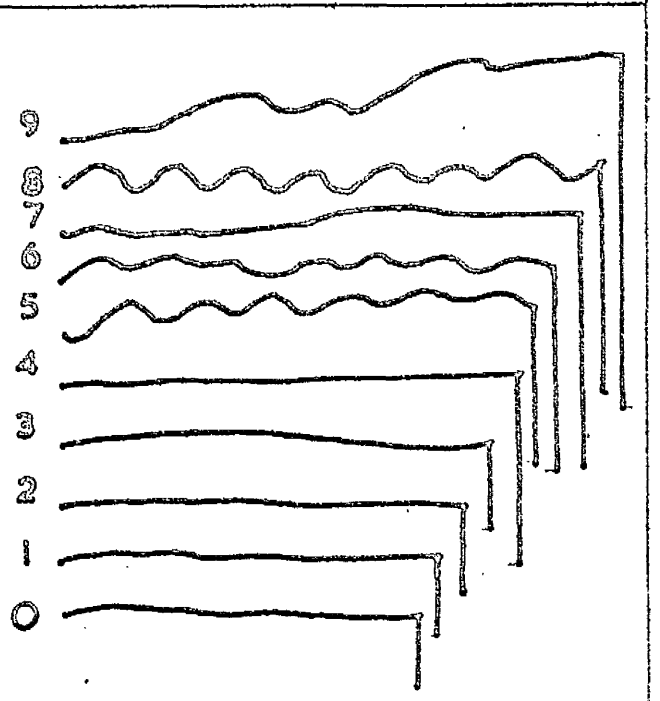




No.20 7.9.61 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)
Basal	0	7 (0)
5	4	9 - 11 (1)
10	9	9 - 13 (2)
15	10	10 - 12 (3)
20	10	21 - 24 (4)
25	8	16 - 21 (5)
30	8	24 - 27 (6)
35	8	28 - 31 (7)
40	9	23 - 29 (8)
45	10	31 - 42 (9)

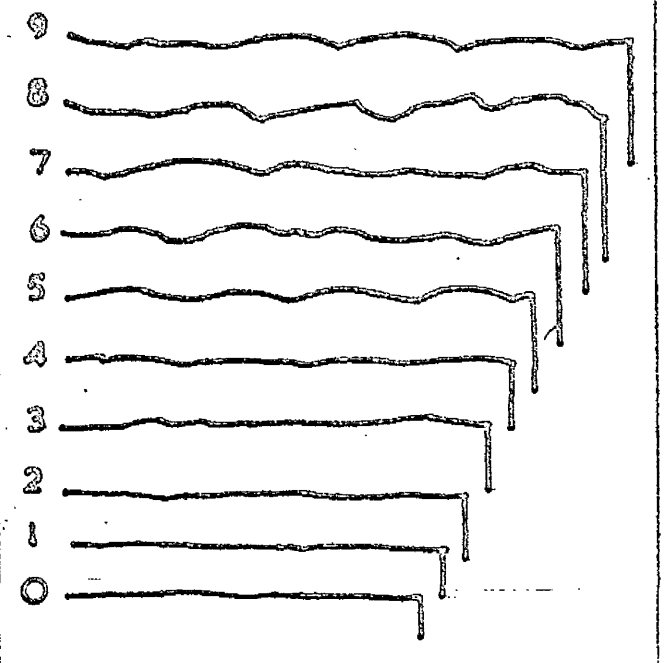
Pressure Tracing
20 sec



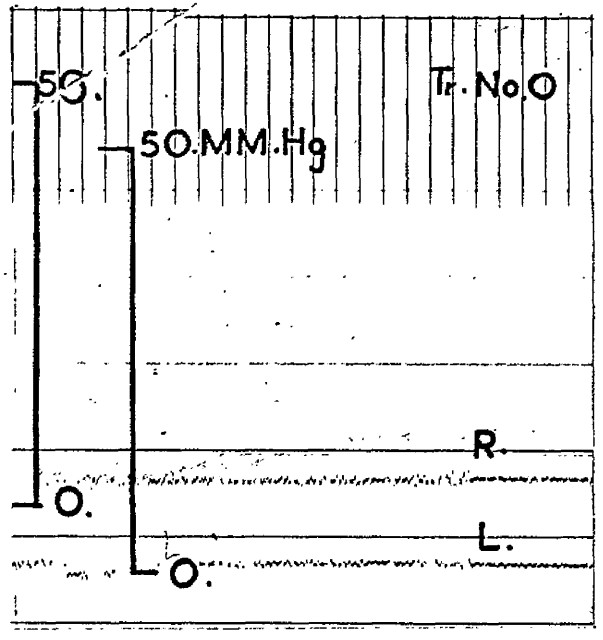
No.20 25.1.62 Left Kidney Osmotic Diuresis

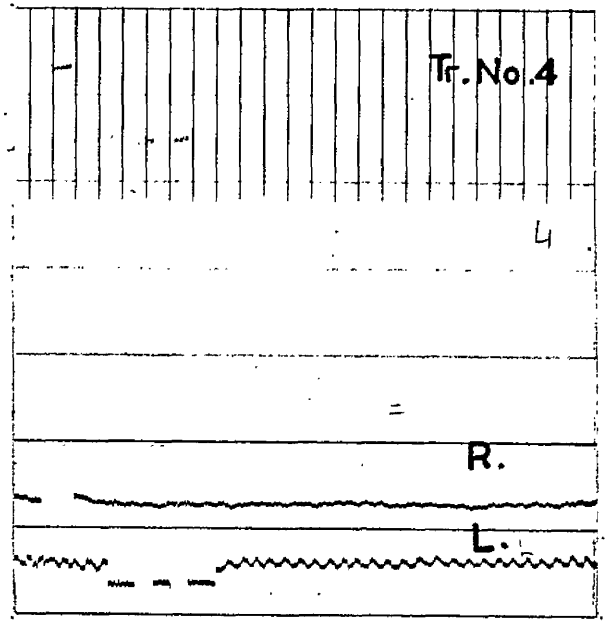
Time (Mins)	Urine/5 min L.Kid	Pelv. Press mm Hg.(Tr.No)
Basal	- mls	5 (0)
5	3	6 (1)
10	3	6 - 8 (2)
15	5	7 - 9 (3)
20	10	7 - 9 (4)
25	22	10 - 14 (5)
30	31	11 - 15 (6)
35	37	13 - 15 (7)
40	42	16 - 20 (8)
45	58	13 - 15 (9)

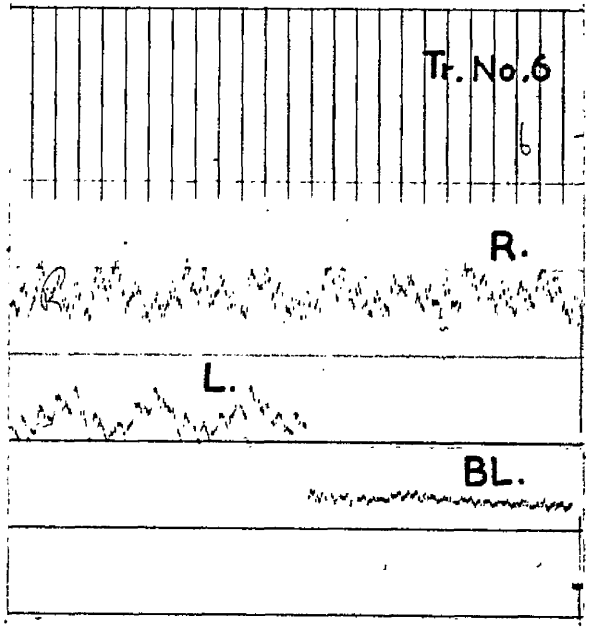
Pressure Tracing
20 sec



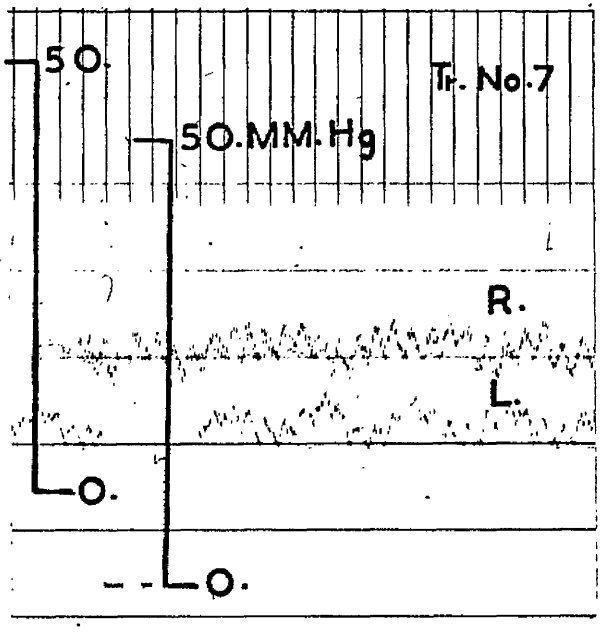
No 20 14/9/61

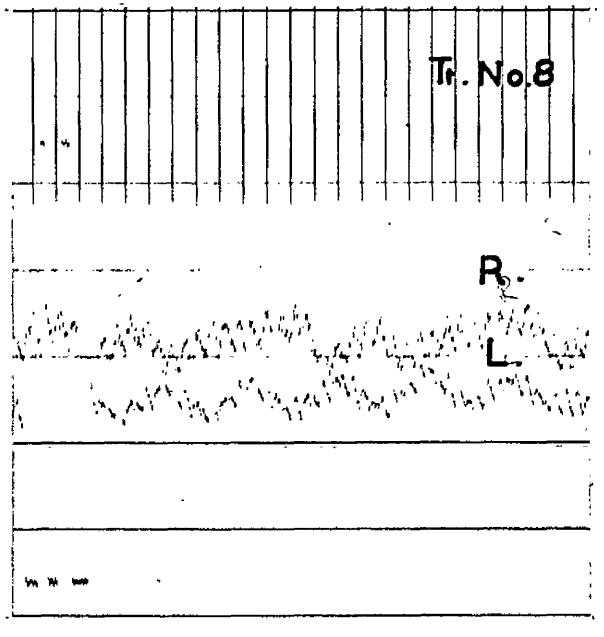


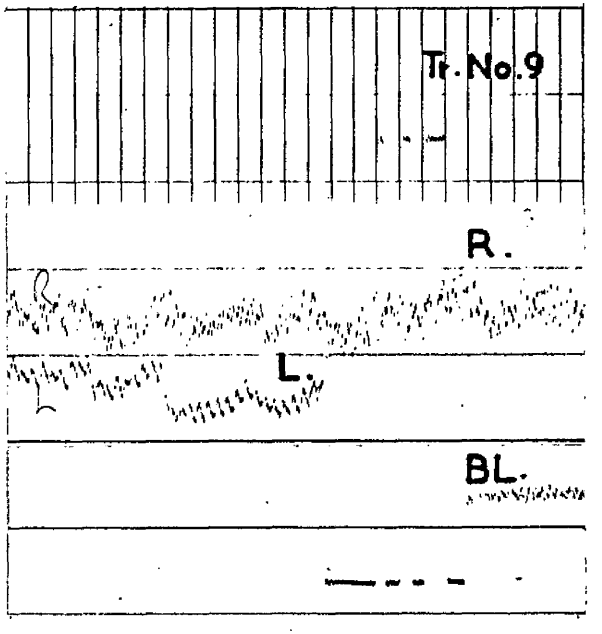




No 20 14/9/61

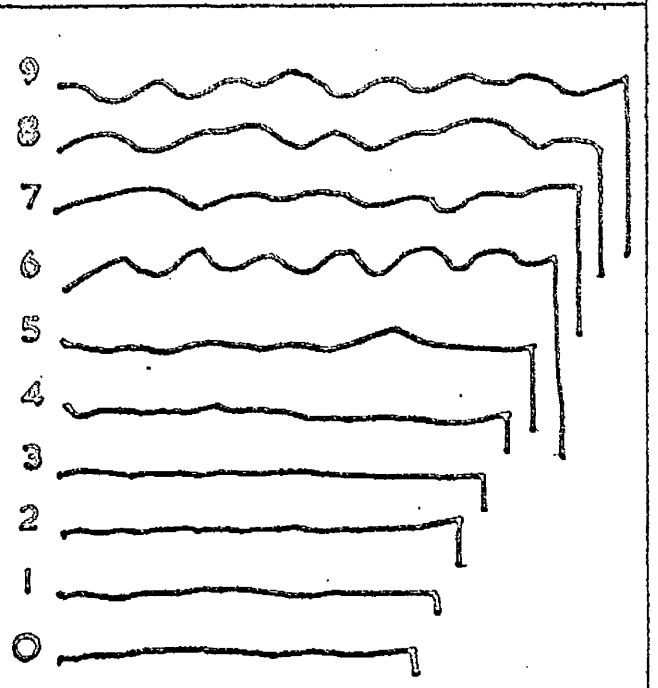






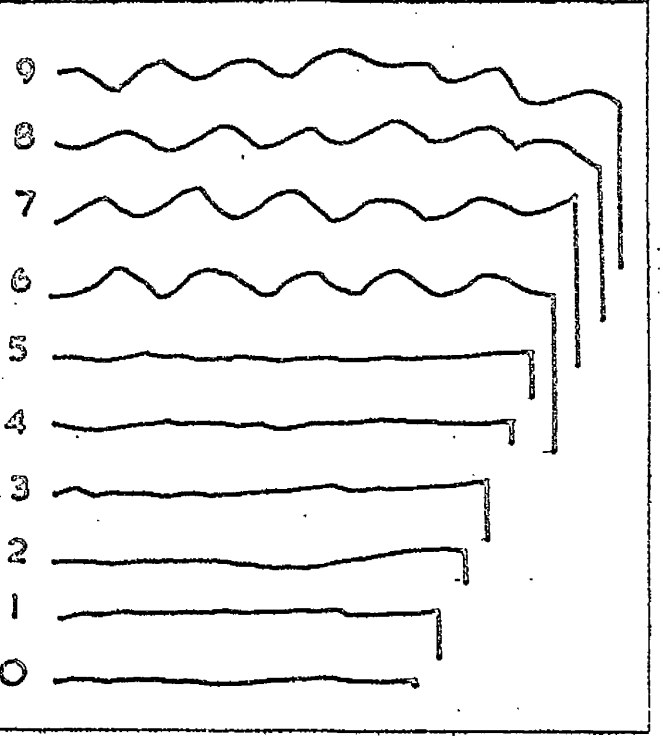
No.20 14.9.61 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	10	2 - 3 (0)	
5	10	2 (1)	
10	9	4 - 6 (2)	
15	9	4 - 6 (3)	
20	9	10 - 13 (4)	
25	10	10 - 12 (5)	
30	9	19 - 26 (6)	
35	10	17 - 19 (7)	
40	10	15 - 20 (8)	
45	10	19 - 23 (9)	



No.20 14.9.61 Left Kidney Osmotic Diuresis

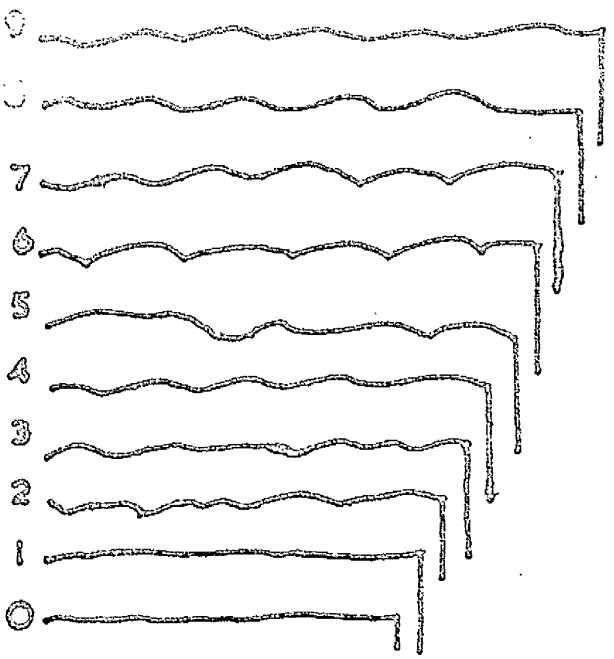
Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	10	1 - 2 (0)	
5	10	4 - 6 (1)	
10	9	2 - 4 (2)	
15	9	5 - 7 (3)	
20	9	3 - 4 (4)	
25	10	5 - 6 (5)	
30	9	19 - 22 (6)	
35	10	17 - 22 (7)	
40	10	17 - 24 (8)	
45	10	19 - 26 (9)	

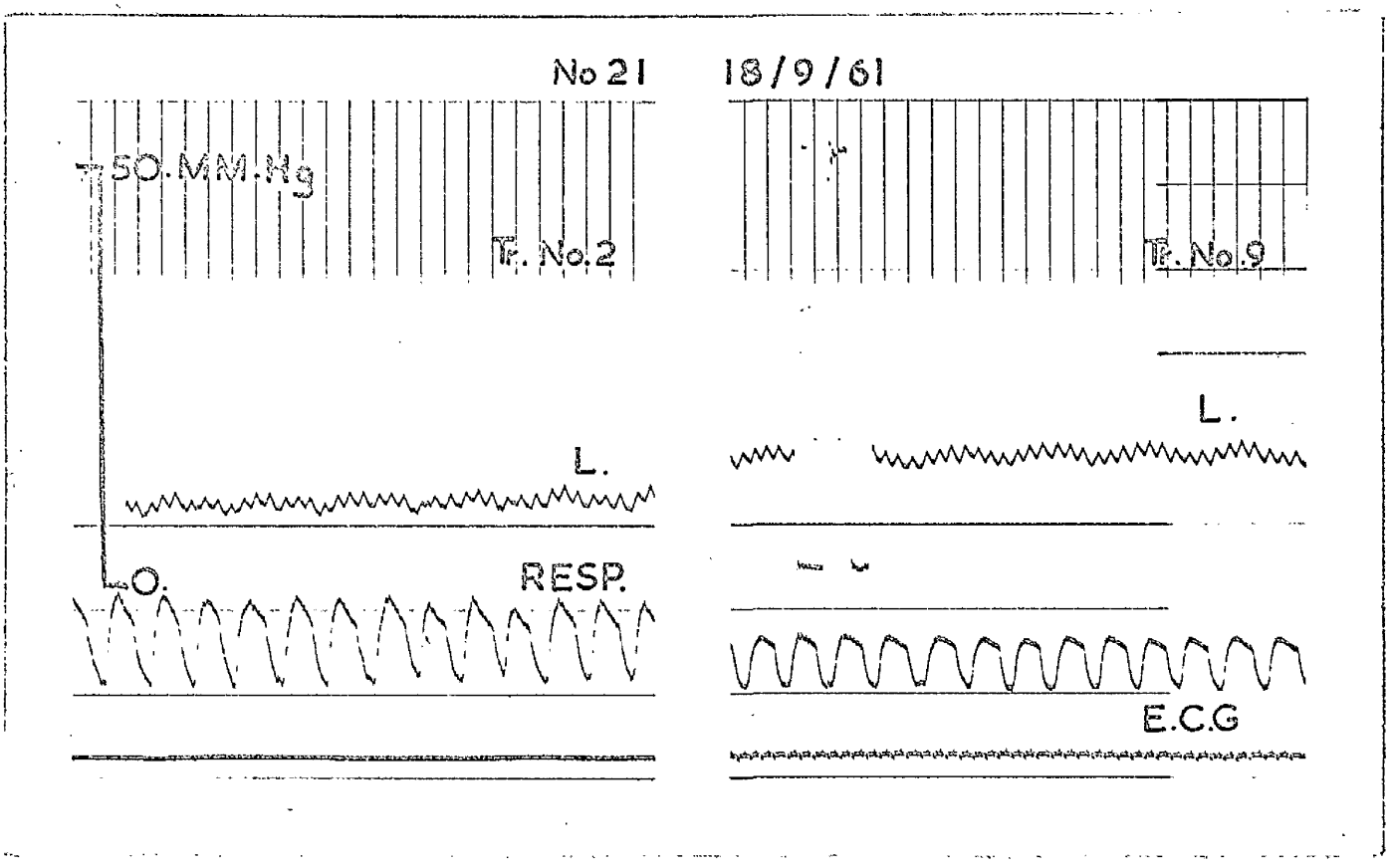


No.21 18.9.61 Left Kidney Osmotic Diuresis

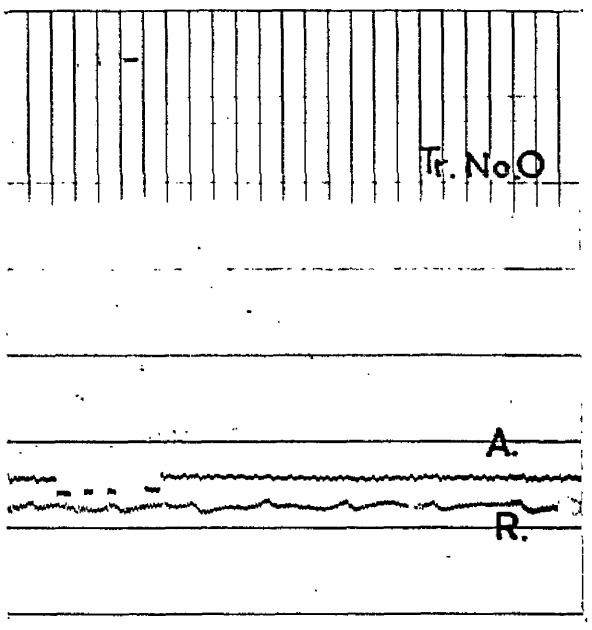
Time (Mins)	Blad. Press mm.Hg	Pelv. Press mm Hg. (Tr.No)
Basal	4	3 - 4 (0)
5	5	10 - 12 (1)
	5	7 - 12 (2)
15	5	10 - 14 (3)
20	6	10 - 17 (4)
25	6	12 - 18 (5)
30	8	7 - 16 (6)
35	8	12 - 15 (7)
40	9	12 - 16 (8)
45	9	12 - 15 (9)

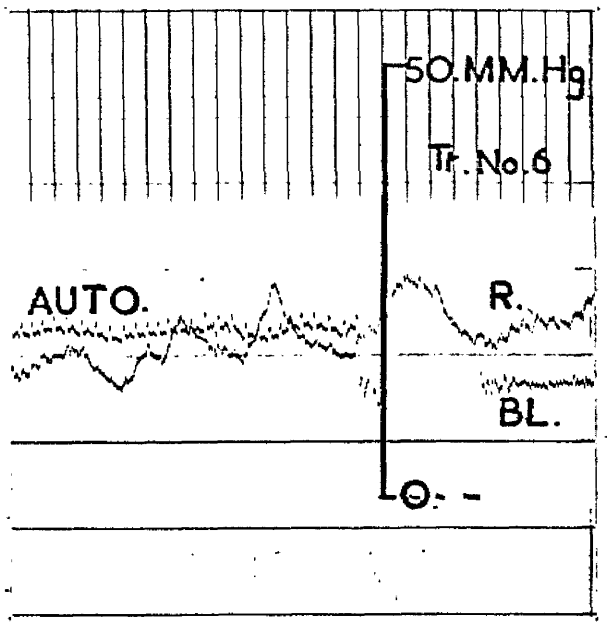
Pressure Tracing — 20 sec

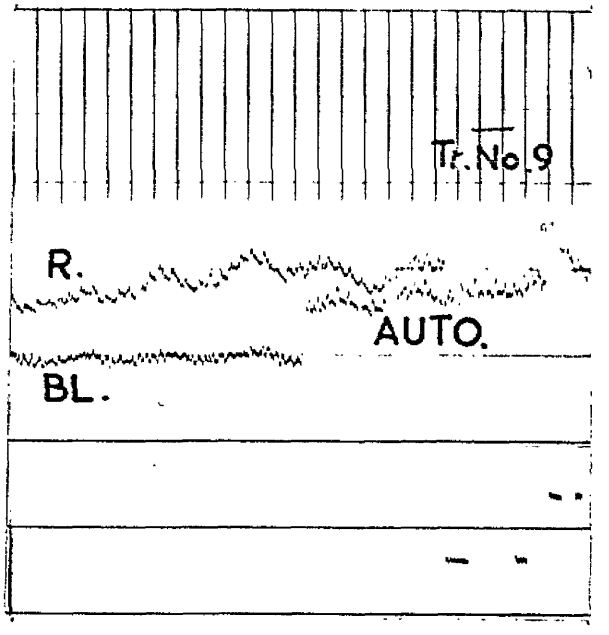




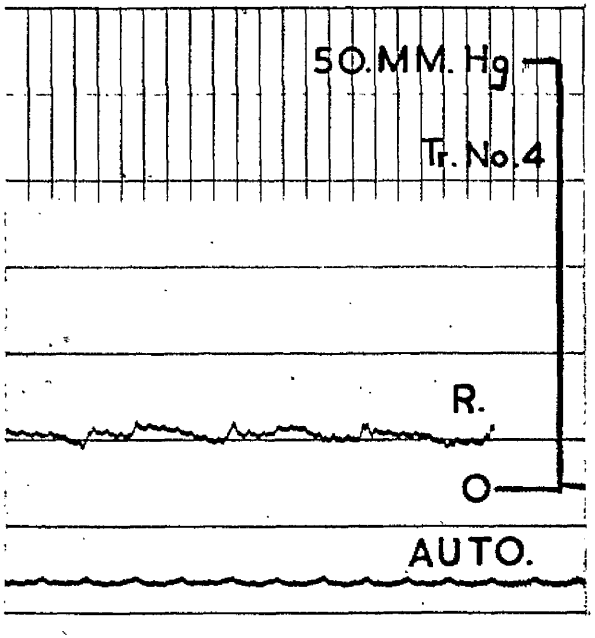
No 22 14/9/61

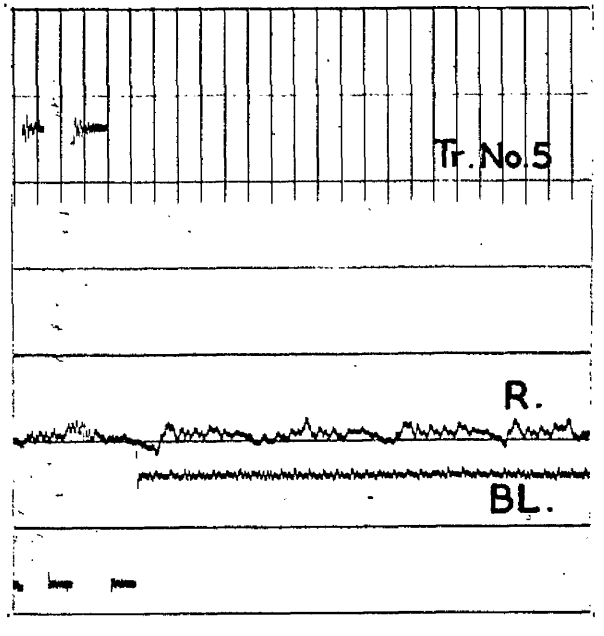


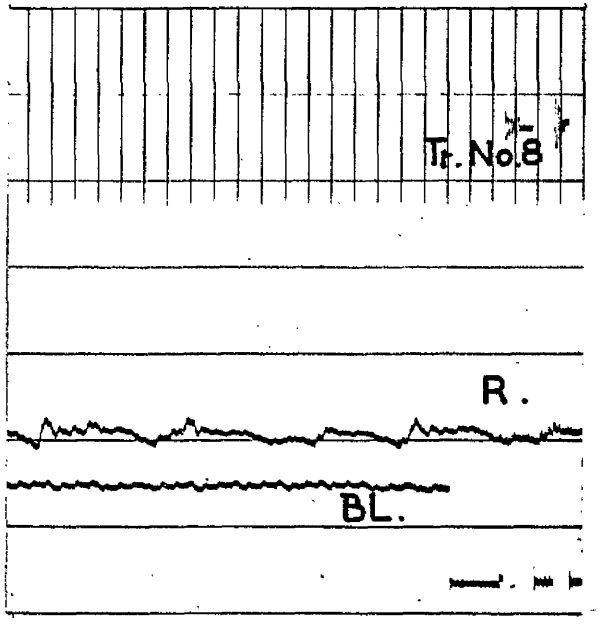




No 22 22/9/61



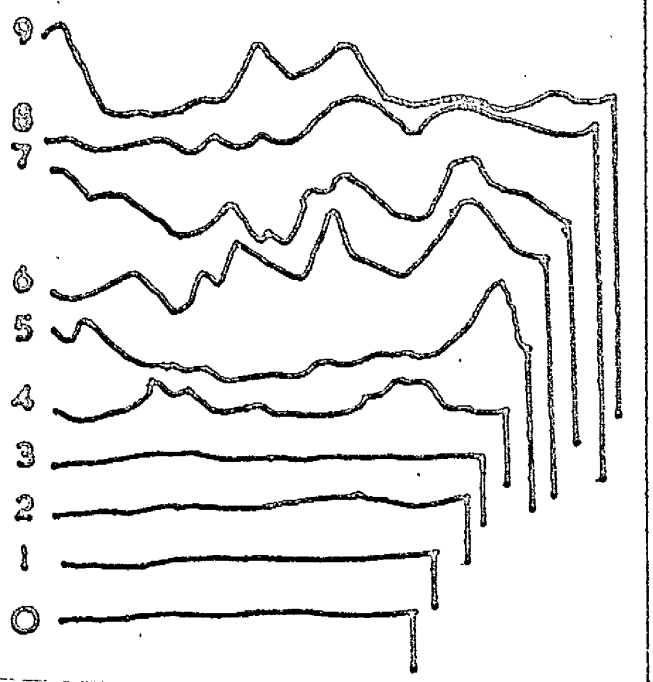




No.22 14.9.61 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg. (Tr.No)
Basal	2	7 (0)
5	4	7 (1)
10	6	8 (2)
15	8	8 (3)
20	11	9 - 18 (4)
25	13	15 - 27 (5)
30	13	22 - 35 (6)
35	16	25 - 35 (7)
40	16	35 - 47 (8)
45	16	30 - 42 (9)

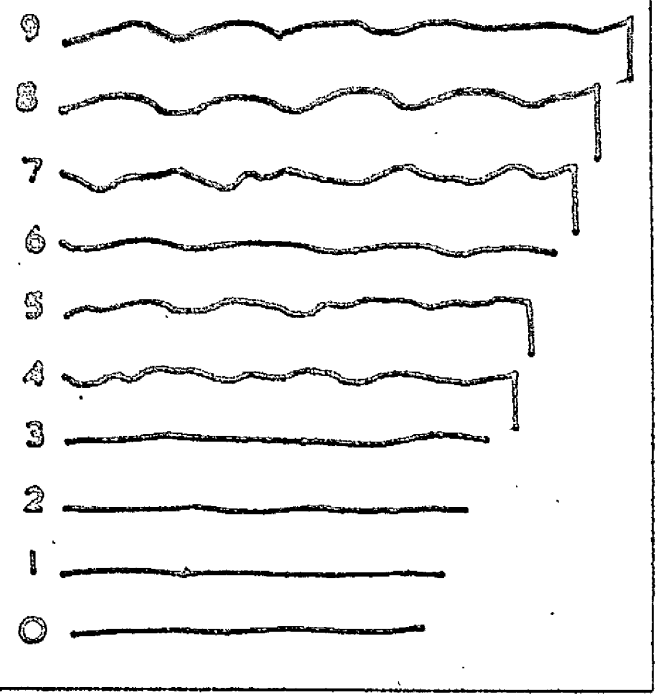
Pressure Tracing
20 sec



No.22 22.9.61 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg. (Tr.No)
Basal	4	0 (0)
5	5	0 (1)
10	7	0 (2)
15	9	0 - 1 (3)
20	11	5 - 8 (4)
25	12	5 - 8 (5)
30	12	0 - 2 (6)
35	13	5 - 8 (7)
40	15	5 - 9 (8)
45	15	5 - 7 (9)

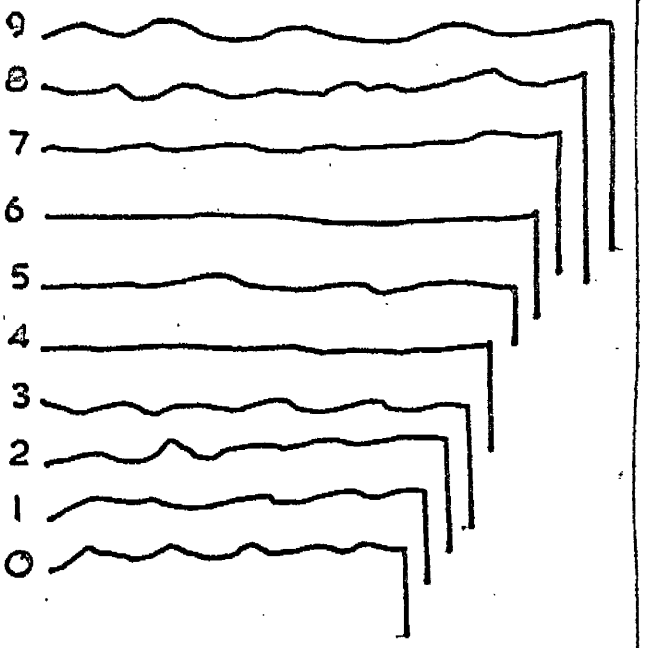
Pressure Tracing
20 sec

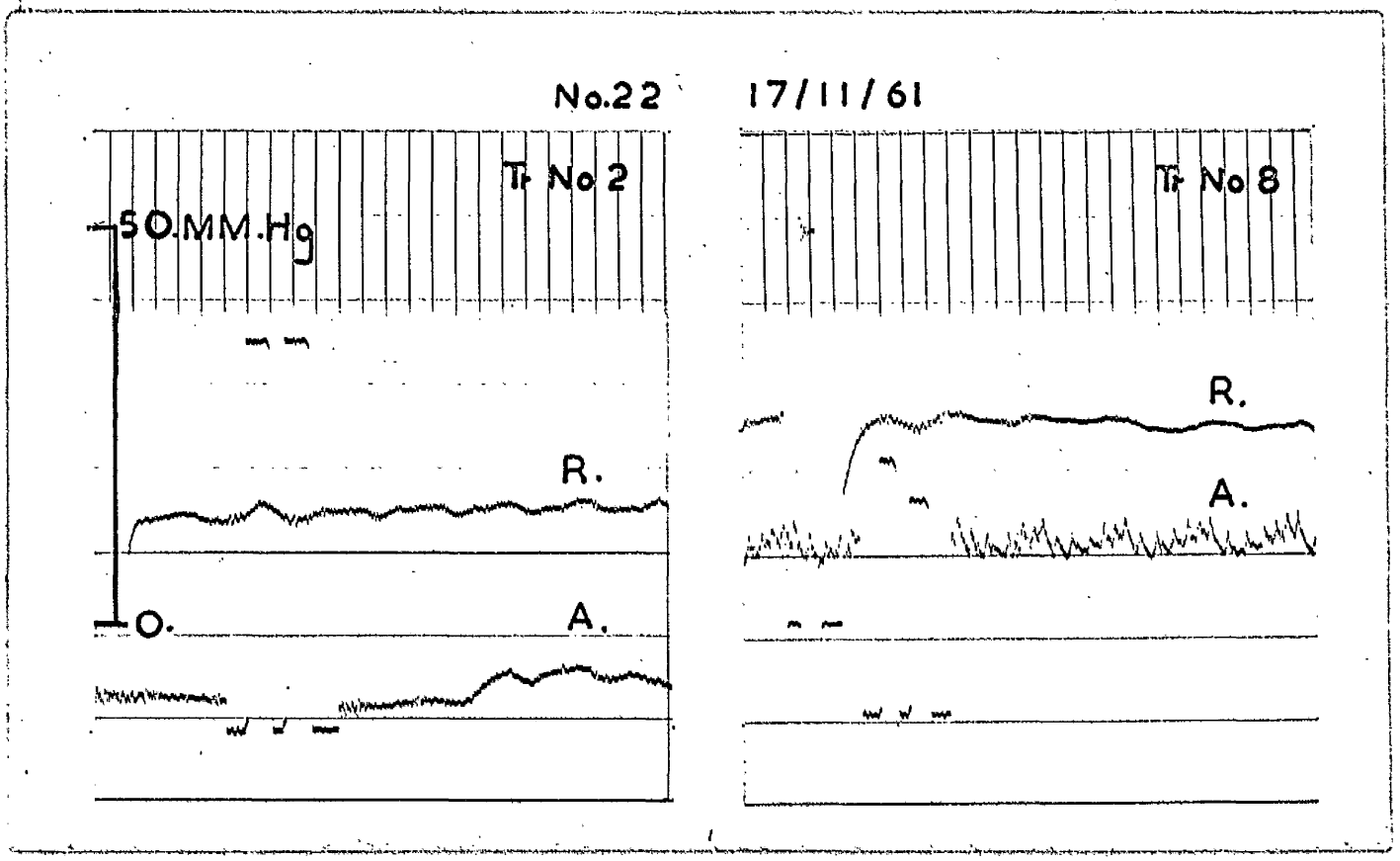


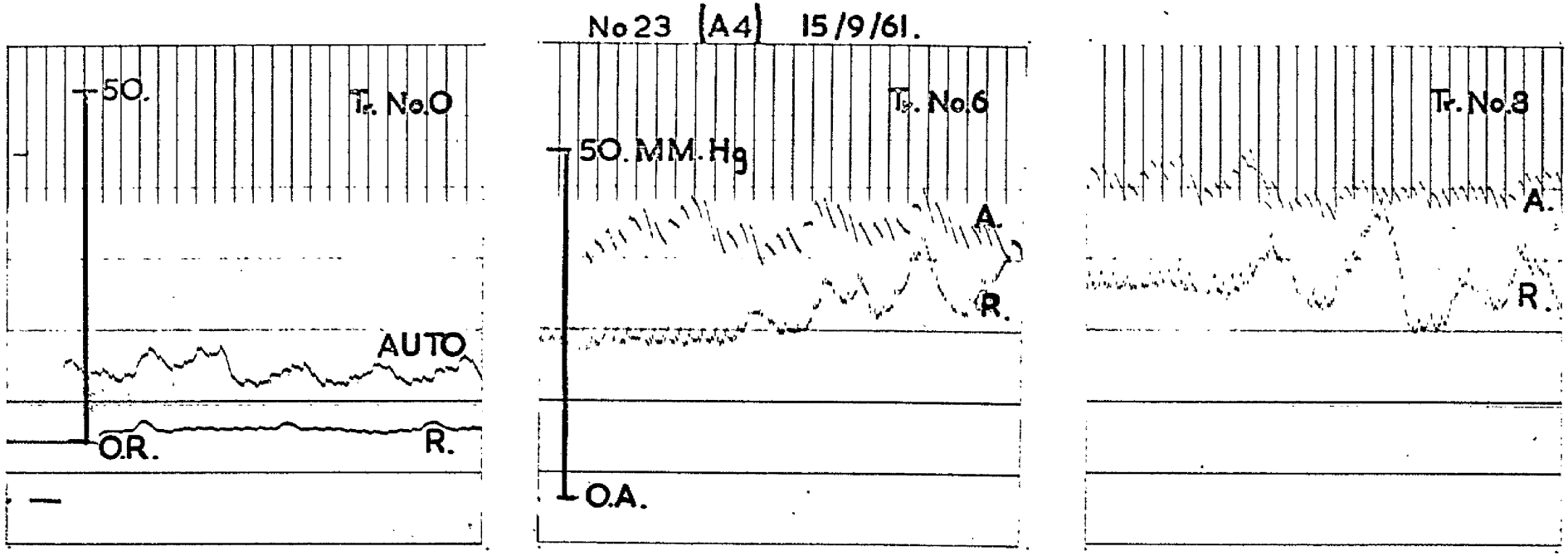
No.22 17.11.61. Right Kidney Osmotic Diuresis

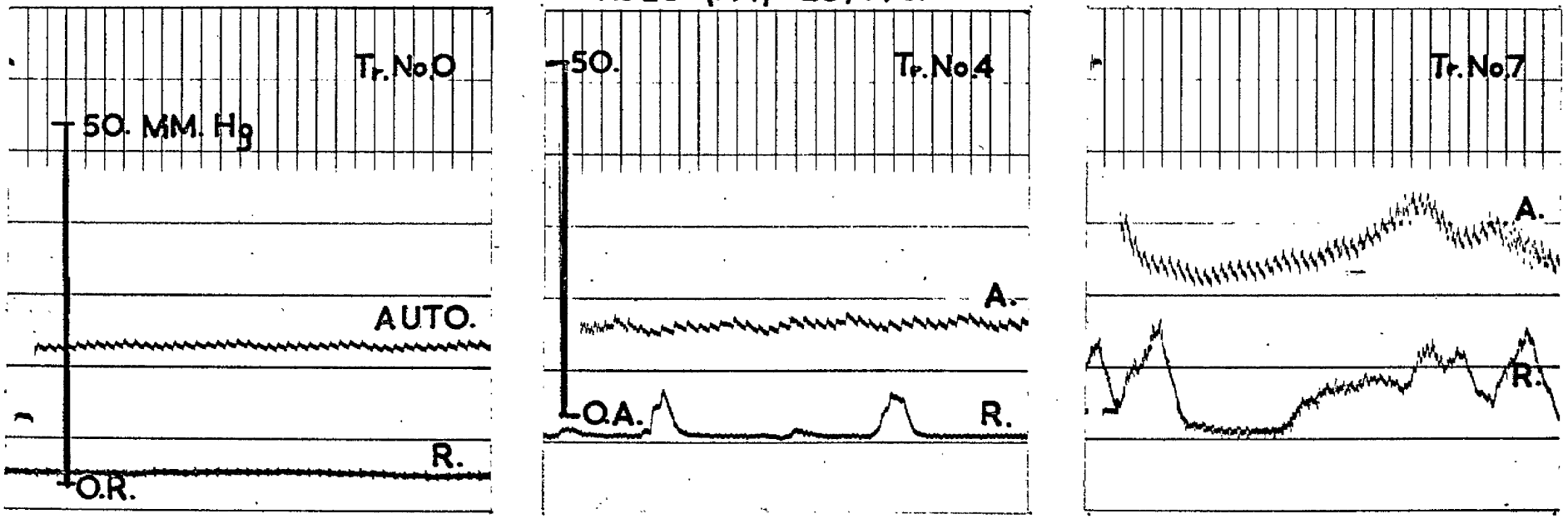
Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg. (Tr.No)
Basal	5	8 - 12 (0)
5	5	10 - 12 (1)
10	8	12 - 15 (2)
15	10	14 - 16 (3)
20	10	12 - 13 (4)
25	3	5 - 9 (5)
30	8	11 - 12 (6)
35	10	13 - 17 (7)
40	15	22 - 26 (8)
45	15	24 - 28 (9)

Pressure Tracing — 20 sec





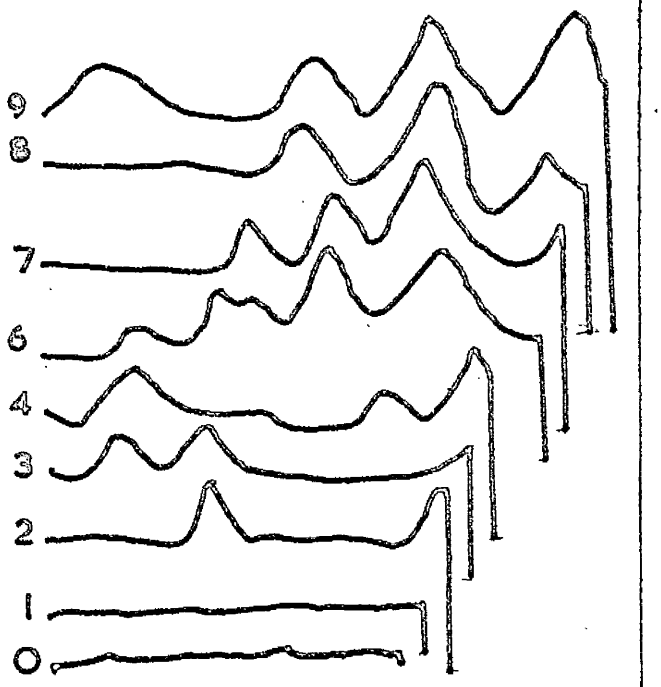

50.
Tr. No.0
AUTO.
O.R.
R.
No 23 (A4) 15/9/61.
Tr. No.6
50. MM. Hg
A.
R.
O.A.
Tr. No.3
A.
R.


Tr. No.0
50. MM. Hg
AUTO.
R.
O.R.
No 23 (A4) 25/9/61
50.
Tr. No.4
A.
O.A.
R.
Tr. No.7
A.
R.

No.23 15.9.61 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)
Basal	5	2 - 3 (0)
5	5	6 (1)
10	8	15 - 25 (2)
15	9	12 - 18 (3)
20	9	13 - 23 (4)
25	-	- (5)
30	15	13 - 26 (6)
35	24	20 - 32 (7)
40	28	15 - 31 (8)
45	29	25 - 38 (9)

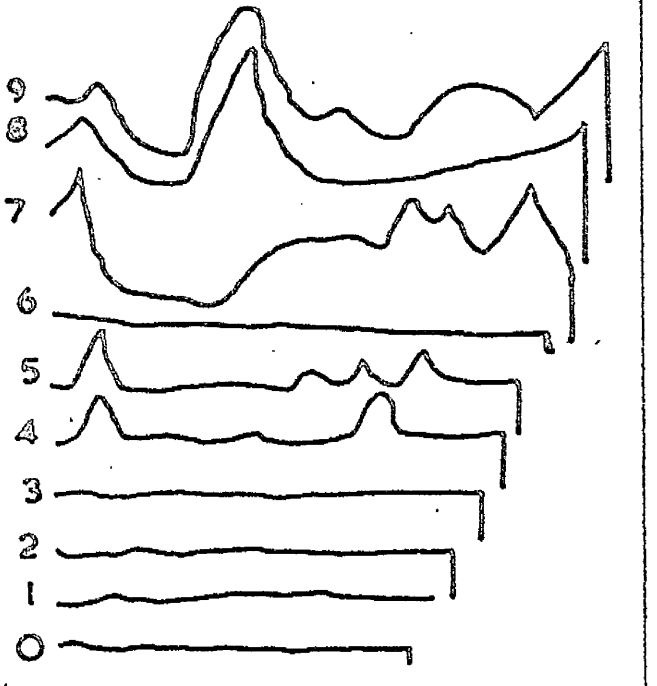
Pressure Tracing — 20 sec

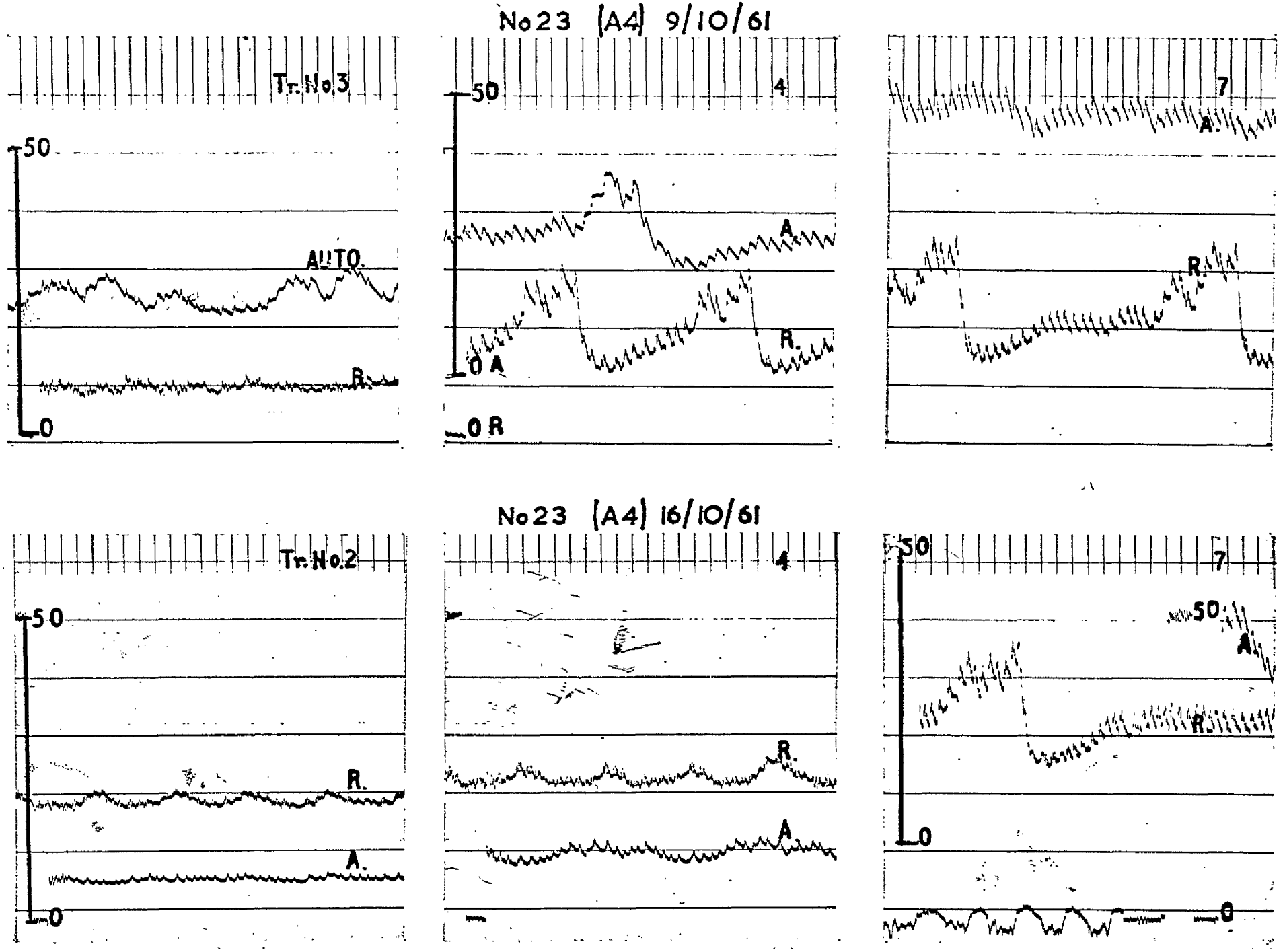


No.23 25.9.61 Right Kidney Osmotic Diuresis

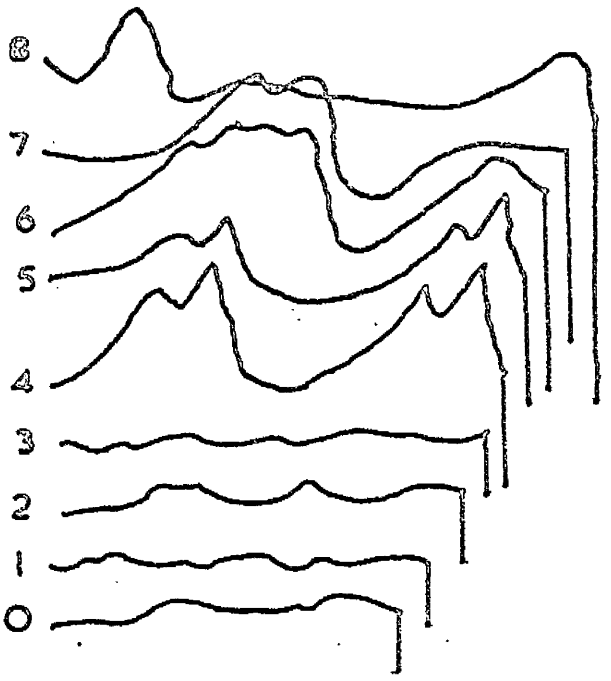
Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)
Basal	0	2 (0)
5	2	0 - 1 (1)
10	3	5 (2)
15	3	6 (3)
20	5	6 - 12 (4)
25	5	7 - 13 (5)
30	6	2 - 3 (6)
35	11	5 - 21 (7)
40	11	10 - 26 (8)
45	11	4 - 22 (9)

Pressure Tracing — 20 sec

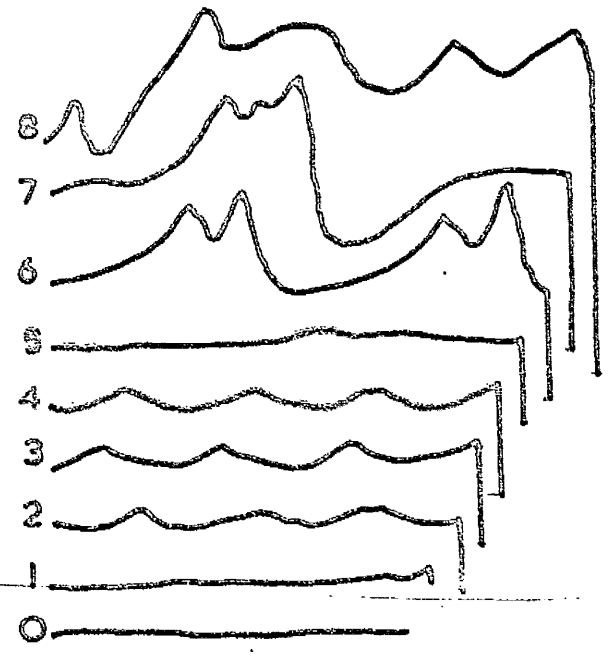



No 23 [A4] 9/10/61
Tr. No 3
50
AUTO.
R.
0
50
4
A
R.
0 A
0 R
7
A.
R.
No 23 [A4] 16/10/61
Tr. No 2
50
R.
A.
0
R.
A
50
7
50
A.
R.
0
0

No.23 9.10.61 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	0	6 - 10 (0)	
5	4	7 - 10 (1)	
10	6	7 - 11 (2)	
15	7	6 - 10 (3)	
20	11	11 - 28 (4)	
25	12	12 - 25 (5)	
30	13	12 - 33 (6)	
35	16	16 - 34 (7)	
40	22 - 25	33 - 49 (8)	
45	-	- (9)	

No.23 16.10.61 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	0	0 (0)	
5	0	0 - 2 (1)	
10	4	8 - 11 (2)	
15	5	9 - 15 (3)	
20	9	10 - 15 (4)	
25	11	10 - 11 (5)	
30	14	12 - 27 (6)	
35	18	12 - 35 (7)	
40	20	25 - 44 (8)	
45	-	- (9)	

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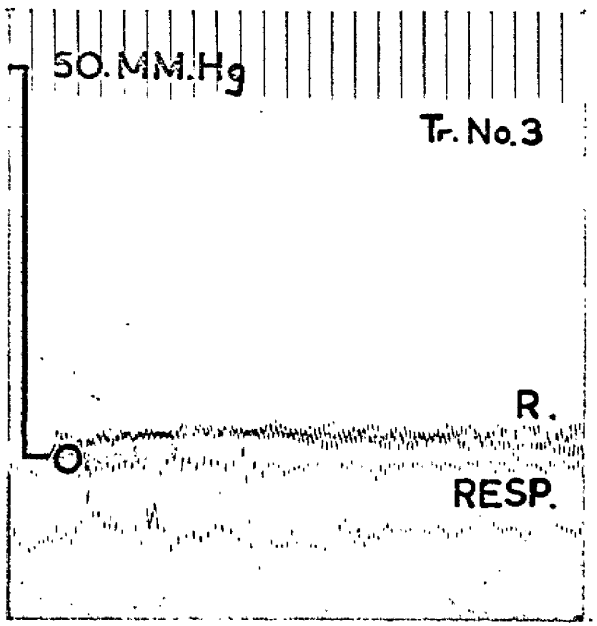
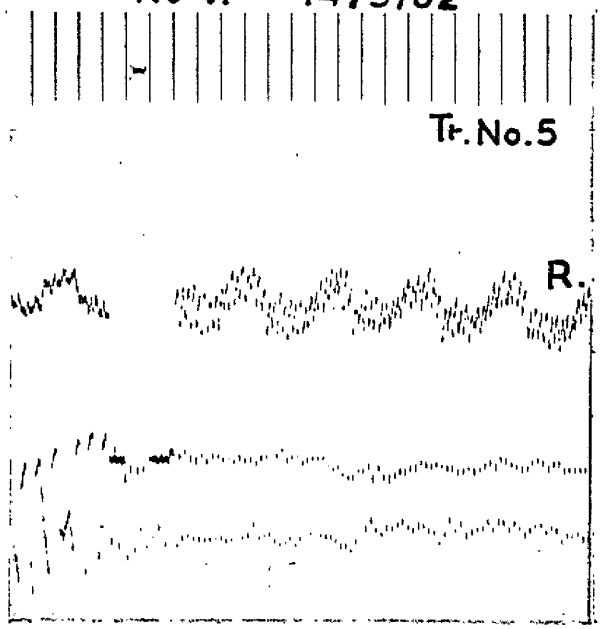
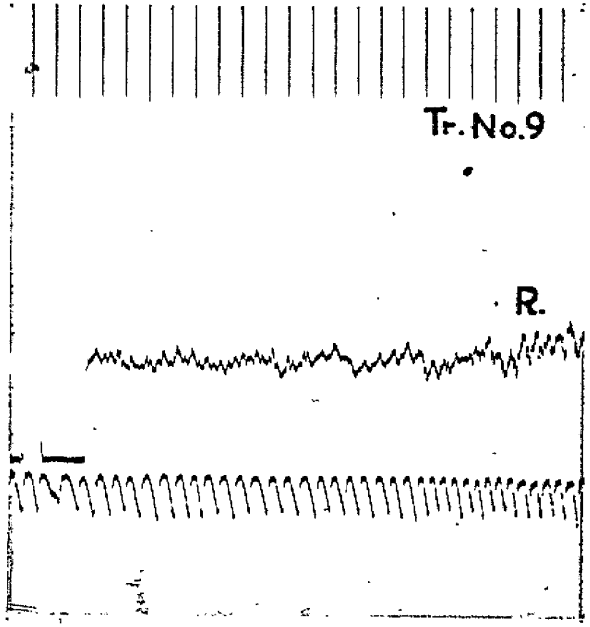
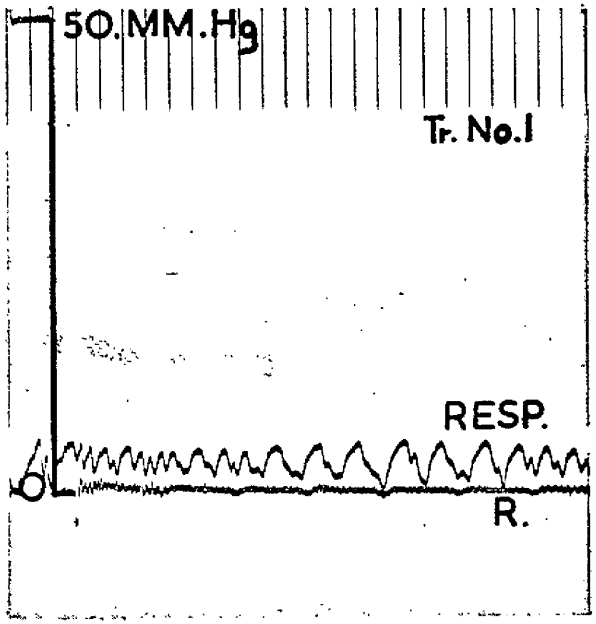
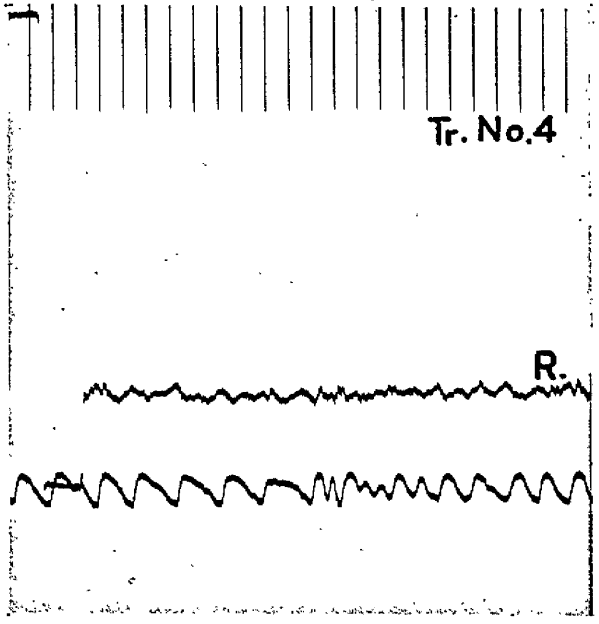


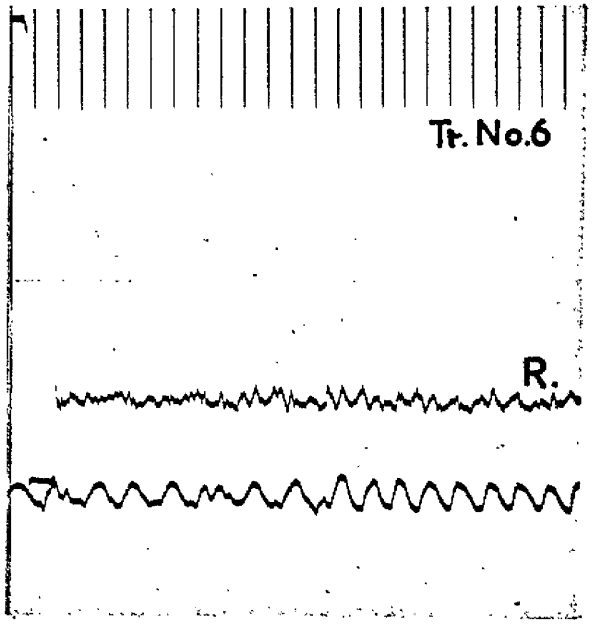




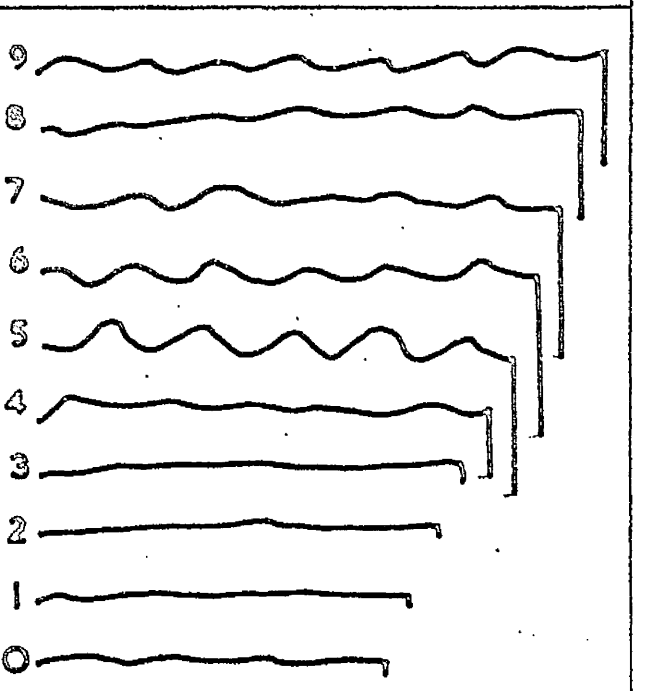
No 41 18/5/62



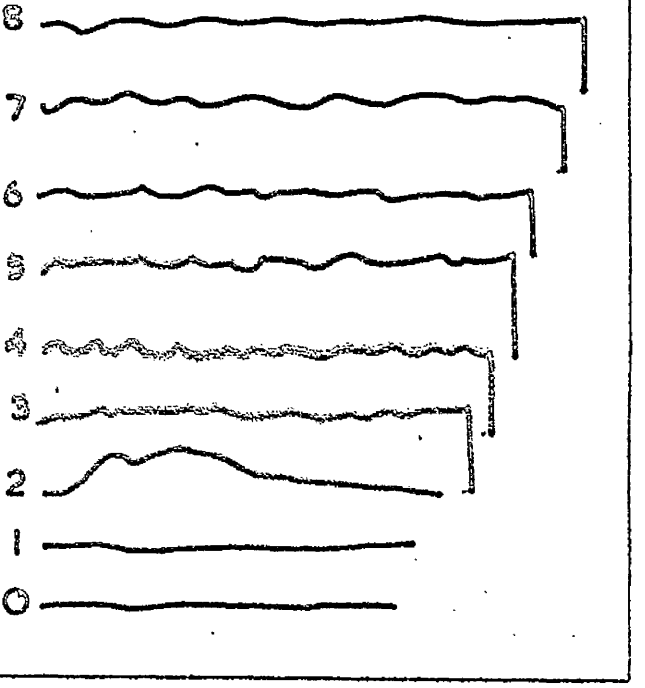




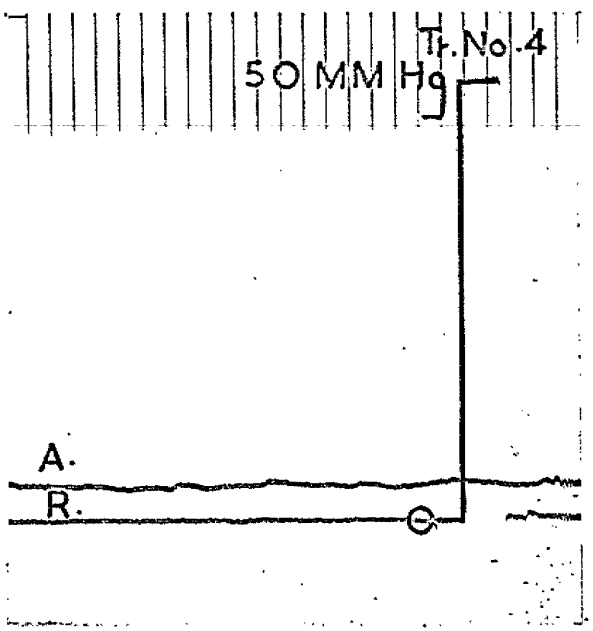
No.41 14.5.62 Right Kidney Osmotic Diuresis

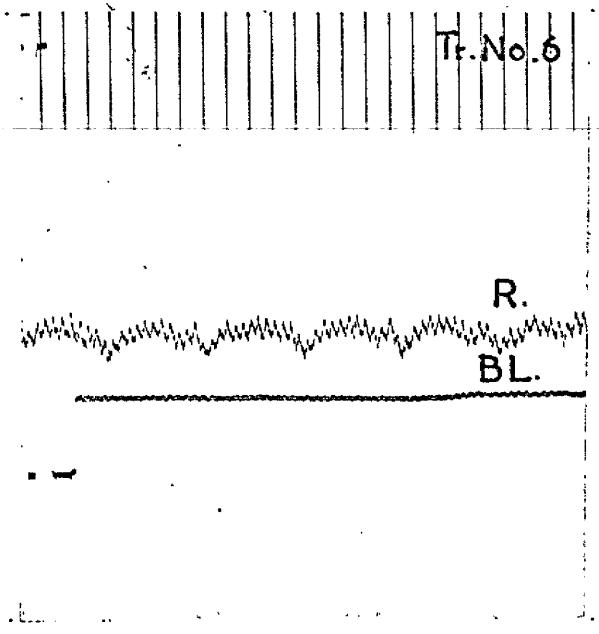
Time (Mins)	Urine/5 min R. Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec
Basal	- mls	0 - 1 (0)	
5	10	1 (1)	
10	13	2 (2)	
15	10	2 (3)	
20	37	6 - 11 (4)	
25	33	12 - 20 (5)	
30	37	17 - 23 (6)	
35	42	15 - 21 (7)	
40	37	10 - 14 (8)	
45	28	11 - 15 (9)	

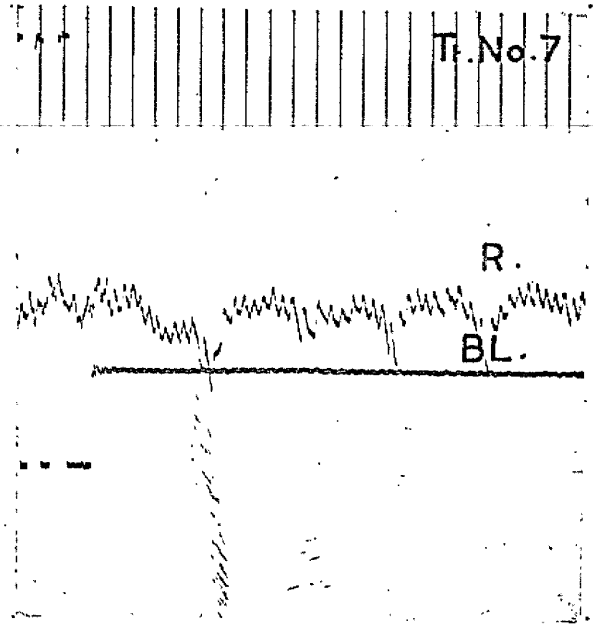
No.41 18.5.62 Right Kidney Water Diuresis

Time (Mins)	Urine/5 min R. Kid.	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec
Basal	- mls	0 (0)	
5	11	0 (1)	
10	12	6 (2)	
15	13	7 - 10 (3)	
20	14	9 - 11 (4)	
25	17	10 - 12 (5)	
30	24	8 - 10 (6)	
35	17	7 - 11 (7)	
40	10	7 - 10 (8)	
45	-	- (9)	

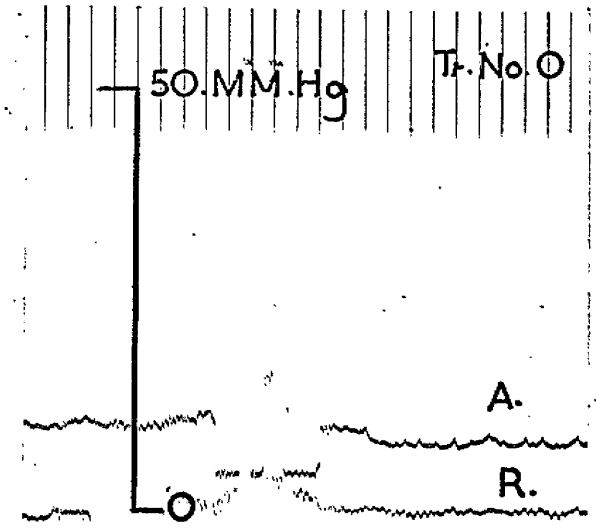
No. 43 25/5/62

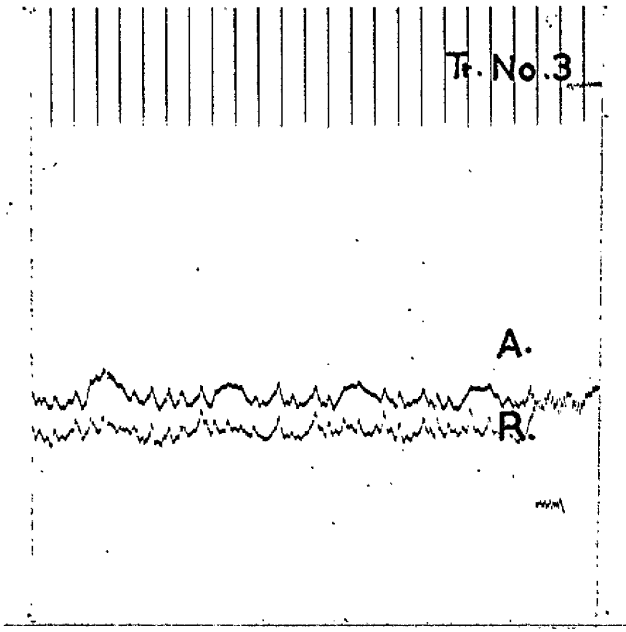


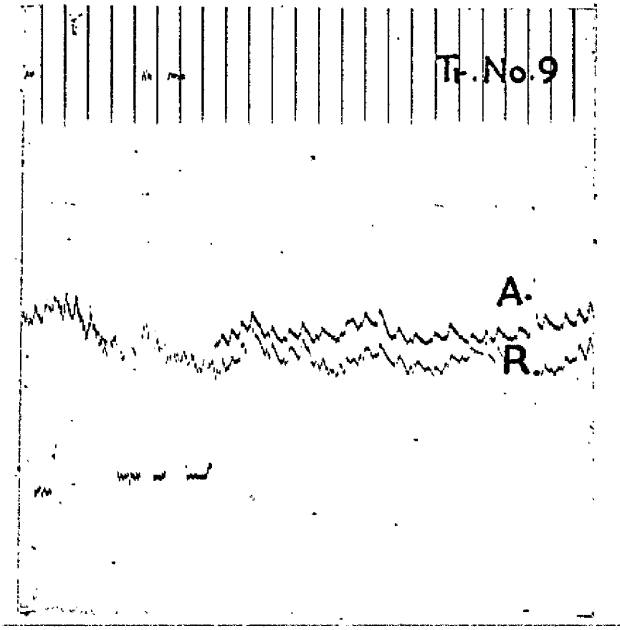




No. 43 25/6/62

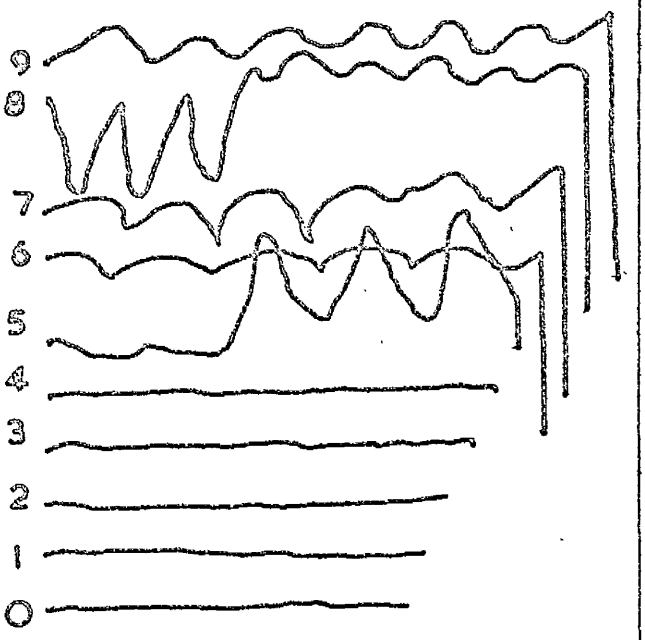






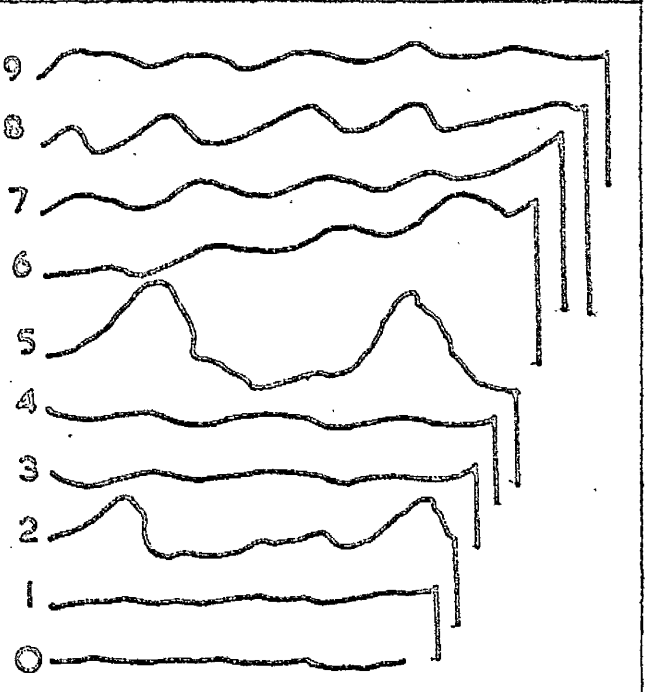
No.43 25.5.62 Right Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	5	0 - 0 (0)	
5	5	0 - 0 (1)	
10	6	0 - 0 (2)	
15	7	0 - 0 (3)	
20	7	0 - 0 (4)	
25	8	1 - 18 (5)	
30	8	19 - 23 (6)	
35	11	18 - 27 (7)	
40	15	15 - 30 (8)	
45	14	26 - 32 (9)	

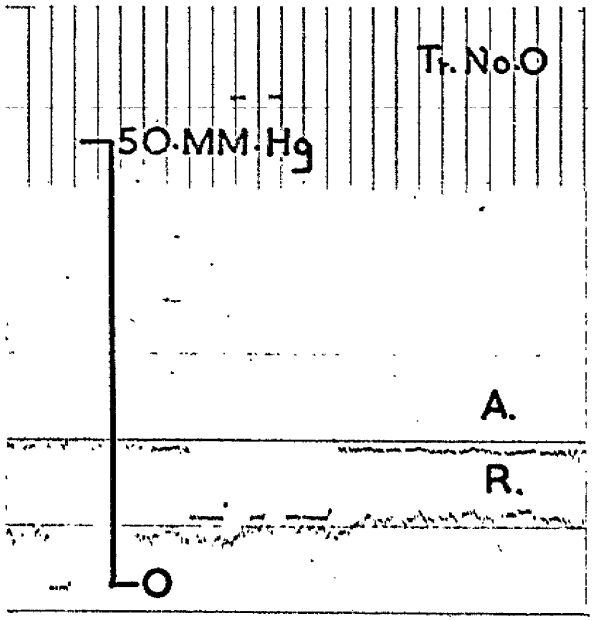


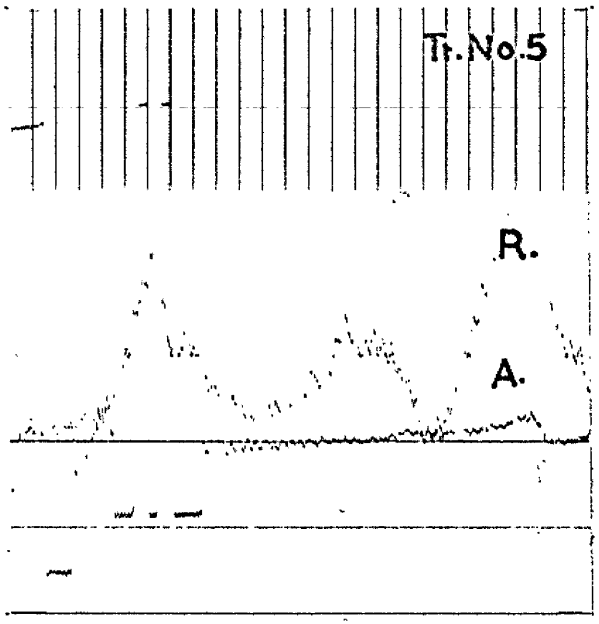
No.43 25.6.52 Right Kidney Osmotic Diuresis

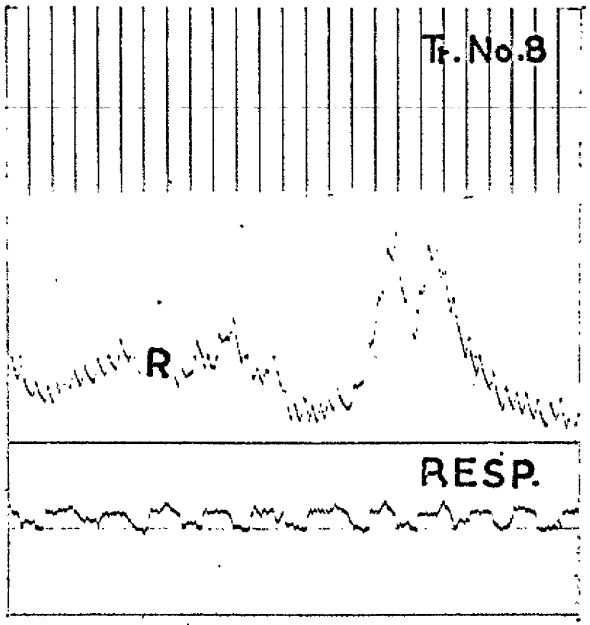
Time (Mins)	Urine/5 min Auto & R.Kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 - 1 (0)	
5	12	6 - 9 (1)	
10	30	8 - 15 (2)	
15	39	6 - 10 (3)	
20	46	7 - 9 (4)	
25	52	10 - 25 (5)	
30	68	11 - 22 (6)	
35	78	11 - 21 (7)	
40	96	20 - 27 (8)	
45	72	15 - 19 (9)	



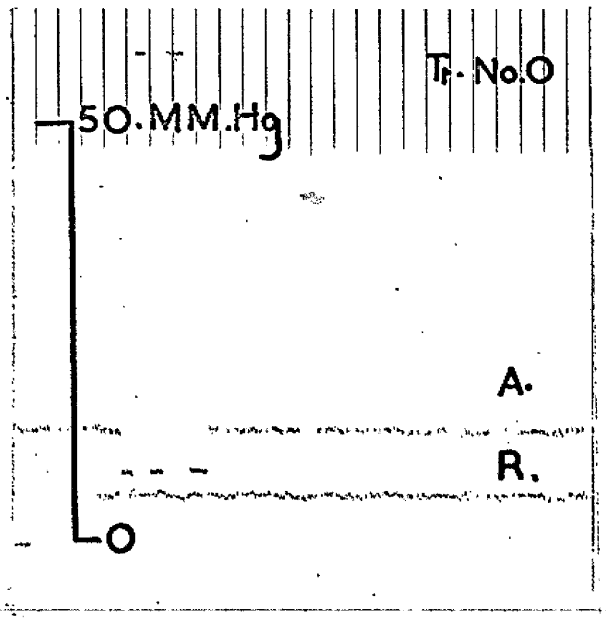
No. 43 19/7/62

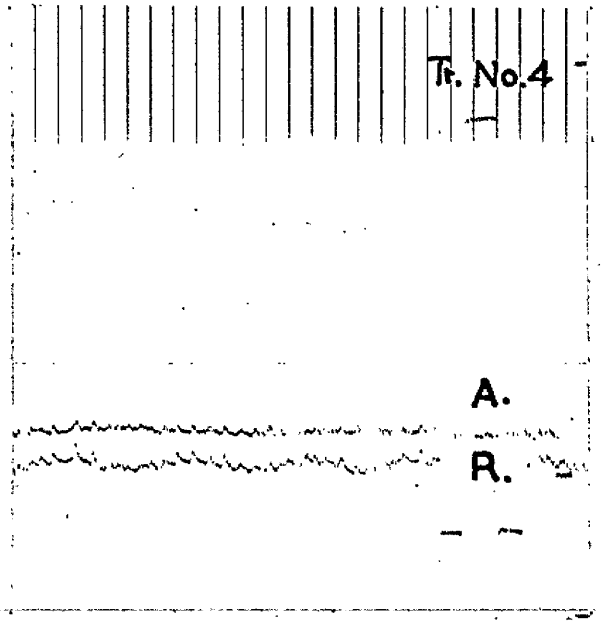


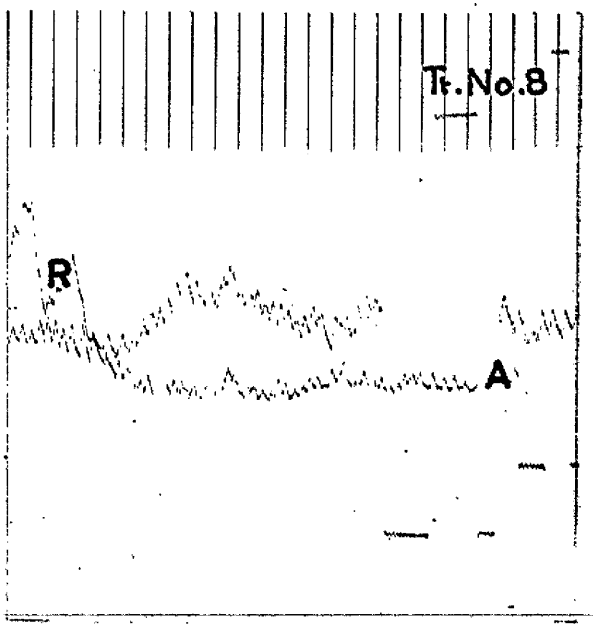




No. 43 16/8/62



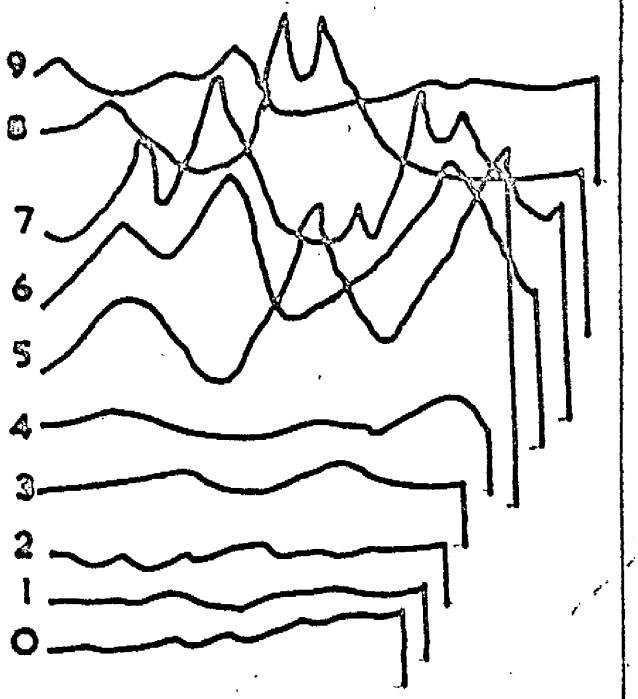




No.43 19.7.62 Rt.Kidney - Ureteric Implant Osmotic Diuresis

Time (Mins)	Urine/5 min Auto & R.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	4 - 9 (0)
5	9	6 - 9 (1)
10	20	5 - 9 (2)
15	23	6 - 11 (3)
20	28	6 - 12 (4)
25	32	14 - 42 (5)
30	52	15 - 34 (6)
35	60	21 - 41 (7)
40	69	16 - 40 (8)
45	65	10 - 17 (9)

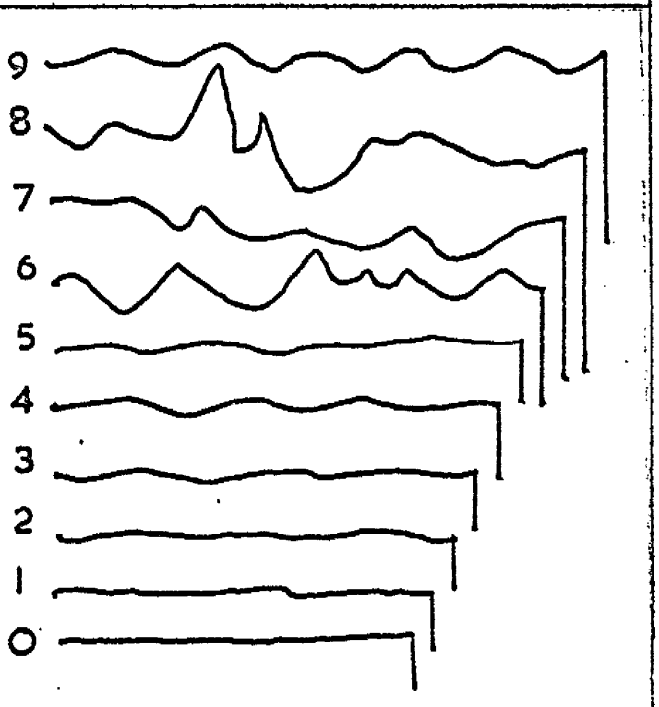
Pressure Tracing — 20 sec



No.43 16.8.63 Rt. Kidney - Ureteric Implant Osmotic Diuresis

Time (Mins)	Urine 15 min. Auto & Rt. Kid.	Pelv. Press mm Hg. (Tr.No)
Basal	- mls.	6 (0)
5	18	6-8 (1)
10	28	6-7 (2)
15	33	6-8 (3)
20	39.	7-10 (4)
25	55	7-9 (5)
30	65	11-18 (6)
35	70	13-22 (7)
40	88	22-37 (8)
45	103	19-24 (9)

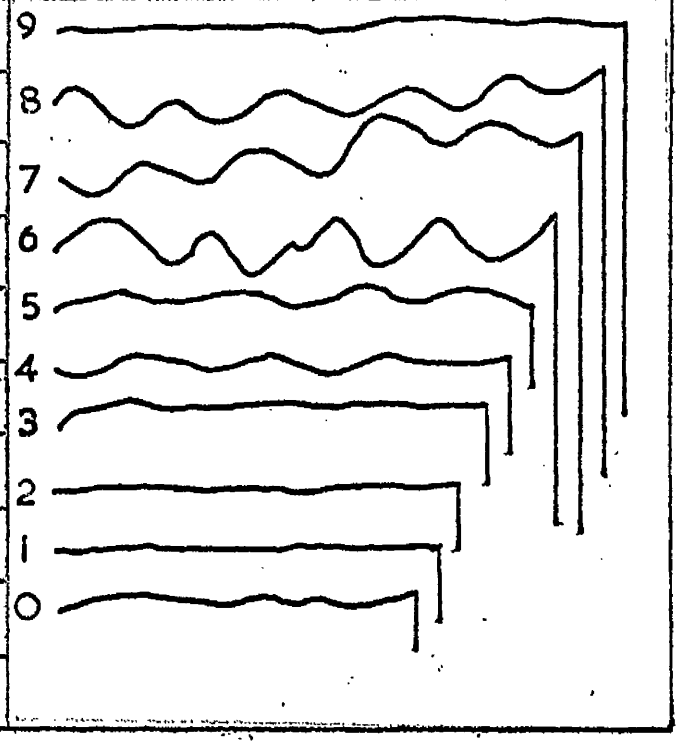
Pressure Tracing — 20 sec

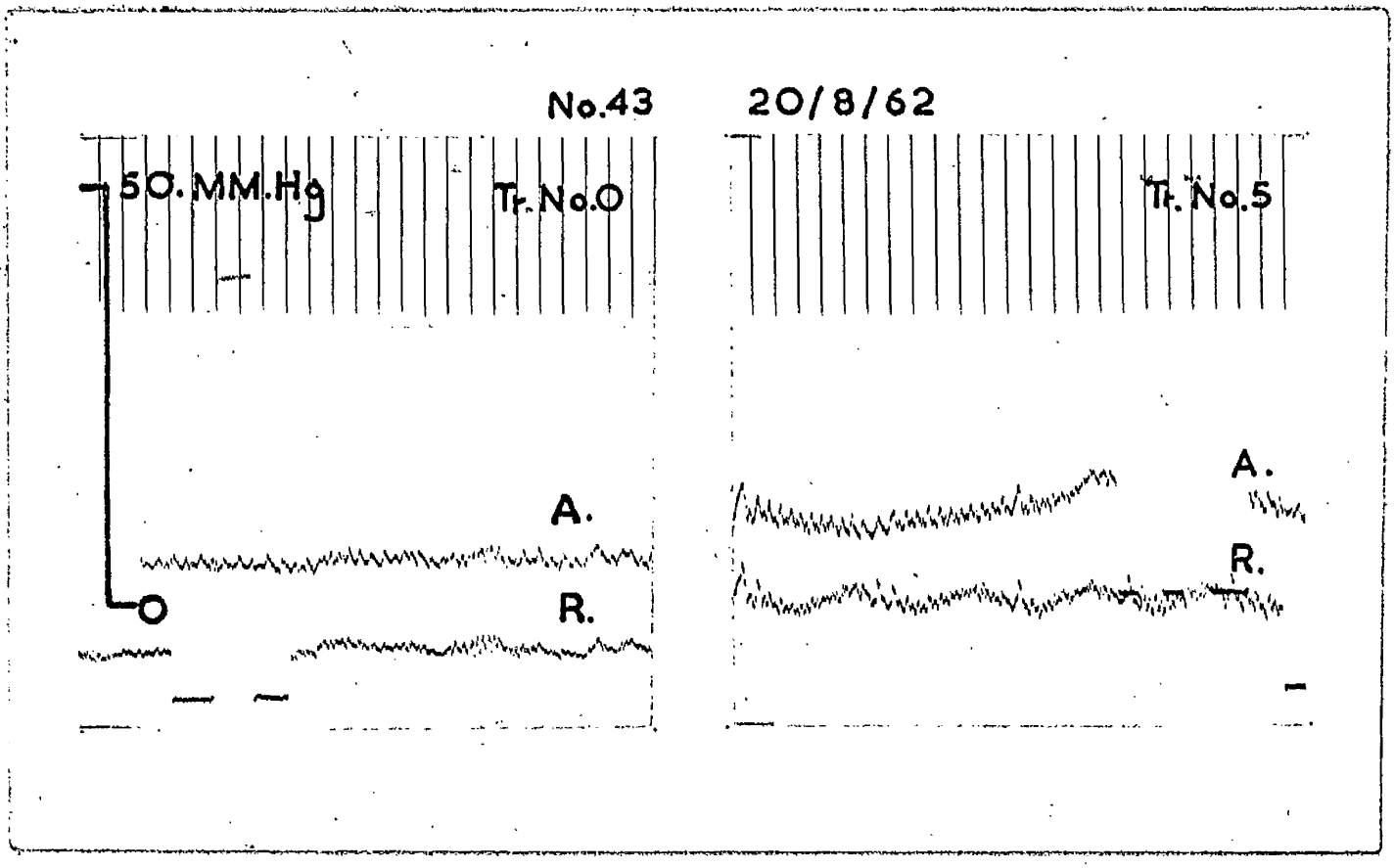


No.43 20.8.62 Rt. Kidney - Ureteric Implant Osmotic Diuresis

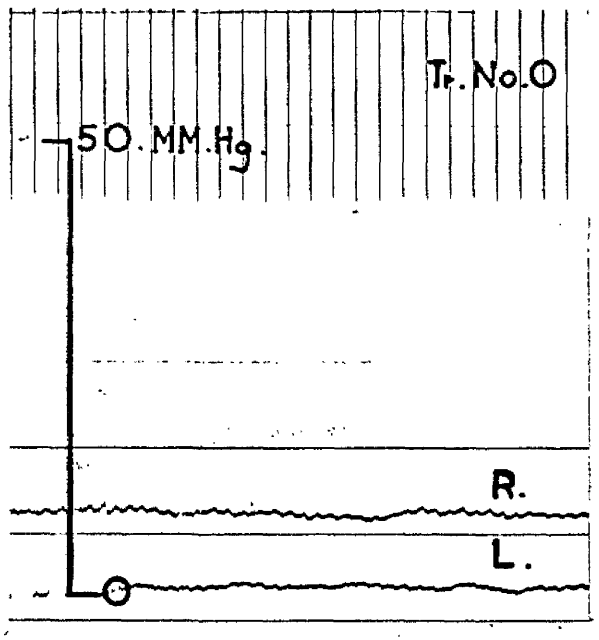
Time (Mins)	Blad. Press. mm. Hg.	Pelv. Press mm Hg. (Tr.No)
Basal	6	4-6 (0)
5	7	7-10 (1)
10	6	8-9 (2)
15	8	8-10 (3)
20	10	9-12 (4)
25	14	9-12 (5)
30	18	30-36 (6)
35	32	40-50 (7)
40	33	42-48 (8)
45	48	46-48 (9)

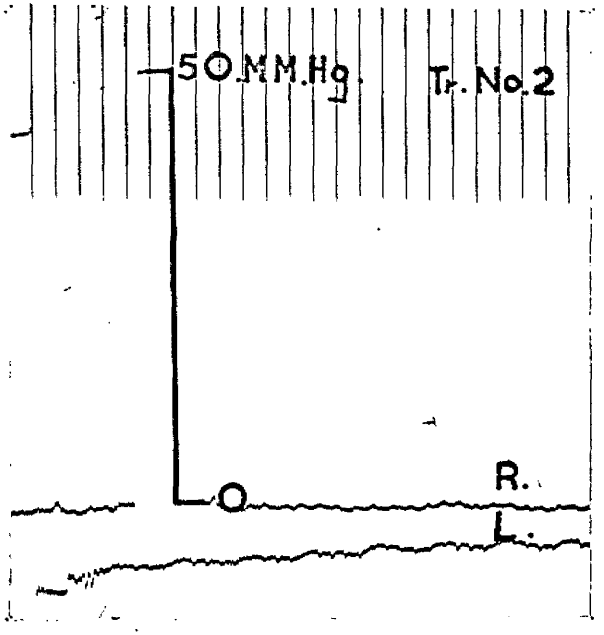
Pressure Tracing — 20 sec

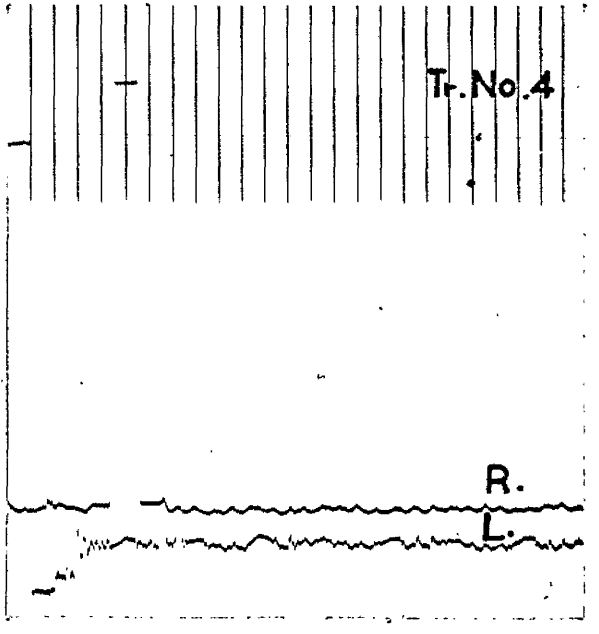




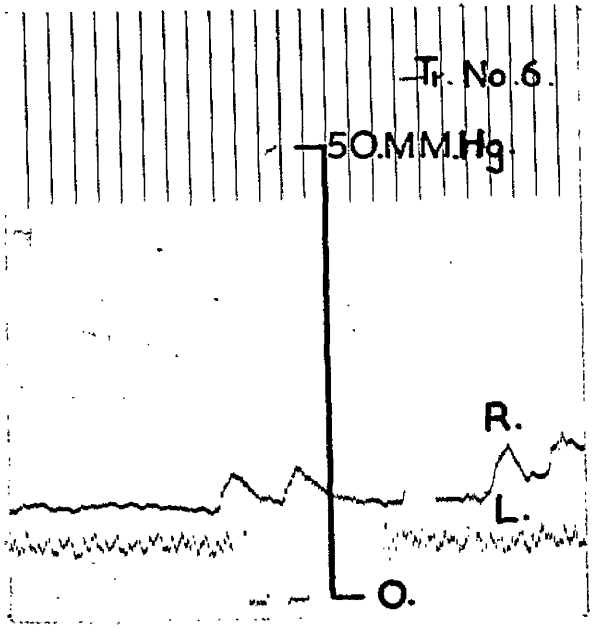
No 45. 19/7/62

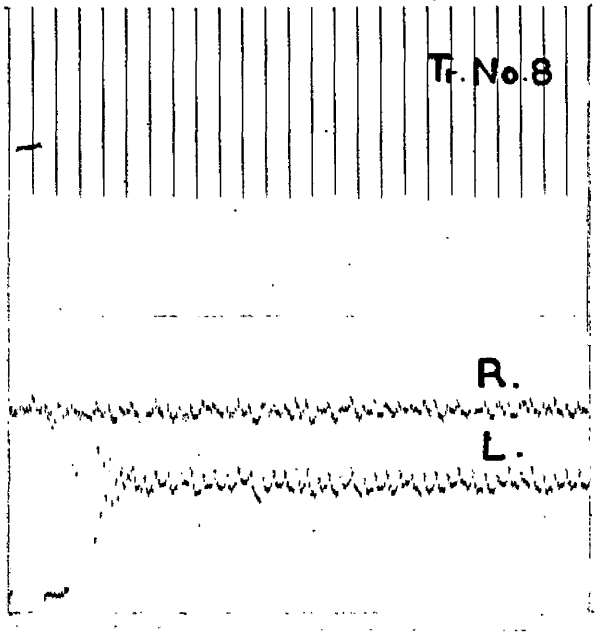


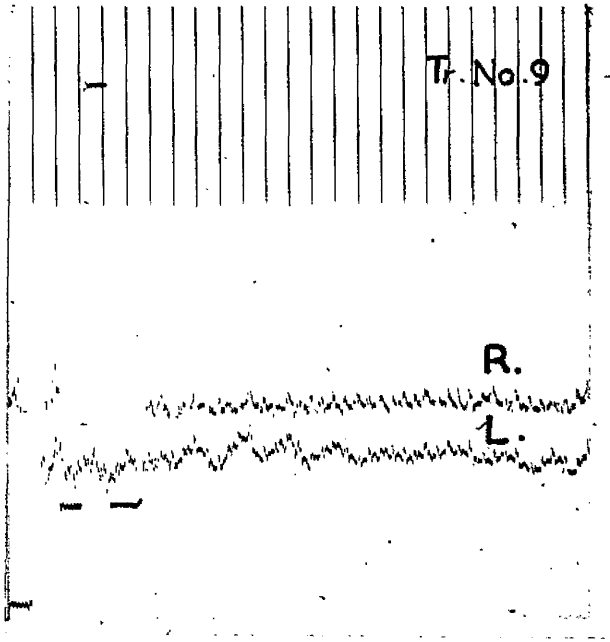




No 45. 19/7/62



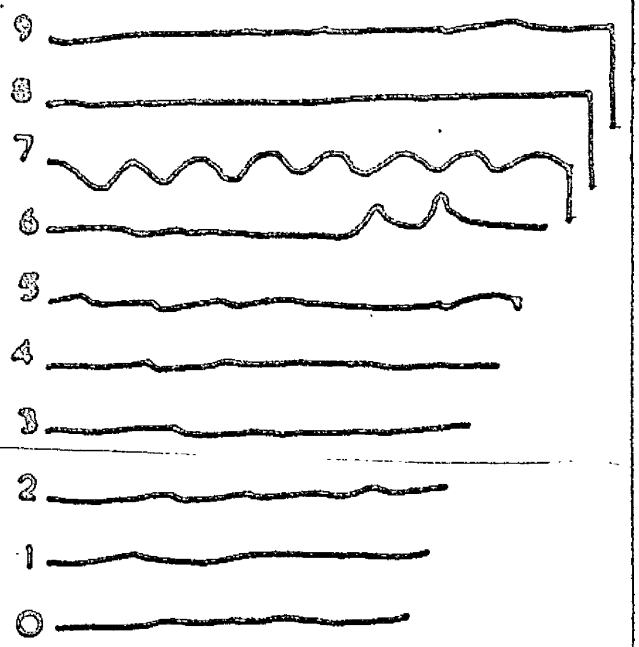




No.45 19.7.62 Rt.Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min R & L.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 2 (0)
5	2	0 - 3 (1)
10	2	1 - 2 (2)
15	10	0 - 1 (3)
20	14	0 - 1 (4)
25	28	3 - 4 (5)
30	33	0 - 5 (6)
35	38	3 - 9 (7)
40	32	10 - 11 (8)
45	29	11 - 13 (9)

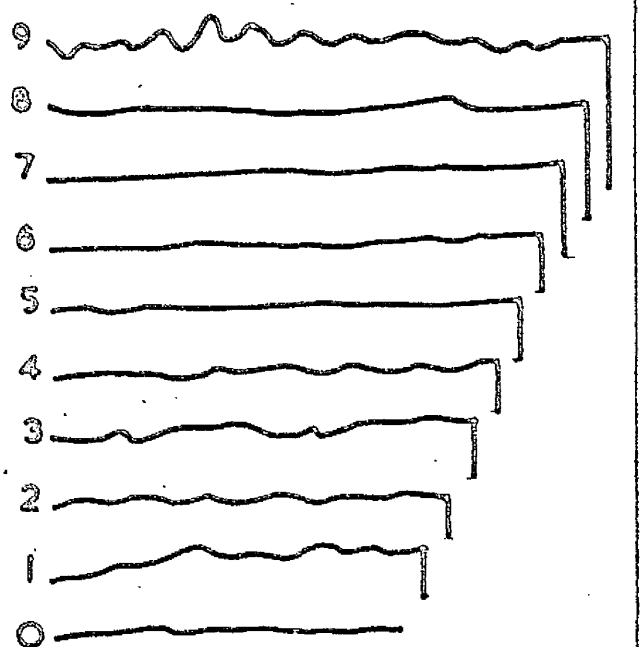
Pressure Tracing
20 sec



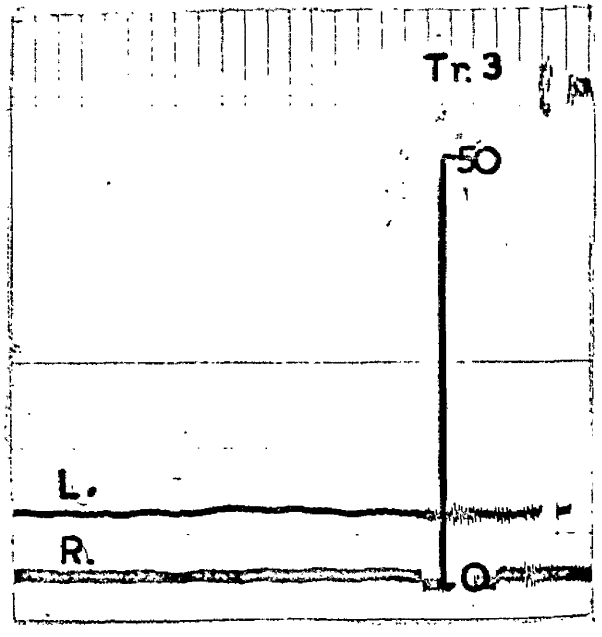
No.45 19.7.62 Lt.Kidney Osmotic Diuresis

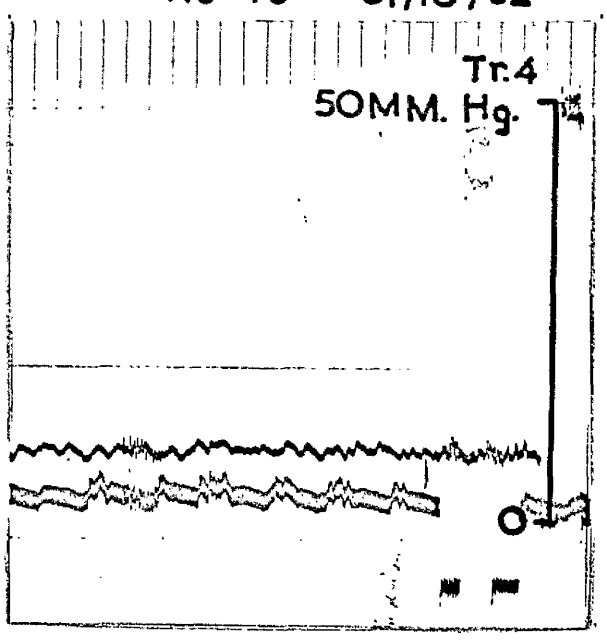
Time (Mins)	Urine/5 min R & L Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	0 - 0 (0)
5	2	5 - 9 (1)
10	2	5 - 7 (2)
15	10	5 - 8 (3)
20	14	5 - 7 (4)
25	28	5 - 6 (5)
30	33	6 - 7 (6)
35	38	11 - 12 (7)
40	32	12 - 15 (8)
45	29	15 - 20 (9)

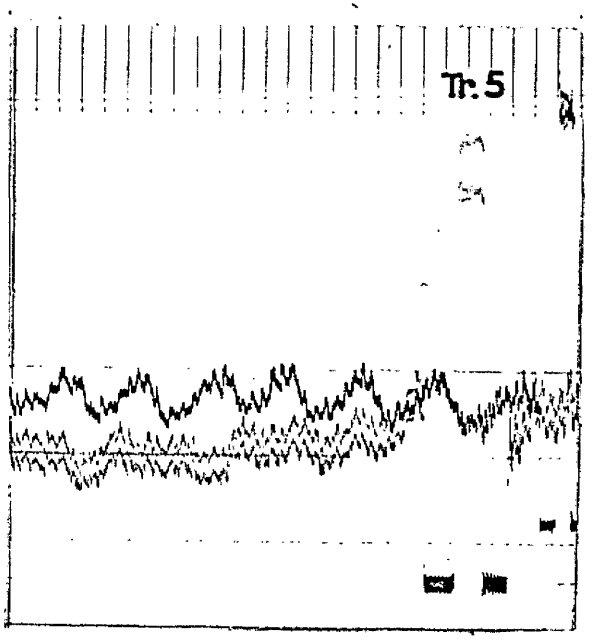
Pressure Tracing
20 sec



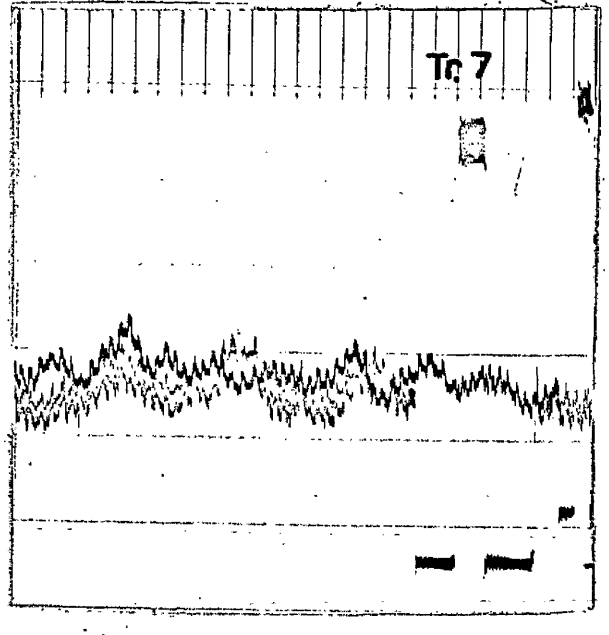
No 46 31/10/62

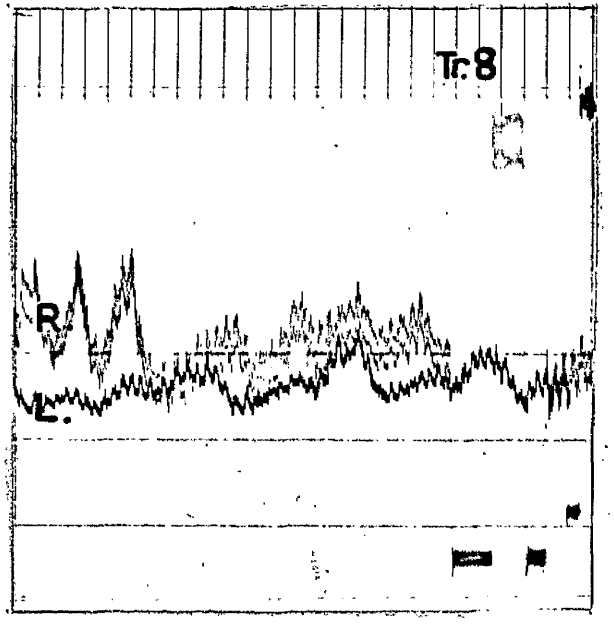


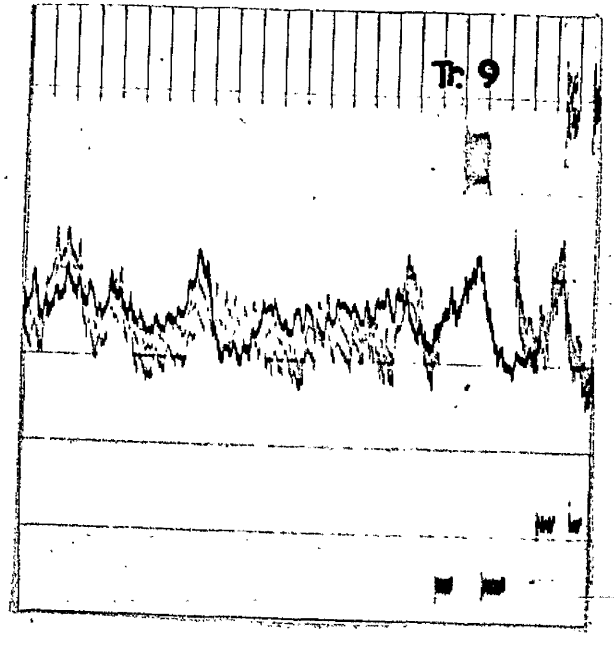




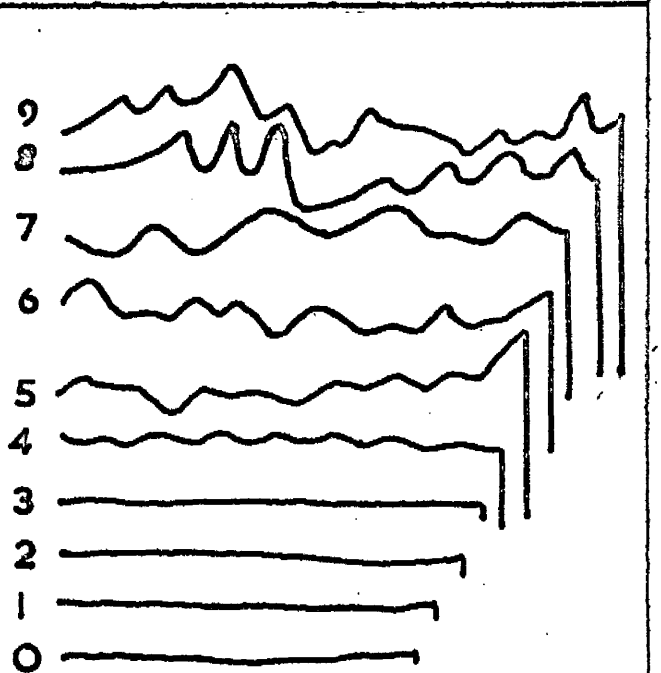
No 46 31/10/62



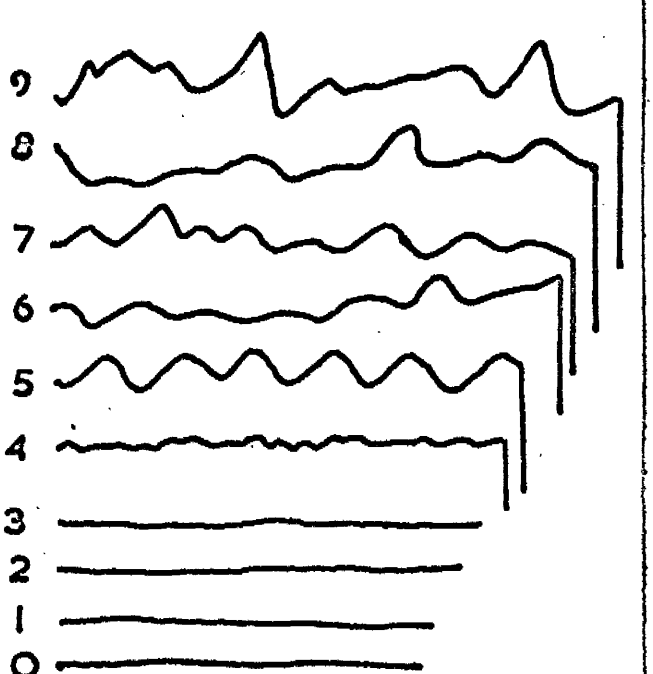




No.46 31.10,62 Right Kidney Osmotic Diuresis

Time (Mins)	Blad. Press. mm. Hg.	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec
Basal	4	1 (0)	
5	5	1 (1)	
10	5	2 (2)	
15	5	2 (3)	
20	5	8-11 (4)	
25	6	12-22 (5)	
30	7	12-20 (6)	
35	8	15-23 (7)	
40	9	20-30 (8)	
45	9	24-35 (9)	

No.46 31.10.62 Left Kidney Osmotic Diuresis

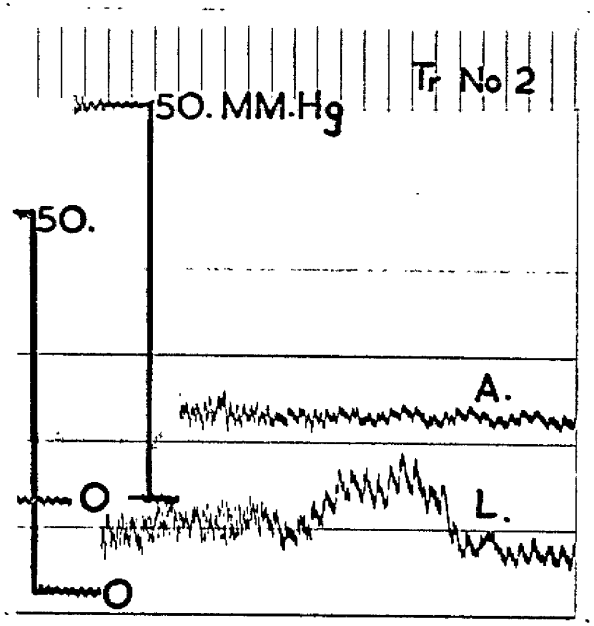
Time (Mins)	Blad. Press. mm. Hg	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec
Basal	4	0 (0)	
5	5	0 (1)	
10	5	0 (2)	
15	5	0 (3)	
20	5	6-8 (4)	
25	6	11-16 (5)	
30	7	11-17 (6)	
35	8	13-20 (7)	
40	9	18-24 (8)	
45	9	18-27 (9)	

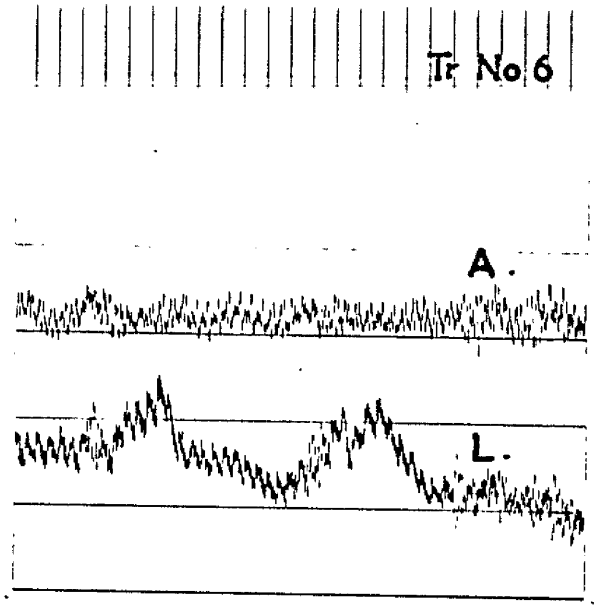
PROTOCOLS TO EXPERIMENTS

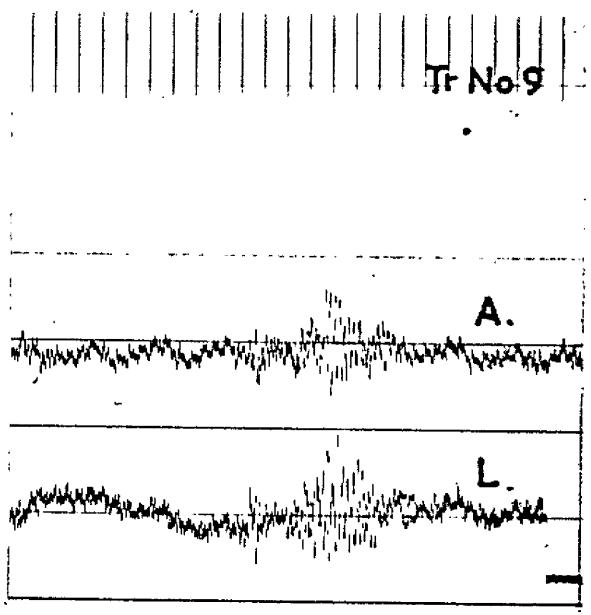
B. PRESSURE RECORDINGS

ii) Autotransplanted kidneys

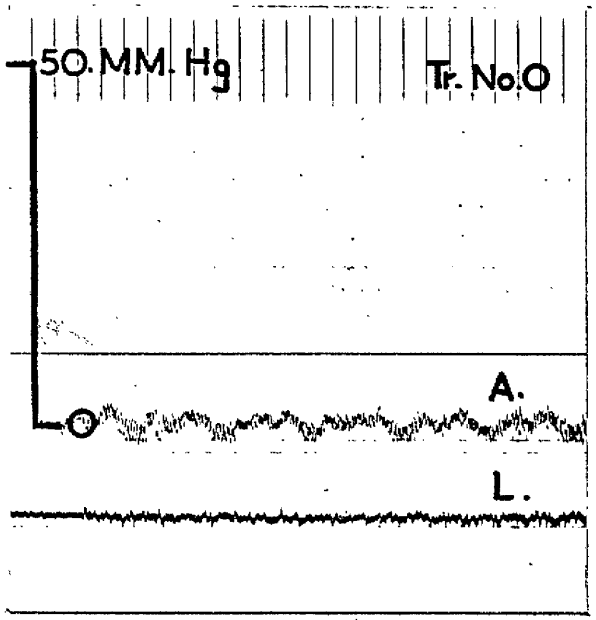
No 17 (A2) 9/6/61

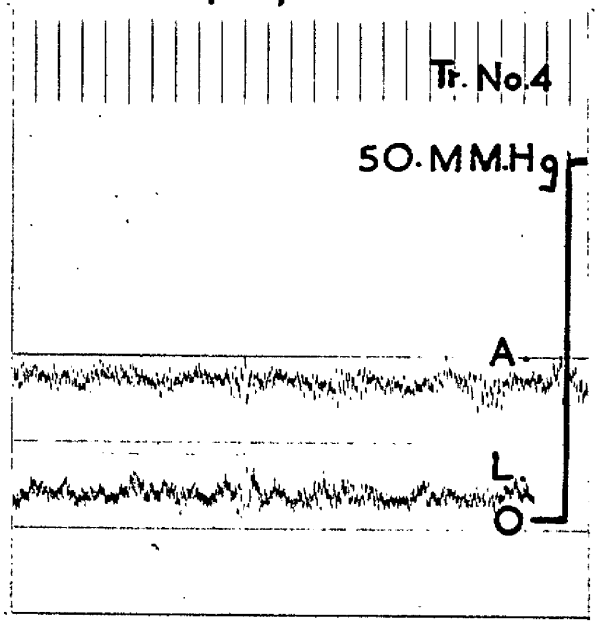


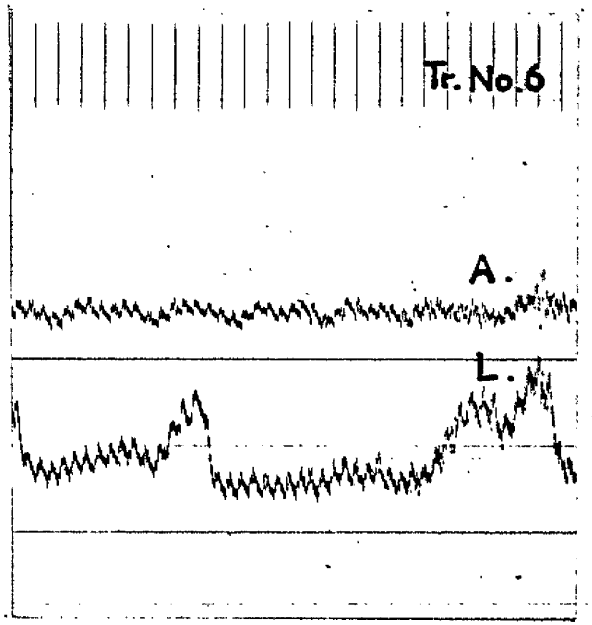




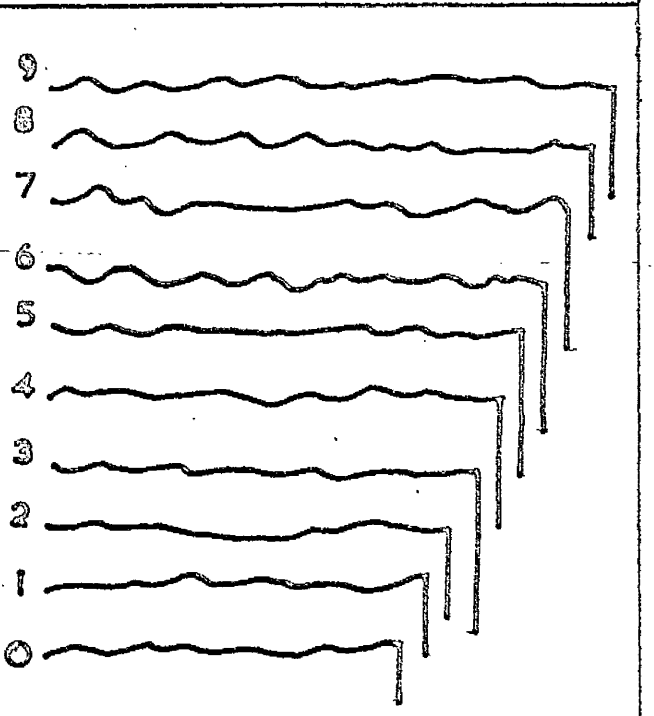
No 17 (A2) 16/6/61



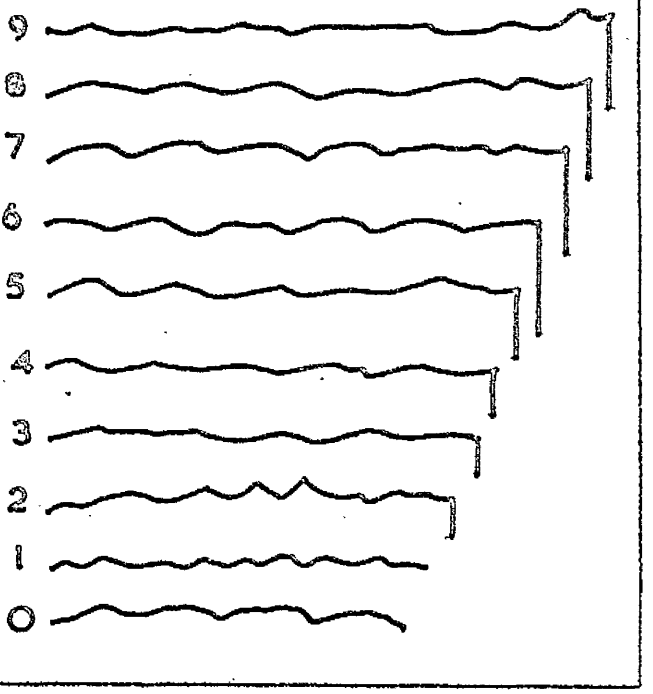




A.2. (No.17) 9.6.61 Auto Kidney Osmotic Diuresis

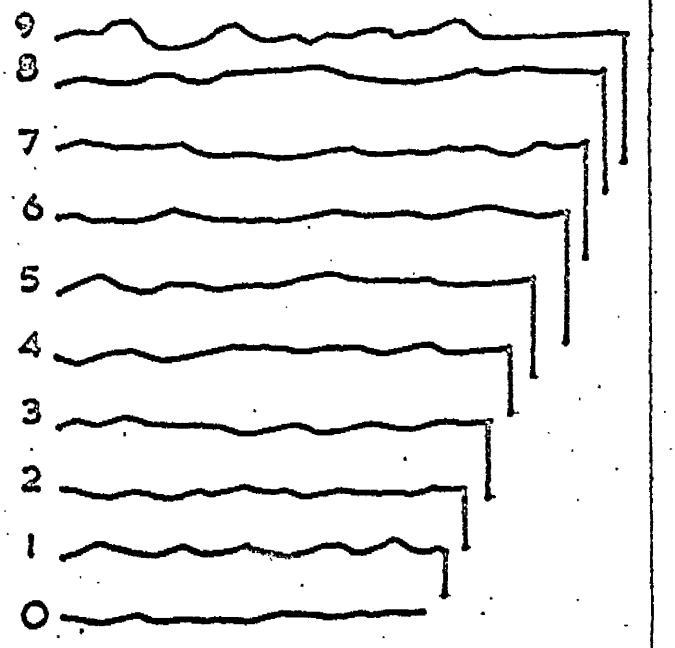
Time (Mins)	Urine/5 min auto & L kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	5 - 7 (0)	
5	23	7 - 10 (1)	
10	19	9 - 11 (2)	
15	32	17 - 19 (3)	
20	33	14 - 16 (4)	
25	44	16 - 18 (5)	
30	46	15 - 18 (6)	
35	54	15 - 18 (7)	
40	72	9 - 12 (8)	
45	70	12 - 15 (9)	

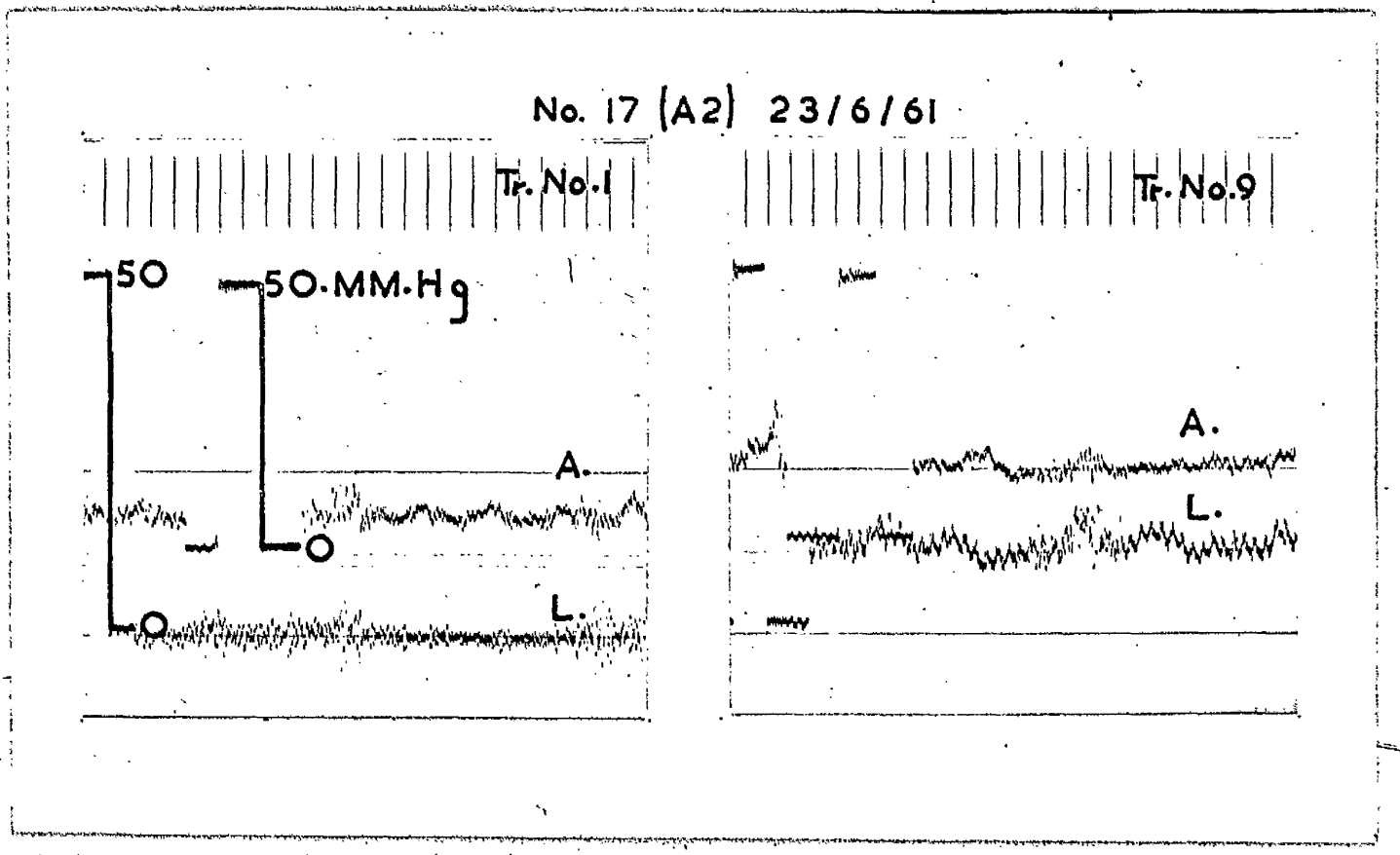
A.2 (No.17) 16.6.61 Auto Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min auto & L.kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	0 - 3 (0)	
5	14	0 - 2 (1)	
10	21	3 - 8 (2)	
15	31	3 - 5 (3)	
20	40	4 - 6 (4)	
25	49	7 - 9 (5)	
30	59	12 - 14 (6)	
35	64	7 - 13 (7)	
40	76	10 - 12 (8)	
45	60	9 - 11 (9)	

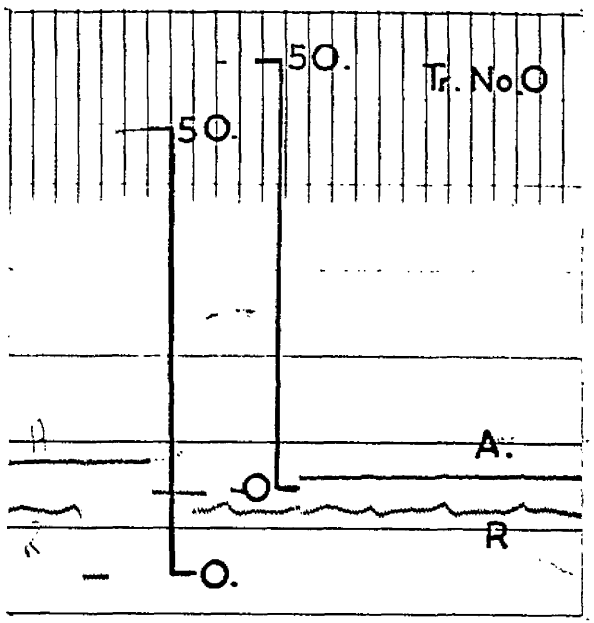
A.2. (No.17) 23.6.61 Auto.Kidney Osmotic Diuresis

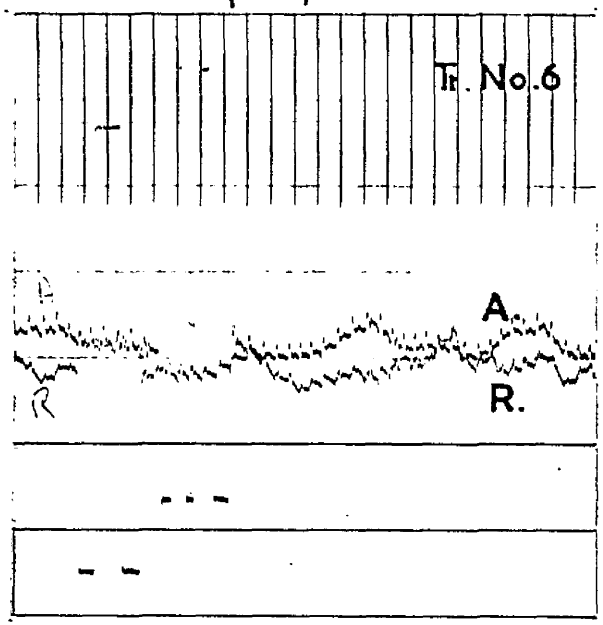
Time (Mins)	Urine/5 min auto & L.kid	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec —
Basal	- mls	0 - 1 (0)	
5	2	5 - 8 (1)	
10	12	7 - 9 (2)	
15	20	7 - 10 (3)	
20	45	6 - 8 (4)	
25	37	11 - 13 (5)	
30	62	15 - 16 (6)	
35	70	13 - 15 (7)	
40	105	14 - 15 (8)	
45	73	13 - 16 (9)	

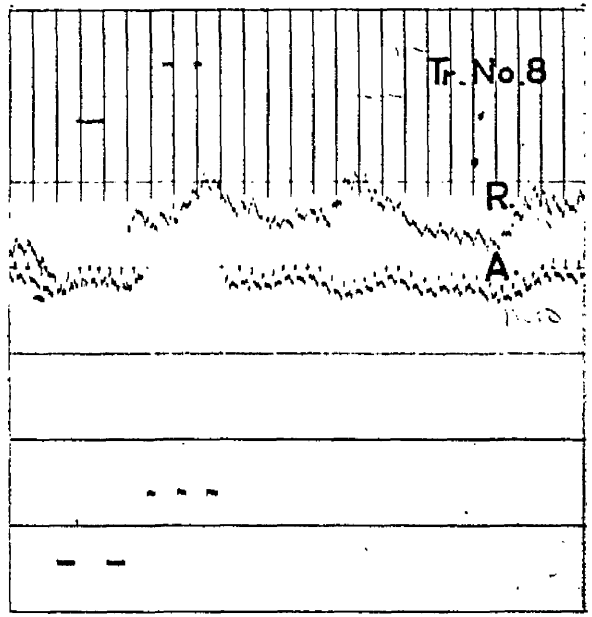




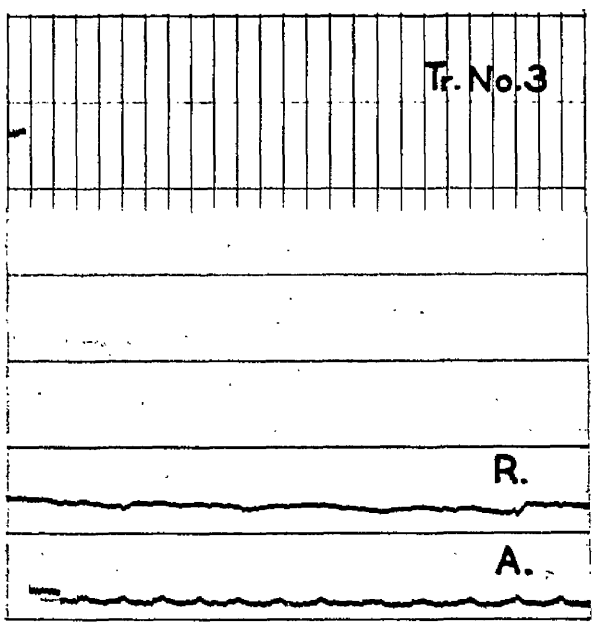
No 22 (A3) 14/9/61

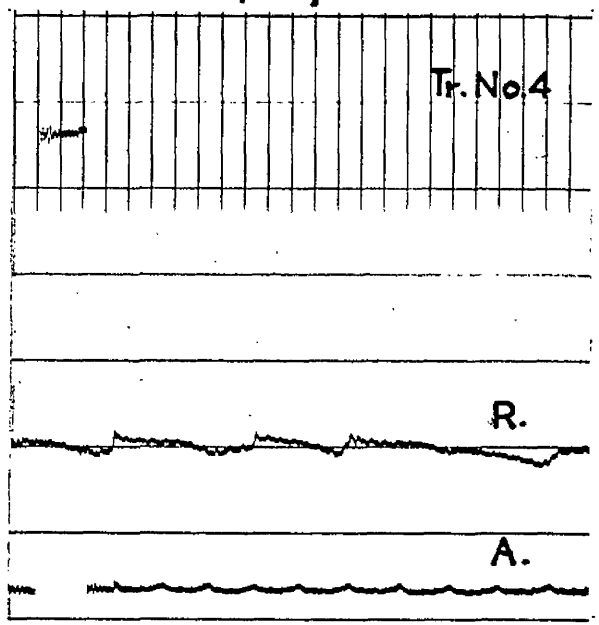


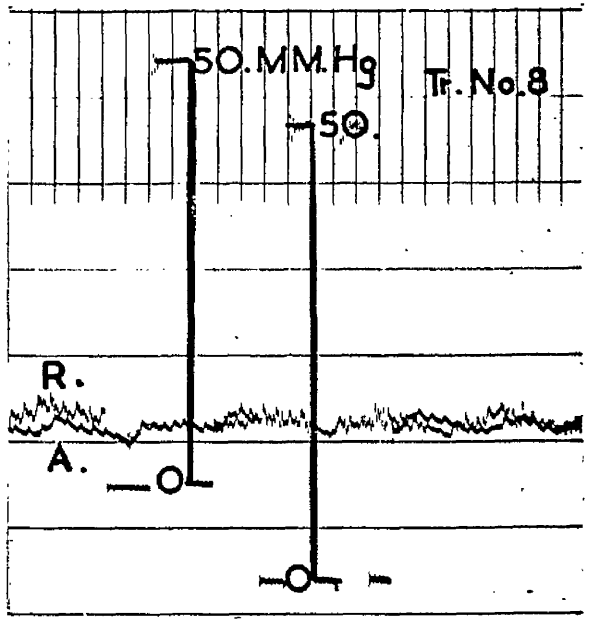




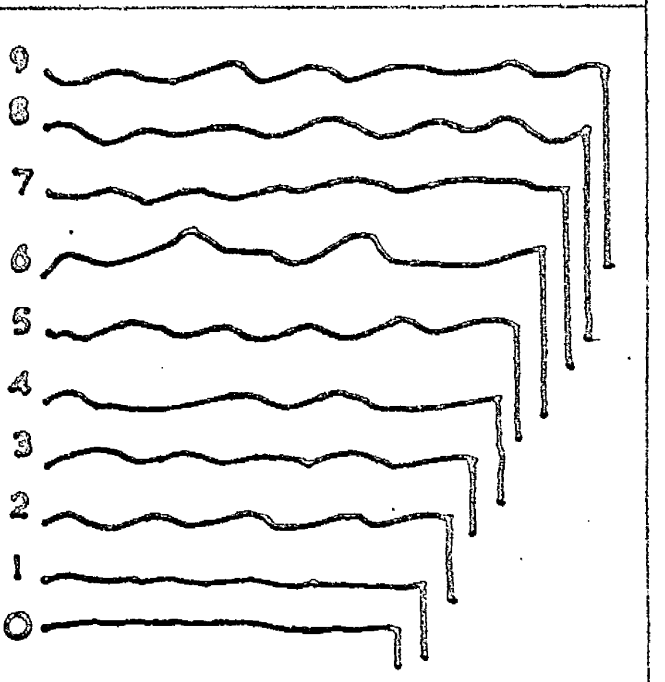
No 22 (A3) 23/9/61



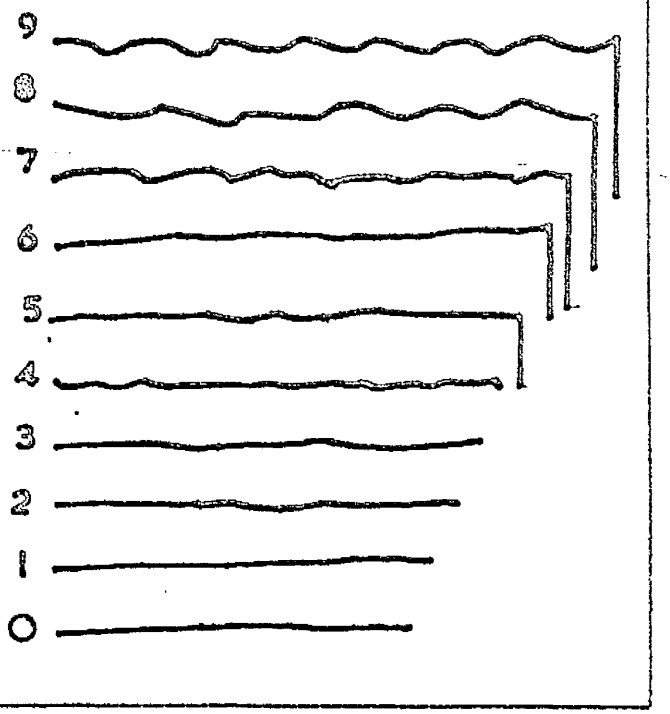




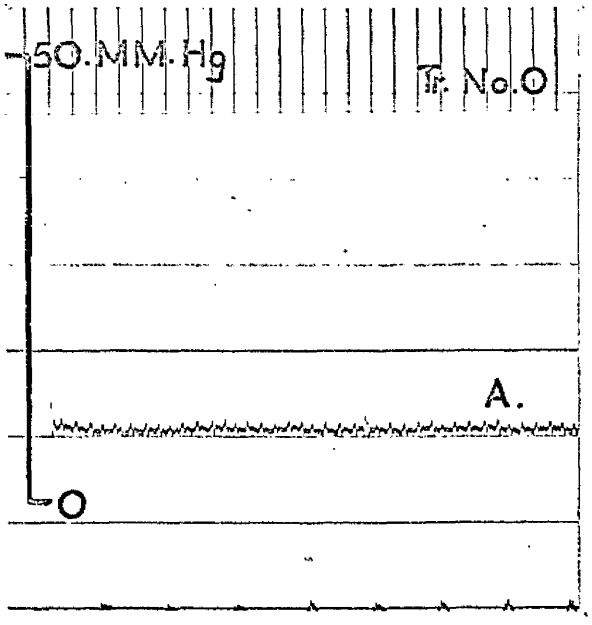
A3 (No.22) 14.9.61 Auto.Kidney Osmotic Diuresis

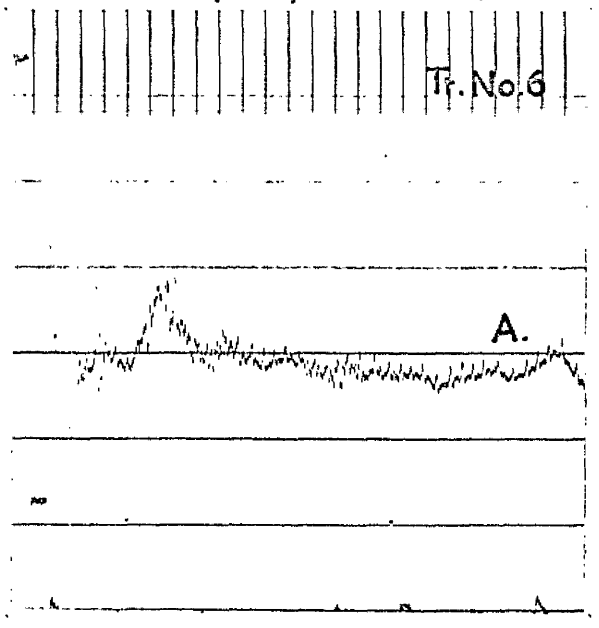
Time (Mins)	Blad Press mm.Hg	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec
Basal	2	5 (0)	
5	4	8 (1)	
10	6	8 - 10 (2)	
15	8	8 - 10 (3)	
20	11	11 - 15 (4)	
25	13	12 - 15 (5)	
30	13	16 - 22 (6)	
35	16	18 - 22 (7)	
40	16	21 - 26 (8)	
45	16	23 - 25 (9)	

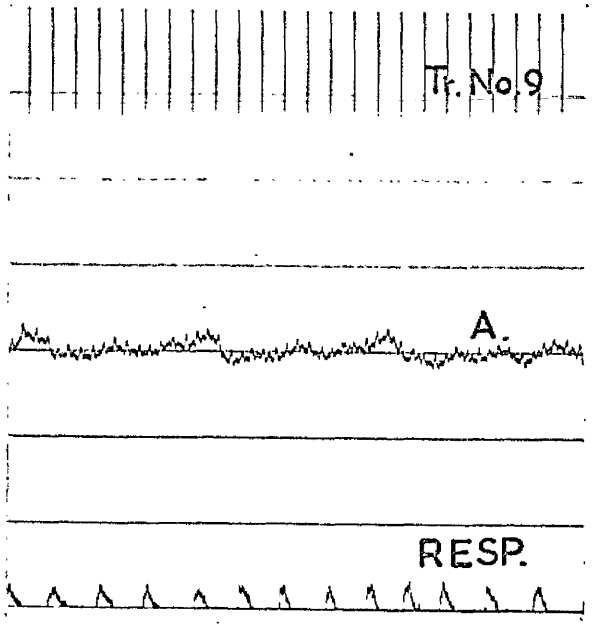
A3 (No.22) 22.9.61 Auto.Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing — 20 sec
Basal	4	0 - 1 (0)	
5	5	0 (1)	
10	7	0 (2)	
15	9	0 (3)	
20	11	1 - 2 (4)	
25	12	7 - 10 (5)	
30	12	9 - 12 (6)	
35	13	15 - 18 (7)	
40	15	16 - 20 (8)	
45	15	16 - 19 (9)	

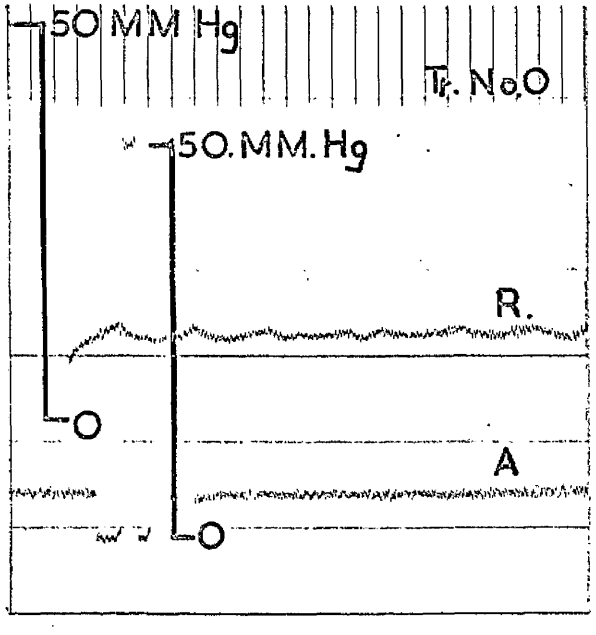
No 22 (A3) 13/10/61

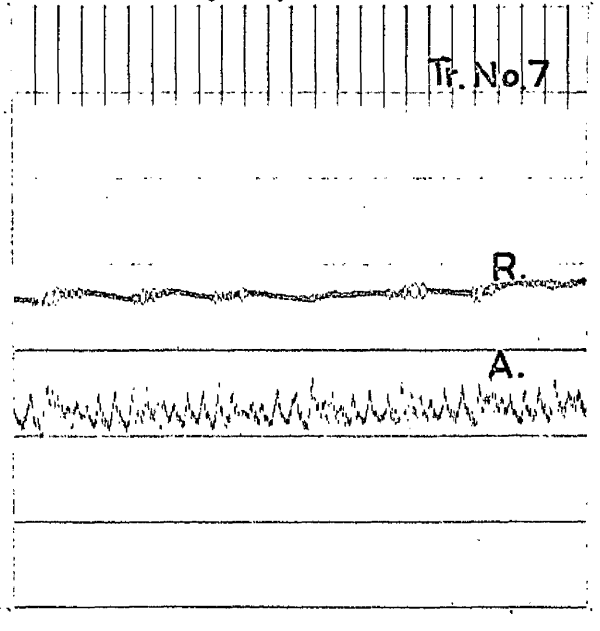


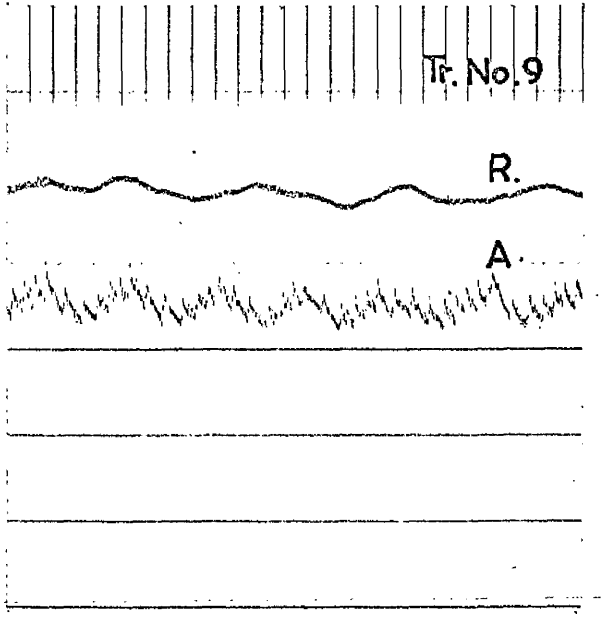




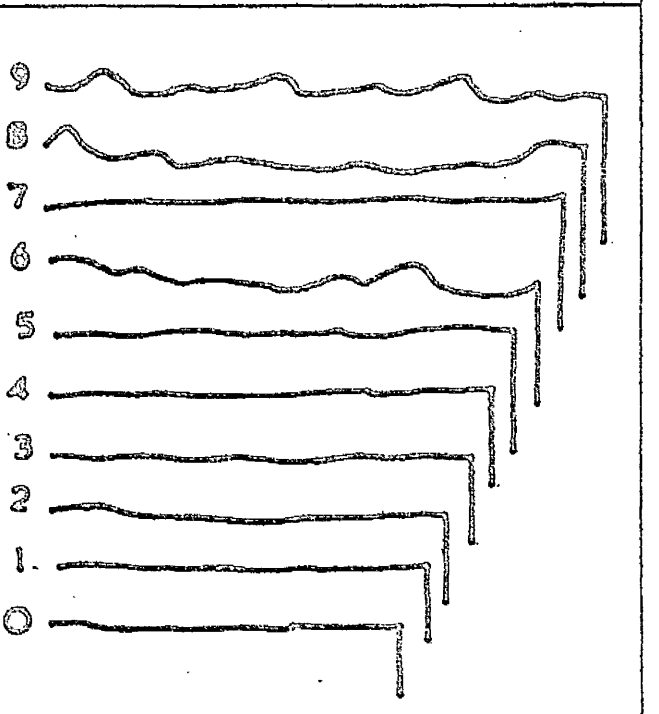
No 22 (A3) 17/11/61



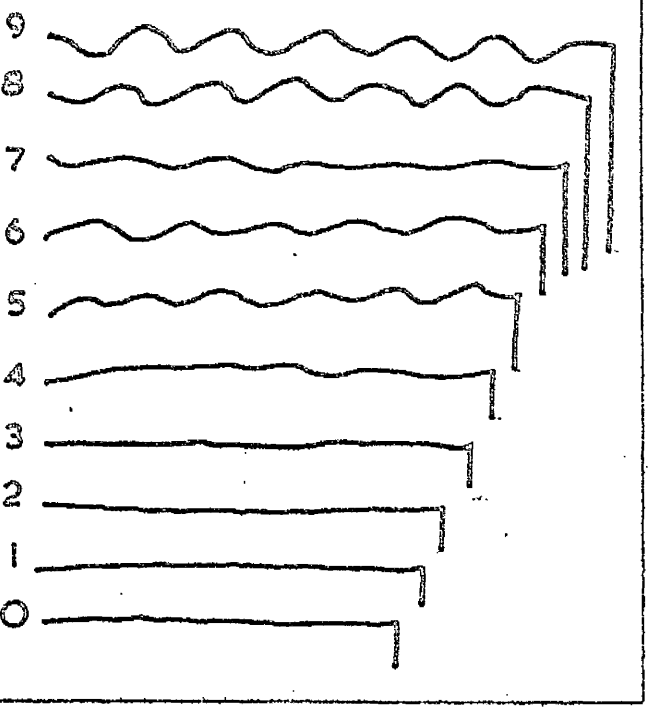




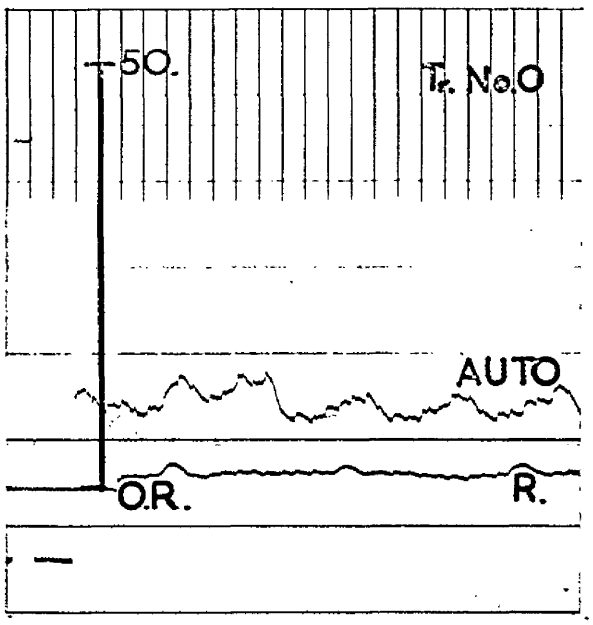
A.3 (No.22) 13.10.61 Auto kidney Osmotic Diuresis

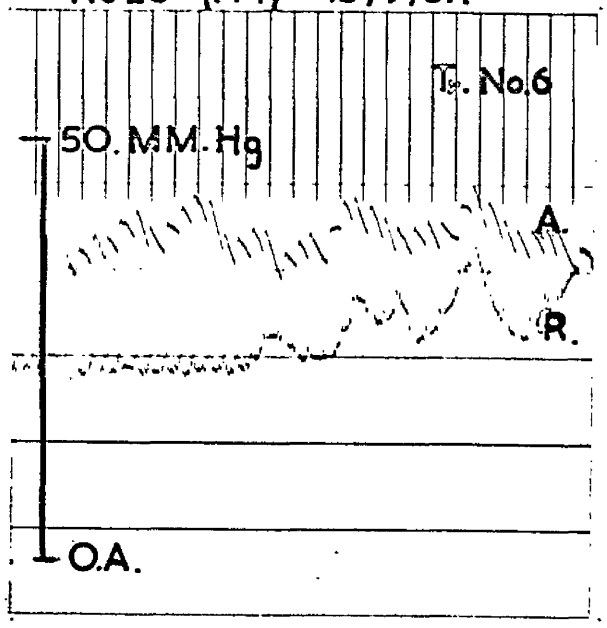
Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	4	7 - 9 (0)	
5	5	8 - 9 (1)	
10	7	9 - 11 (2)	
15	9	9 - 10 (3)	
20	11	10 - 11 (4)	
25	12	13 - 14 (5)	
30	12	12 - 17 (6)	
35	13	15 (7)	
40	15	15 - 21 (8)	
45	15	17 - 20 (9)	

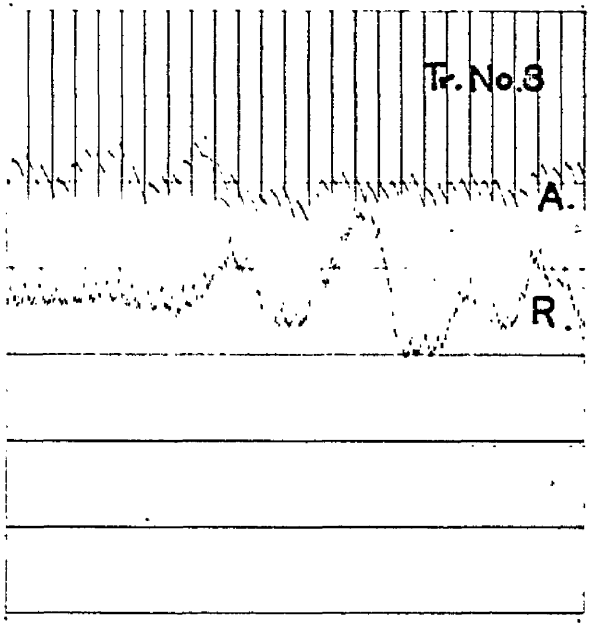
A.3 (No.22) 17.11.61 Auto.Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	5	4 - 6 (0)	
5	5	4 - 5 (1)	
10	8	4 - 6 (2)	
15	10	5 - 6 (3)	
20	10	5 - 8 (4)	
25	3	7 - 10 (5)	
30	8	7 - 10 (6)	
35	10	12 - 15 (7)	
40	15	20 - 24 (8)	
45	15	22 - 28 (9)	

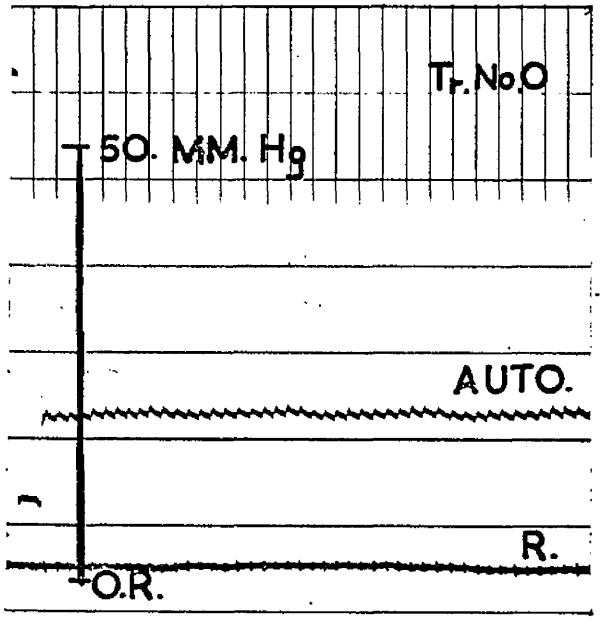
No 23 (A4) 15/9/61.

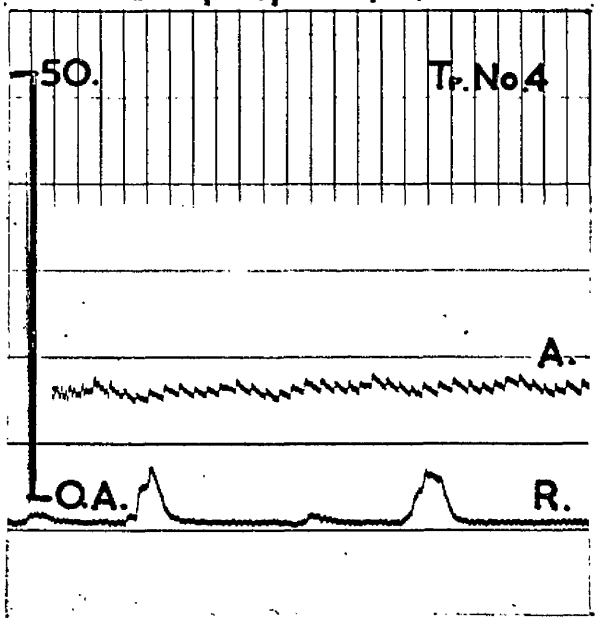


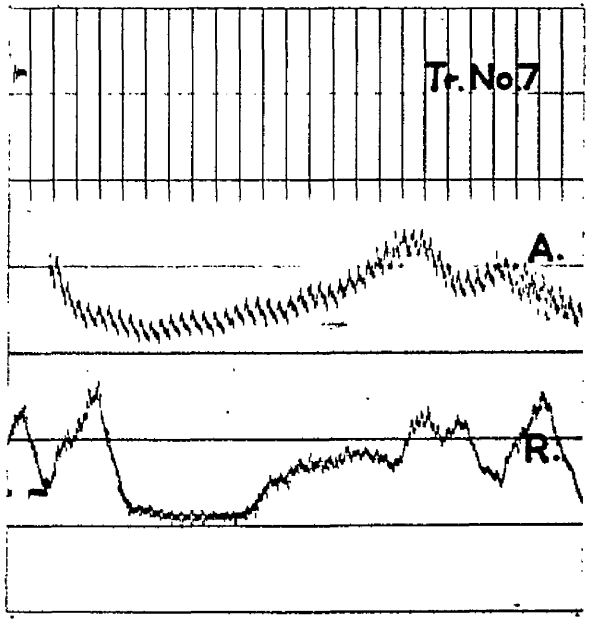




No 23 (A4) 25/9/61



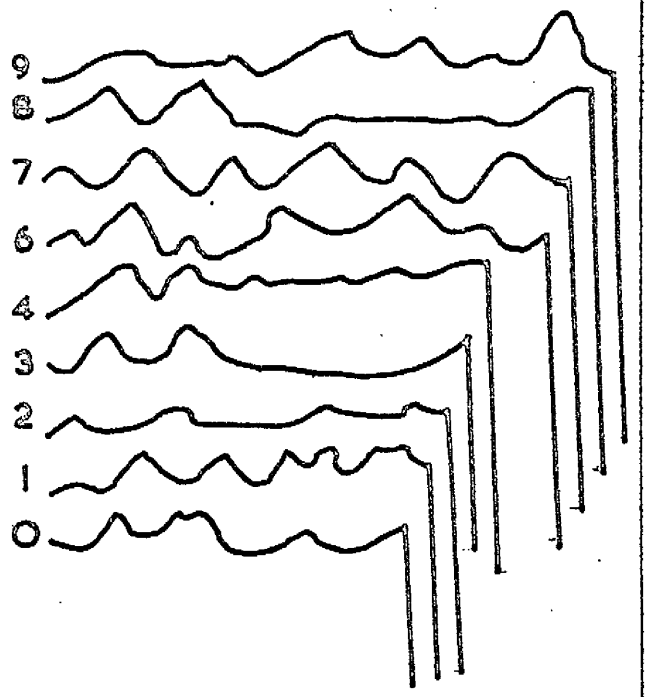




A.4 (No.23) 15.9.61 Auto Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)
Basal	5	18 - 21 (0)
5	5	20 - 28 (1)
10	8	28 - 31 (2)
15	9	21 - 29 (3)
20	9	32 - 37 (4)
25	-	-, (5)
30	15	35 - 43 (6)
35	24	36 - 44 (7)
40	28	40 - 48 (8)
45	29	44 - 50 (9)

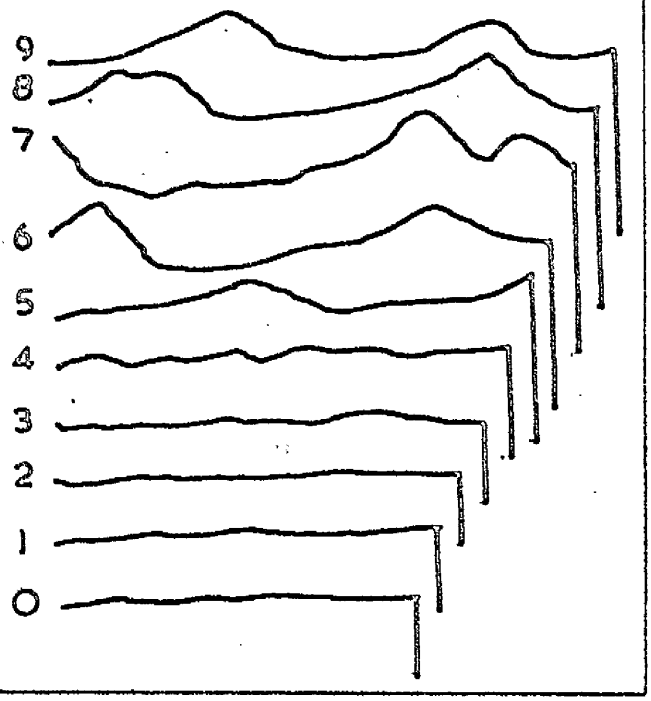
Pressure Tracing
20 sec



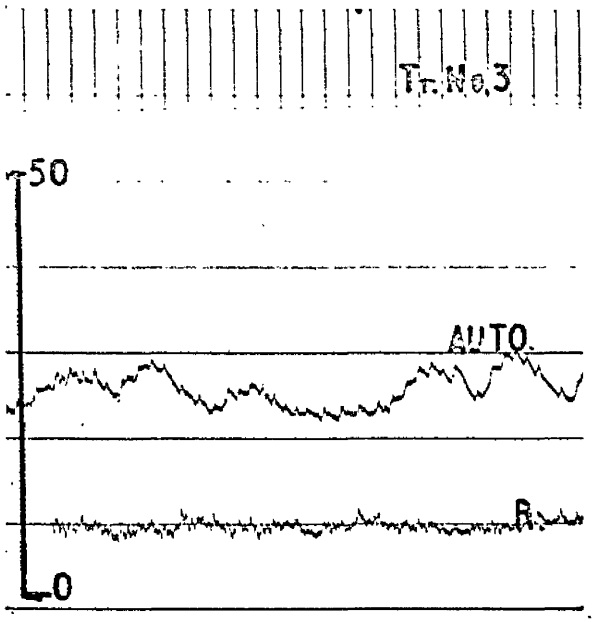
A.4 (No.23) 25.9.61 Auto Kidney Osmotic Diuresis

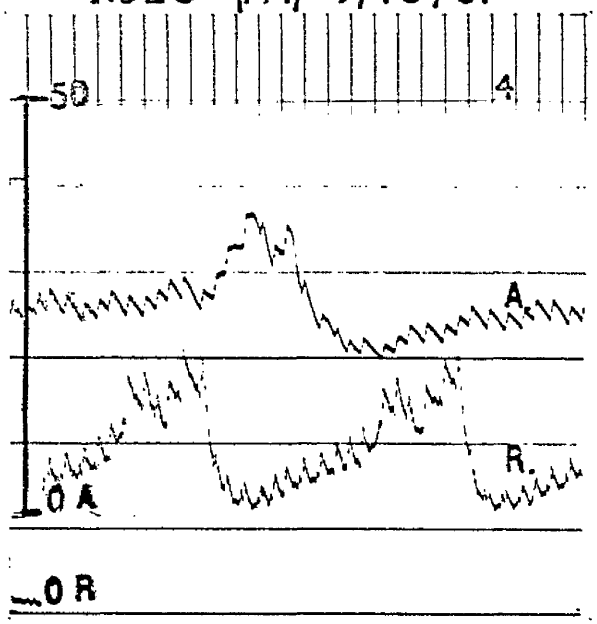
Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)
Basal	0	10 (0)
5	2	8 - 9 (1)
10	3	8 - 9 (2)
15	3	10 - 11 (3)
20	5	12 - 13 (4)
25	5	15 - 22 (5)
30	6	16 - 22 (6)
35	11	20 - 30 (7)
40	11	21 - 31 (8)
45	11	20 - 27 (9)

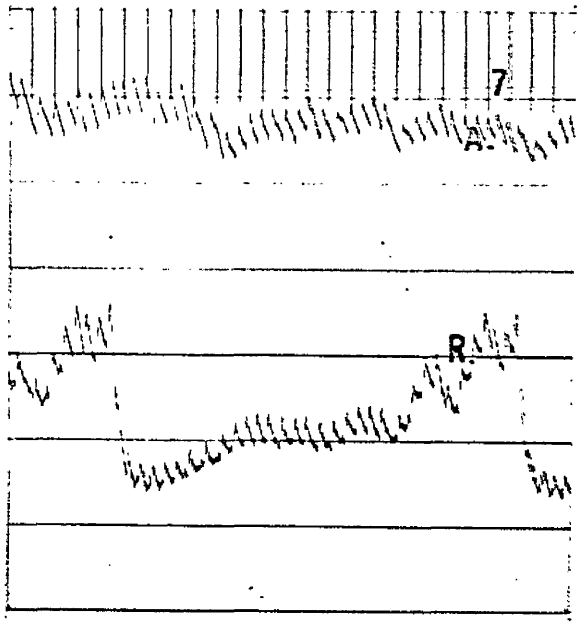
Pressure Tracing
20 sec



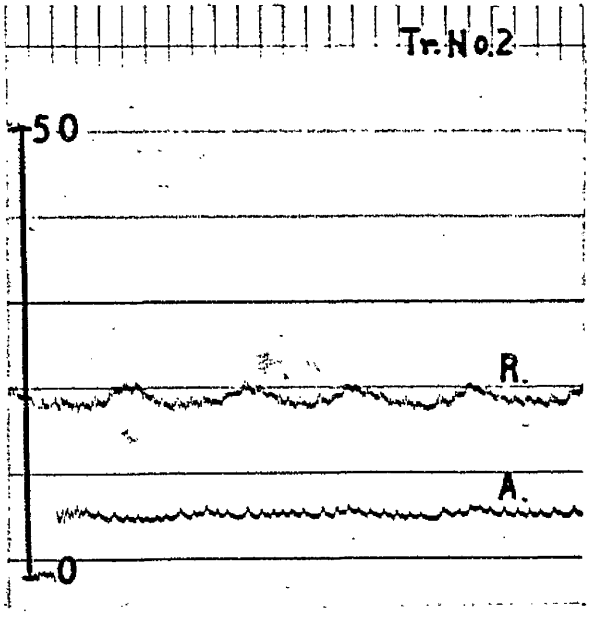
No23 (A4) 9/10/61

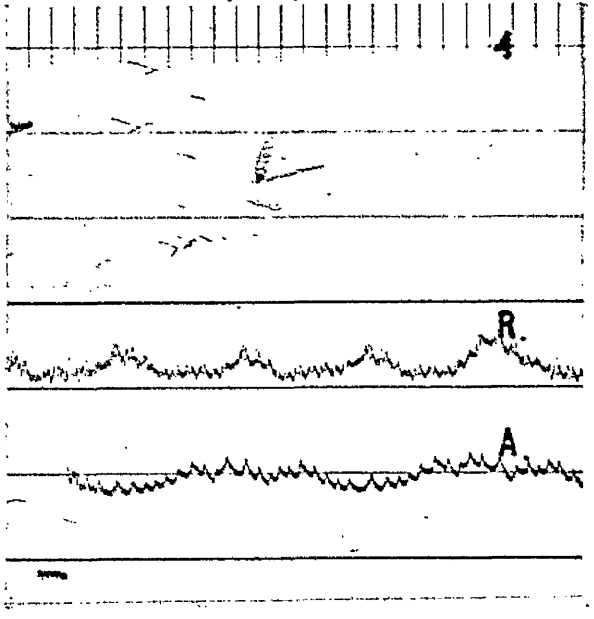


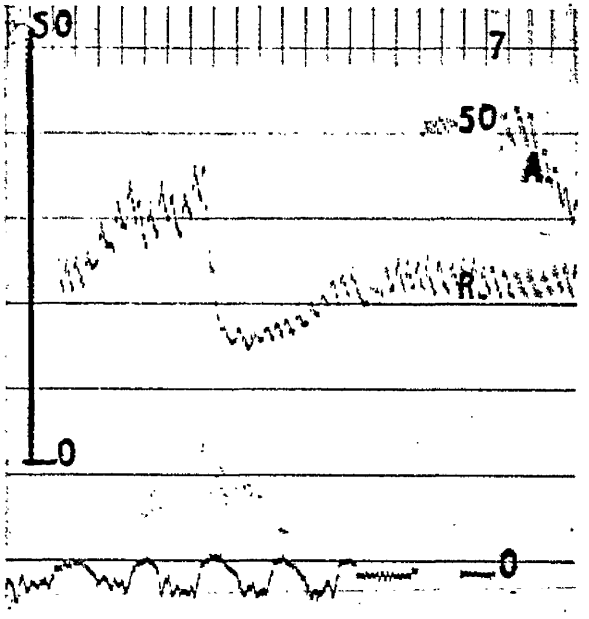




No23 (A4) 16/10/61



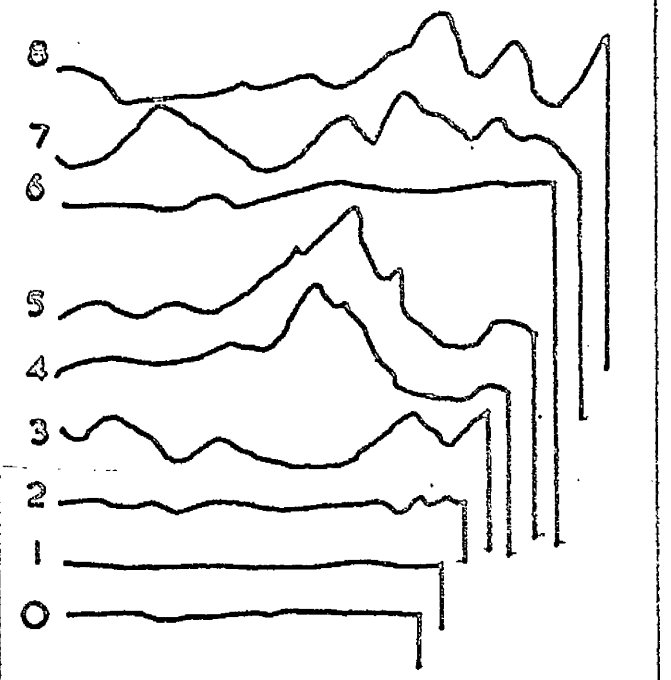




A.4 (No.23) 9.10.61 Auto Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)
Basal	0	7 (0)
5	4	7 (1)
10	6	6 - 9 (2)
15	7	10 - 16 (3)
20	11	17 - 31 (4)
25	12	22 - 38 (5)
30	13	39 - 44 (6)
35	16	29 - 40 (7)
40	22 - 25	30 - 42 (8)
45	-	- (9)

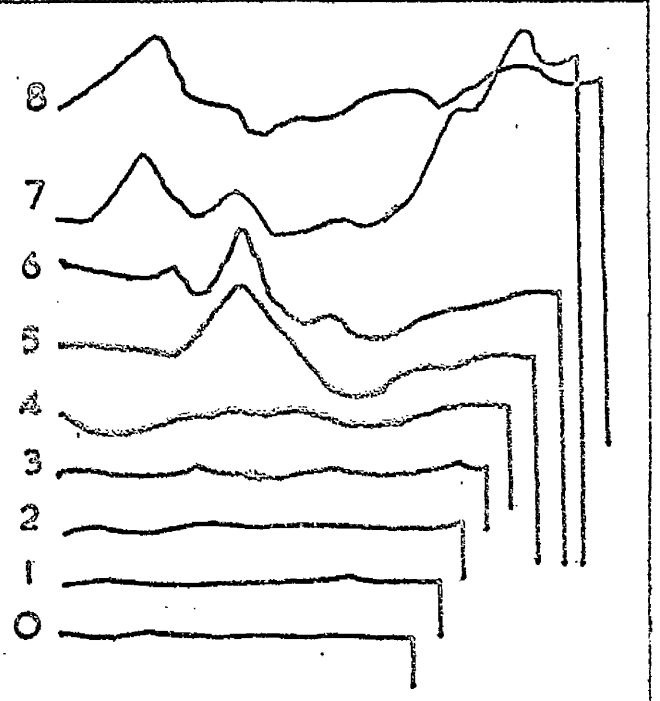
Pressure Tracing — 20 sec

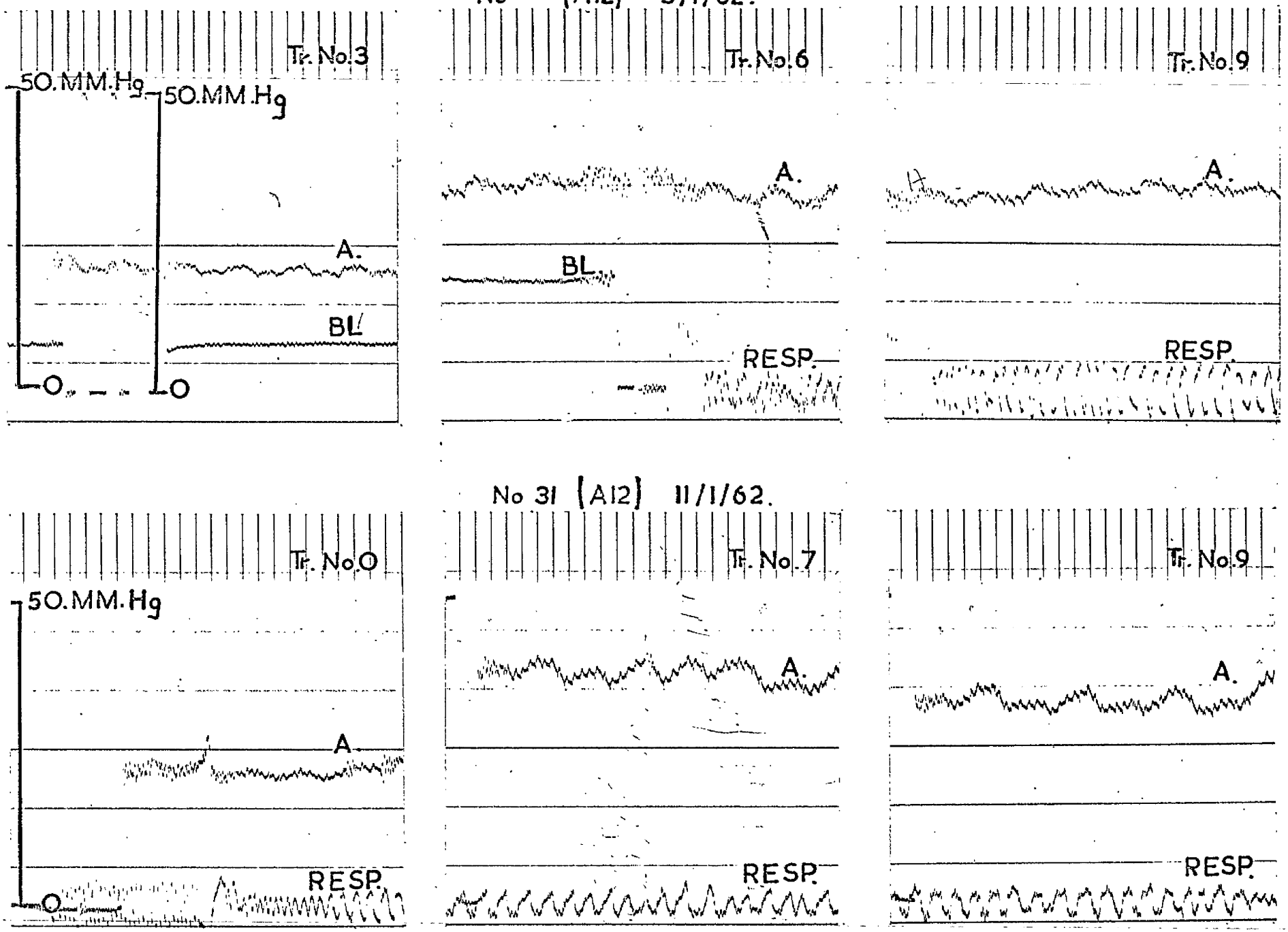


A.4 (No.23) 16.10.61 Auto Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)
Basal	0	6 (0)
5	0	5 (1)
10	4	5 - 7 (2)
15	5	5 - 8 (3)
20	9	5 - 12 (4)
25	11	19 - 27 (5)
30	14	26 - 40 (6)
35	18	38 - 65 (7)
40	20	36 - 50 (8)
45	-	- (9)

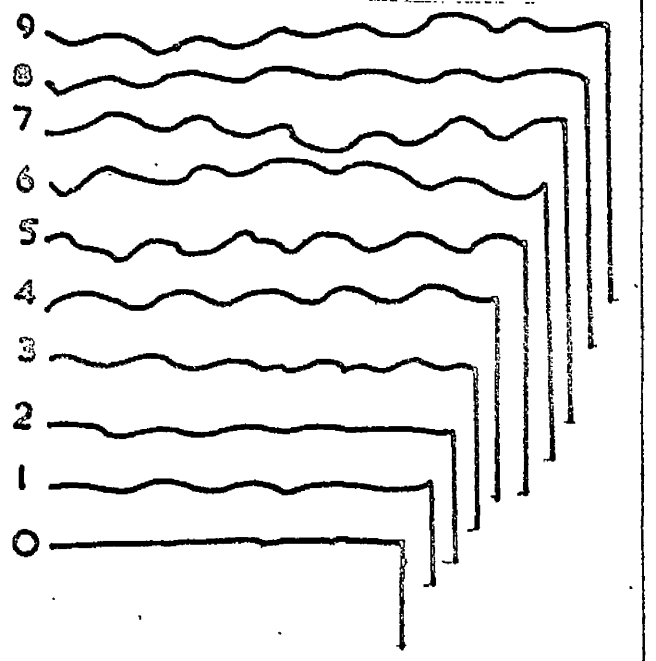
Pressure Tracing — 20 sec




No 31 (A12) 5/1/62.
Tr. No.3
50.MM.Hg
50.MM.Hg
A.
BL
O
O
Tr. No.6
A.
BL.
RESP.
Tr. No.9
A.
RESP.
No 31 (A12) 11/1/62.
Tr. No.0
50.MM.Hg
A.
RESP.
O
Tr. No.7
A.
RESP.
Tr. No.9
A.
RESP.

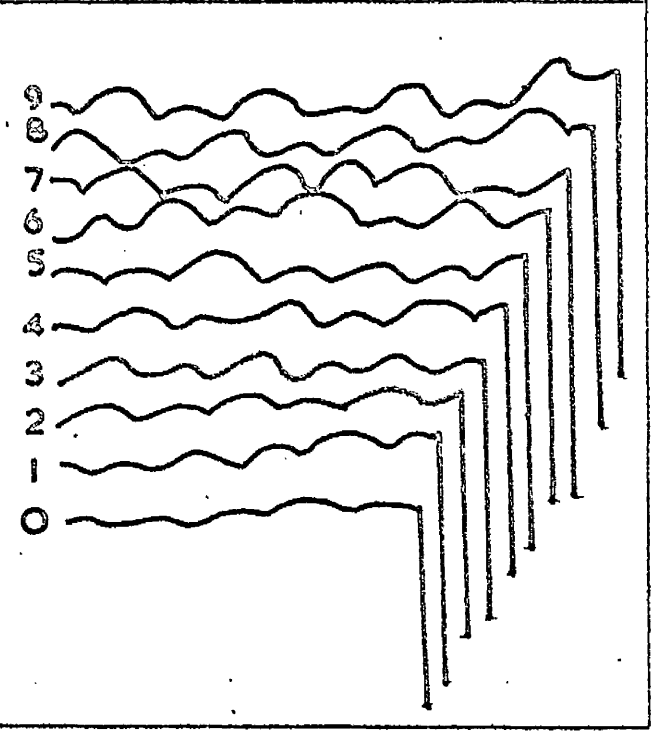
A.12 (No.31) 5.1.62 Auto.Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	0	12 (0)	
5	2	10 - 13 (1)	
10	5	14 - 17 (2)	
15	8	18 - 22 (3)	
20	14	21 - 25 (4)	
25	13	27 - 31 (5)	
30	18	30 - 35 (6)	
35	10	32 - 37 (7)	
40	18	30 - 33 (8)	
45	14	30 - 35 (9)	

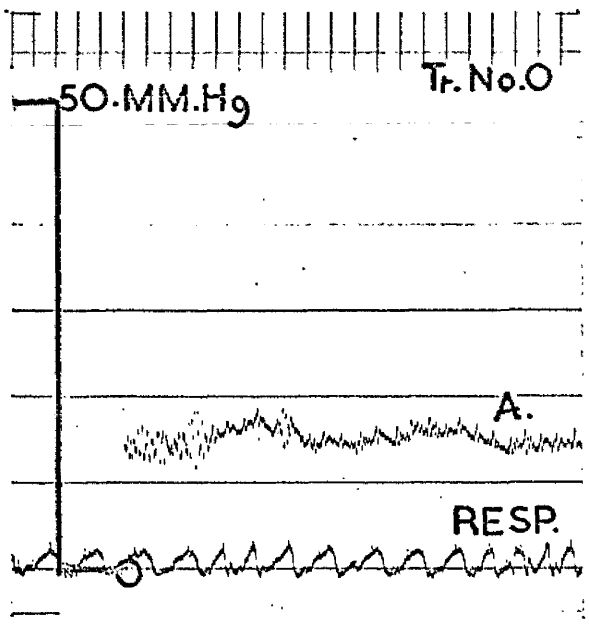


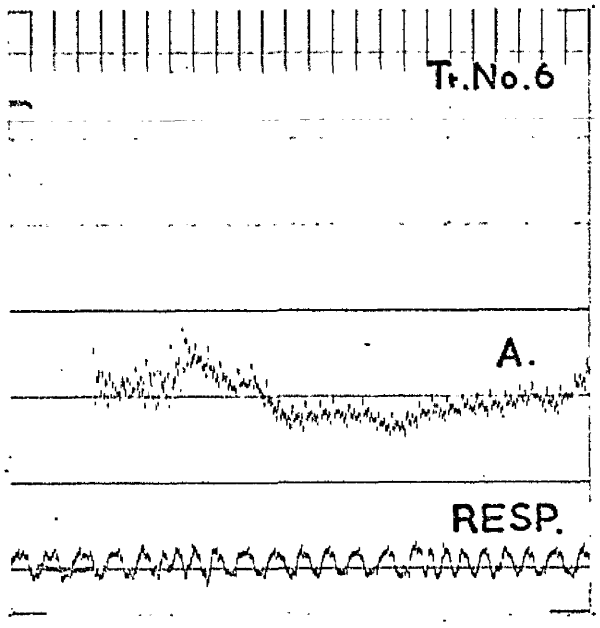
A.12 (No.31) 11.1.62 Auto.Kidney Osmotic Diuresis

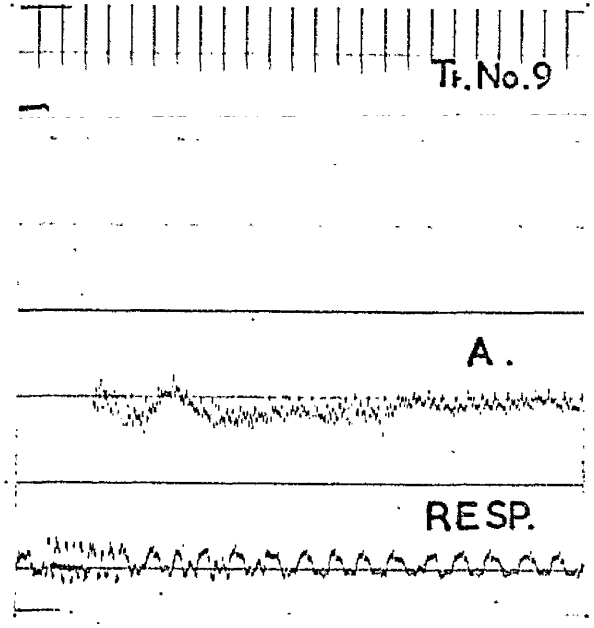
Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- Mls	22 - 26 (0)	
5	8	26 - 31 (1)	
10	20	27 - 31 (2)	
15	24	30 - 33 (3)	
20	25	30 - 35 (4)	
25	28	32 - 37 (5)	
30	34	33 - 38 (6)	
35	41	35 - 41 (7)	
40	56	33 - 39 (8)	
45	55	31 - 38 (9)	



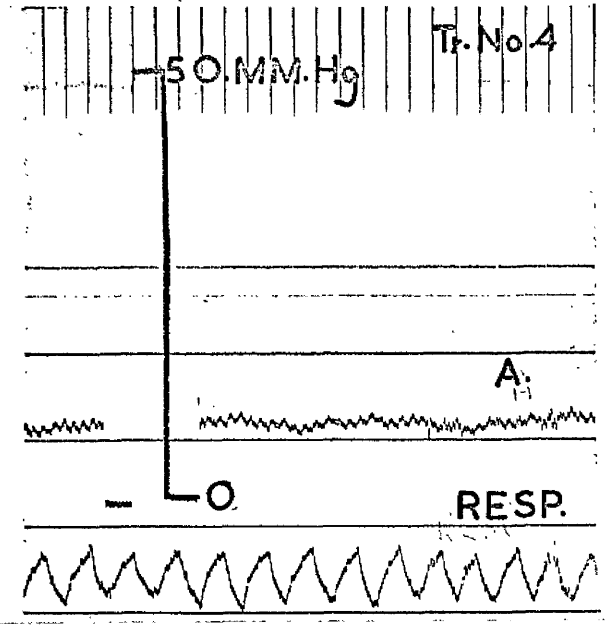
No 31 [A12] 18/1/62

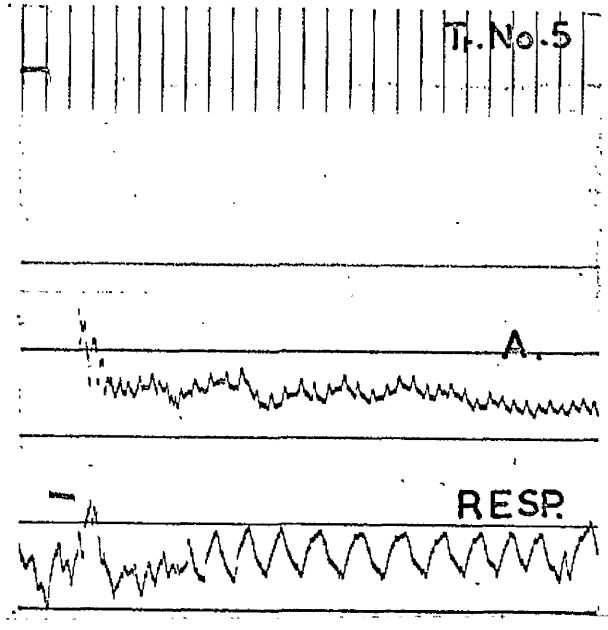


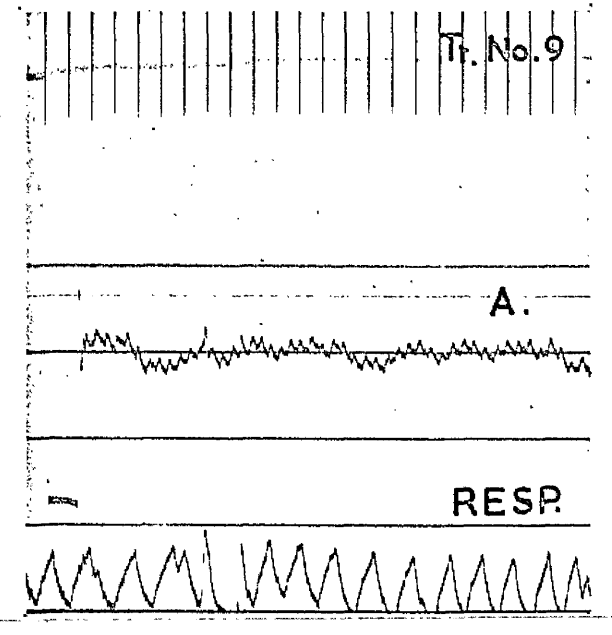




No.31 [A12] 9/2/62

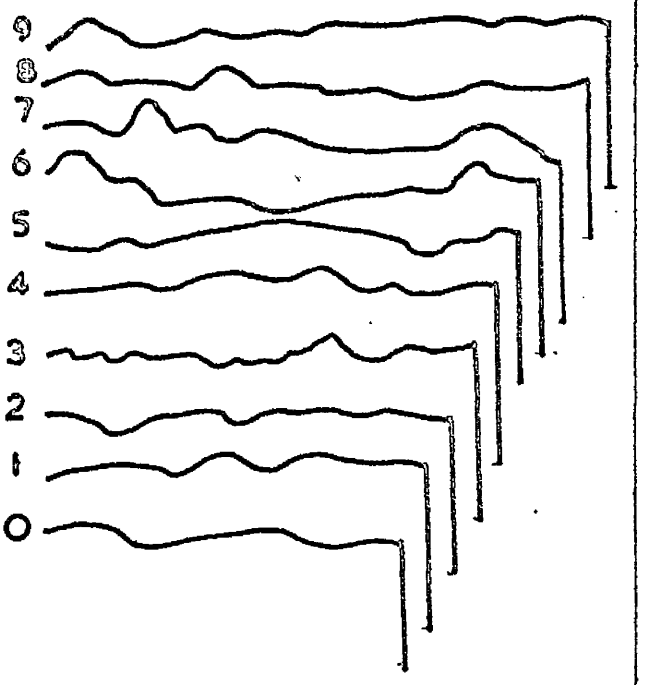






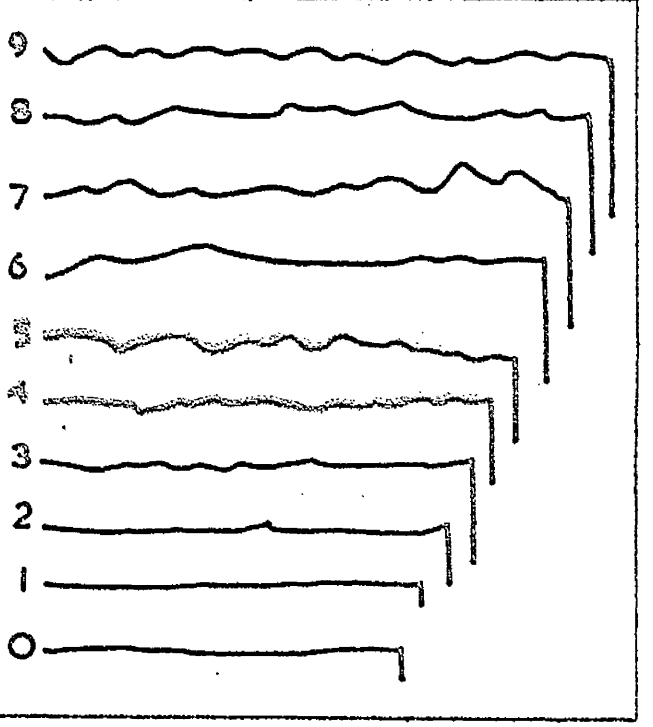
A.12 (No.31) 18.1.62 Auto.Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	13 - 16 (0)	
5	6	17 - 22 (1)	
10	19	17 - 21 (2)	
15	27	18 - 23 (3)	
20	17	21 - 26 (4)	
25	56	16 - 21 (5)	
30	43	16 - 25 (6)	
35	52	19 - 27 (7)	
40	27	15 - 20 (8)	
45	47	17 - 21 (9)	

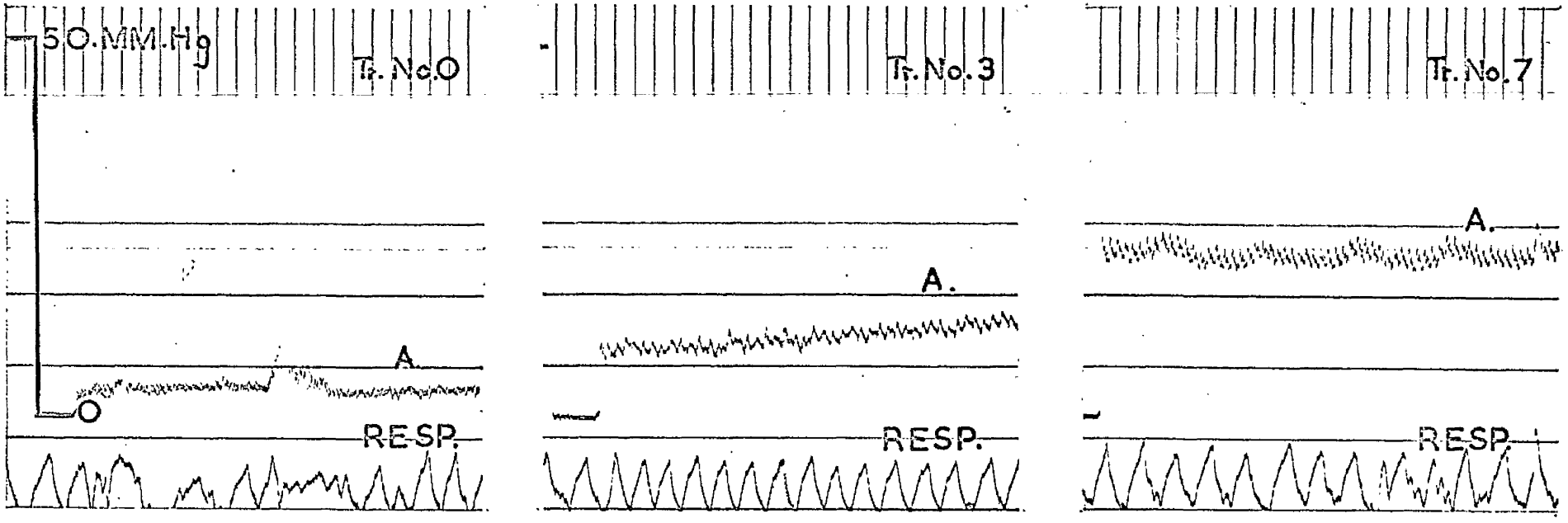


A.12 (No.31) 9.2.62 Auto.Kidney Osmotic Diuresis

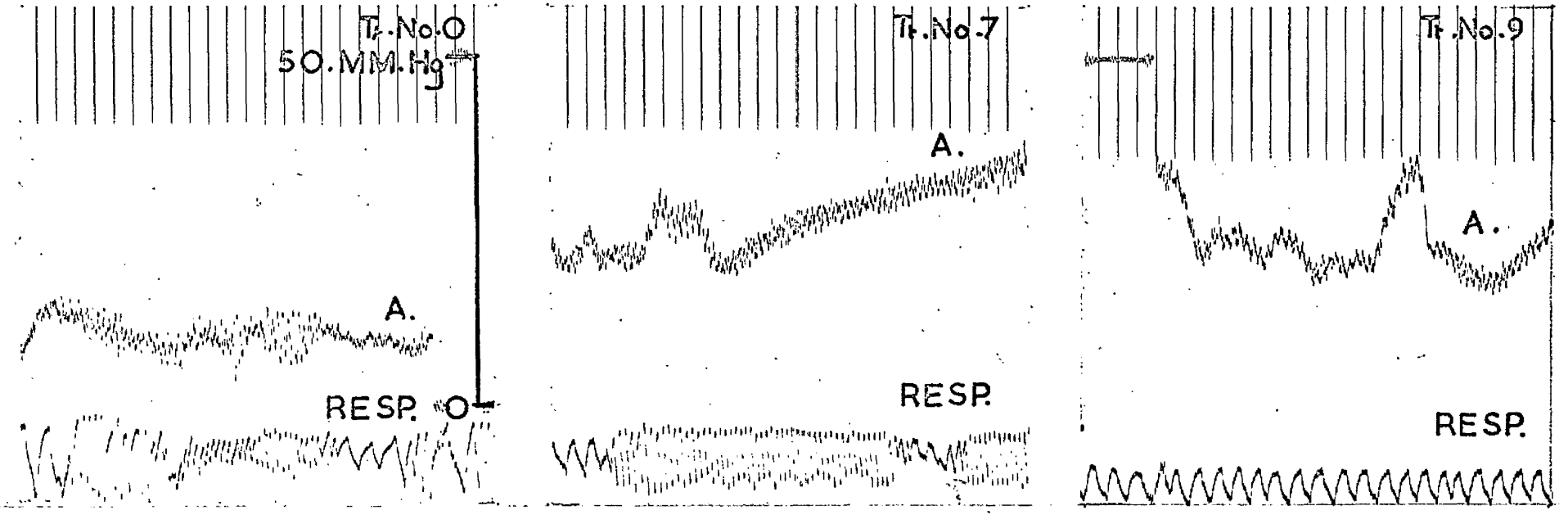
Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg.(Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	4 (0)	
5	0	3 (1)	
10	7	6 - 7 (2)	
15	22	10 - 13 (3)	
20	56	9 - 11 (4)	
25	45	11 - 15 (5)	
30	36	14 - 17 (6)	
35	53	16 - 21 (7)	
40	51	16 - 18 (8)	
45	46	17 - 20 (9)	



No.31 [A12] 15/2/62



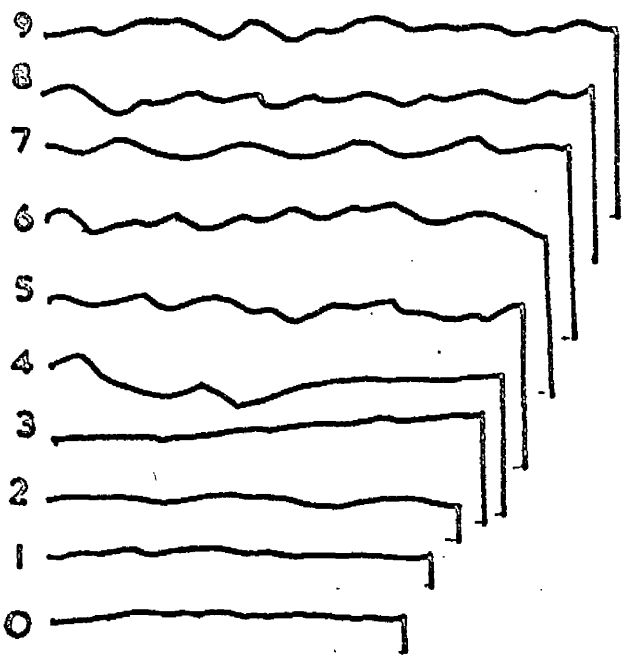
No 31 12 25/5/62



A.12 (No.31) 15.2.62 Auto.Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	4 - 5 (0)
5	20	3 - 7 (1)
10	21	3 - 6 (2)
15	21	10 - 14 (3)
20	33	12 - 20 (4)
25	41	16 - 22 (5)
30	39	18 - 23 (6)
35	46	21 - 26 (7)
40	49	18 - 24 (8)
45	39	21 - 24 (9)

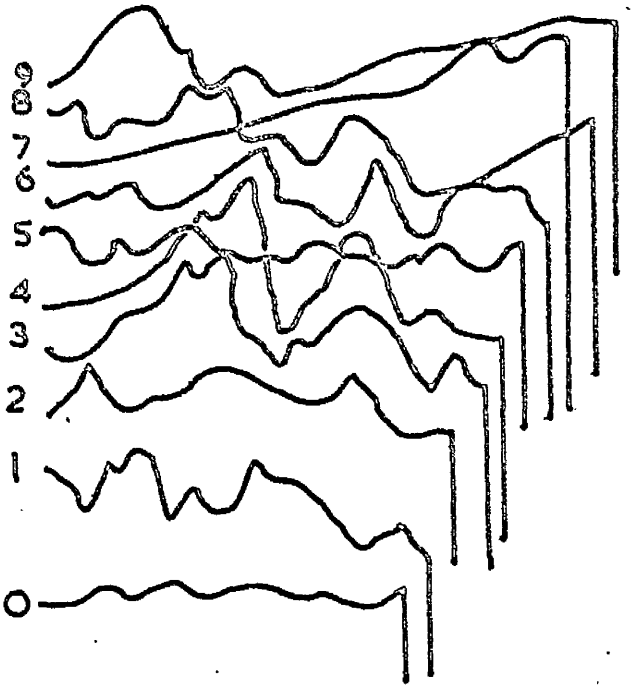
Pressure Tracing — 20 sec



A.12 (No.31) 25.5.62 Auto.Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	8 - 12 (0)
5	9	14 - 28 (1)
10	23	14 - 23 (2)
15	26	21 - 36 (3)
20	29	23 - 42 (4)
25	30	19 - 25 (5)
30	33	22 - 33 (6)
35	47	28 - 45 (7)
40	59	21 - 35 (8)
45	51	21 - 32 (9)

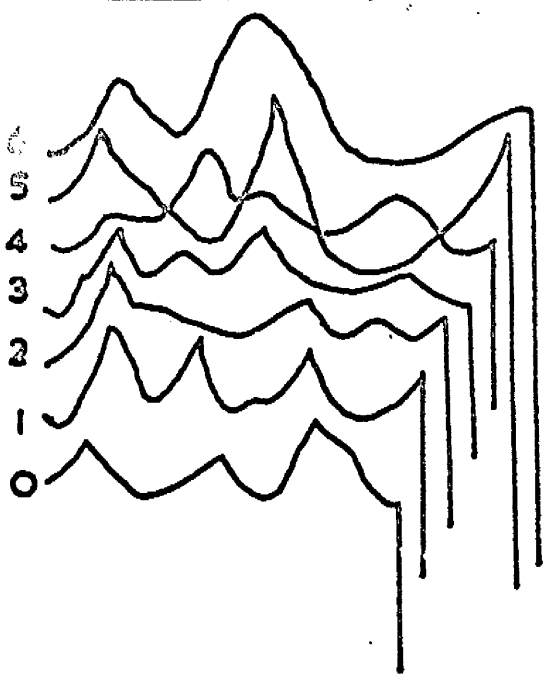
Pressure Tracing — 20 sec

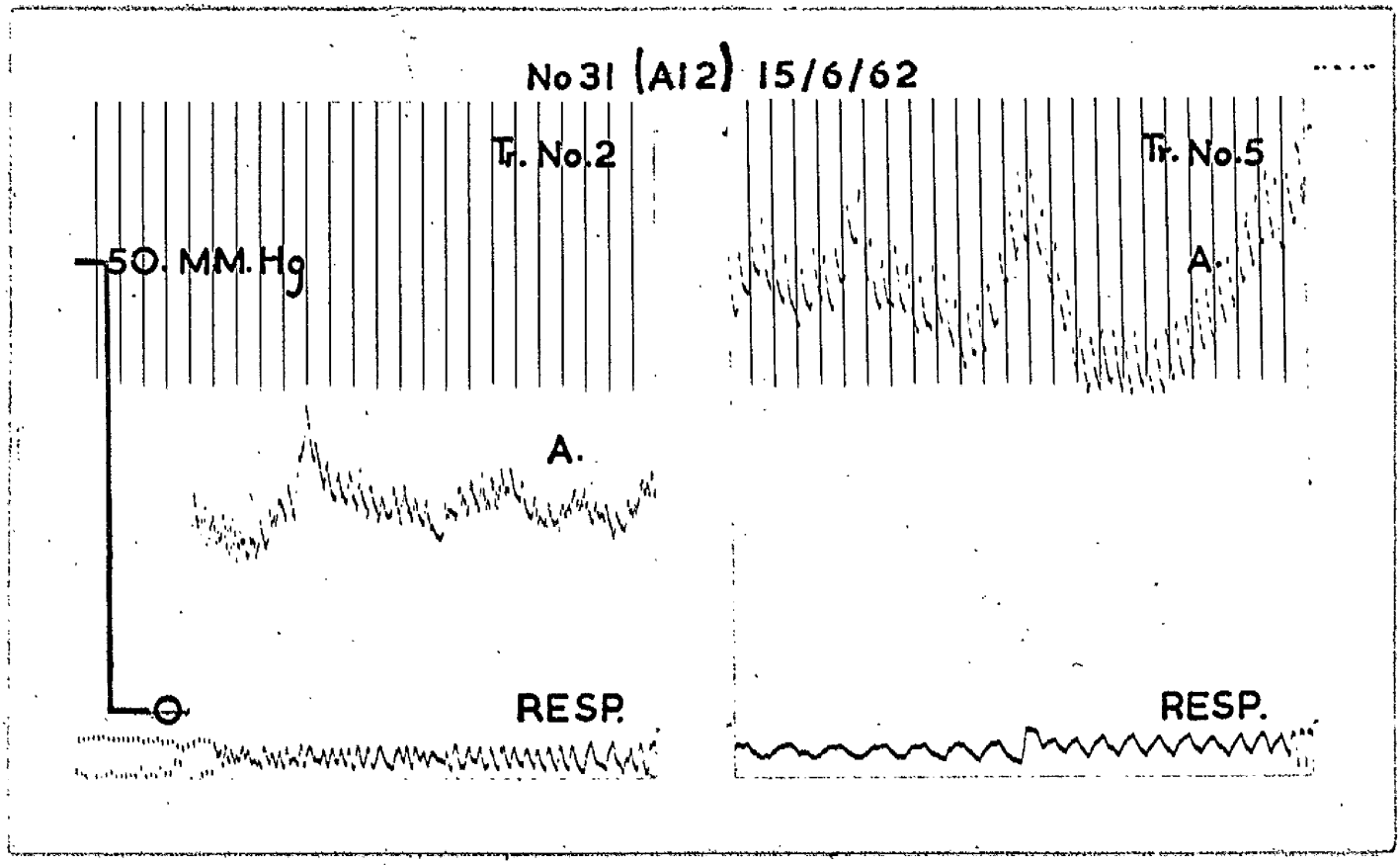


A.12 (No.31) 15.6.62 Auto.Kidney Water Diuresis

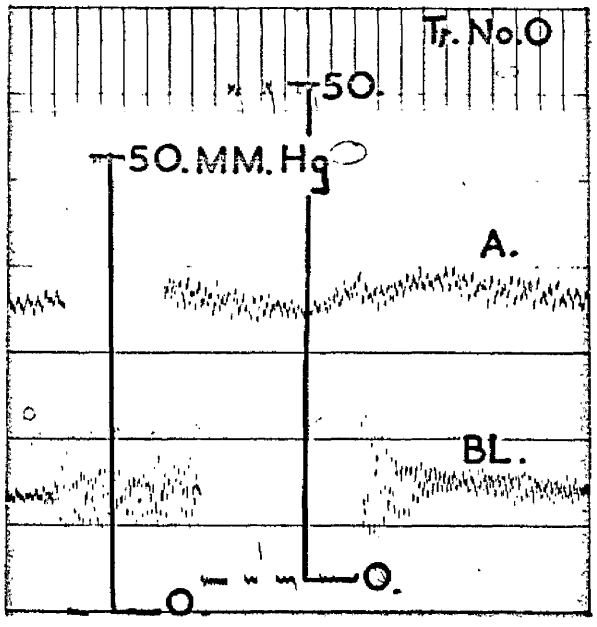
Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	19 - 31 (0)
5	7	19 - 30 (1)
10	11	19 - 31 (2)
15	16	18 - 35 (3)
20	19	20 - 32 (4)
25	10	36 - 59 (5)
30	5	50 - 68 (6)
35	2	27 - 53 (7)
40	15	37 - 72 (8)
45	7	42 - 65 (9)

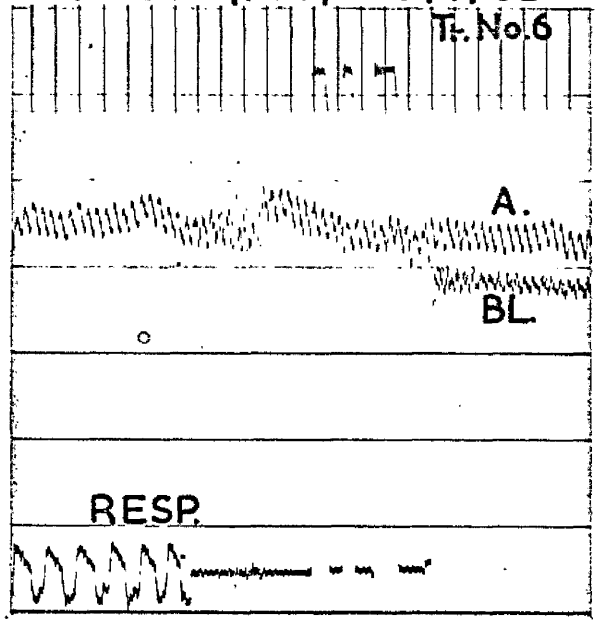
Pressure Tracing
20 sec

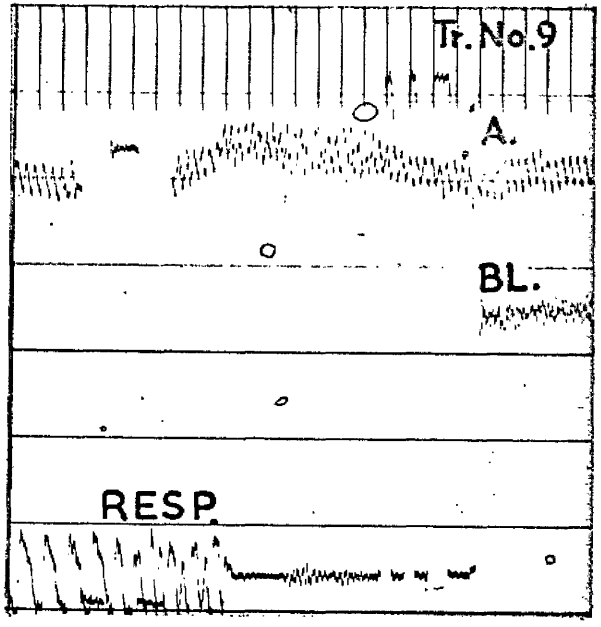




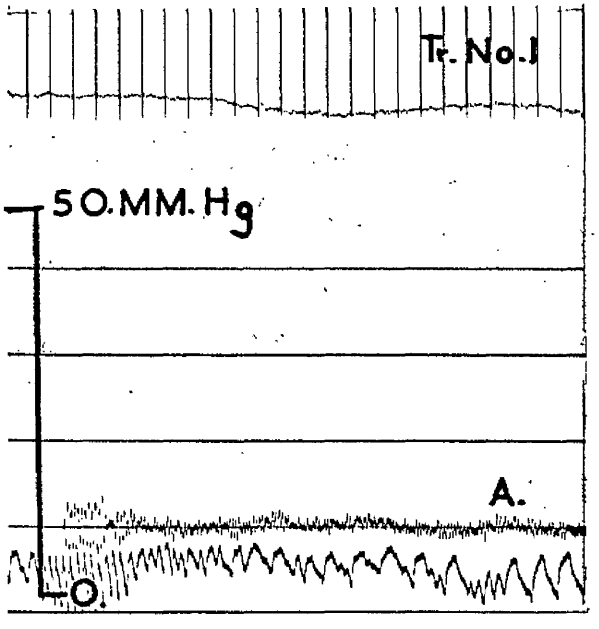
No 33 (A14) 19/1/62

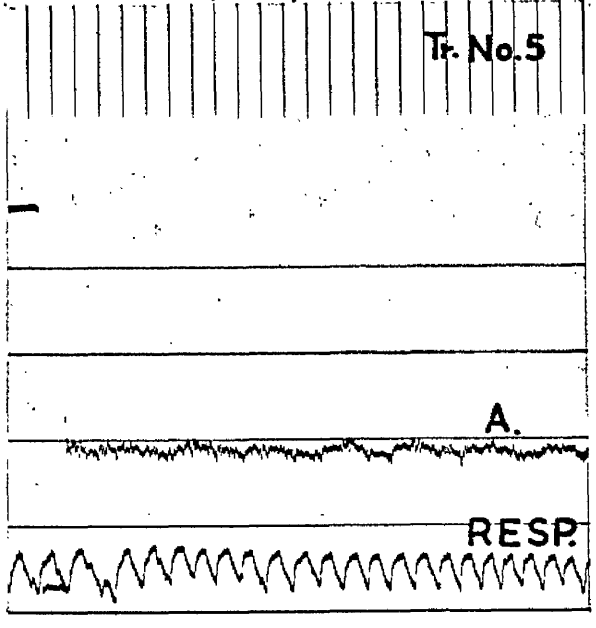


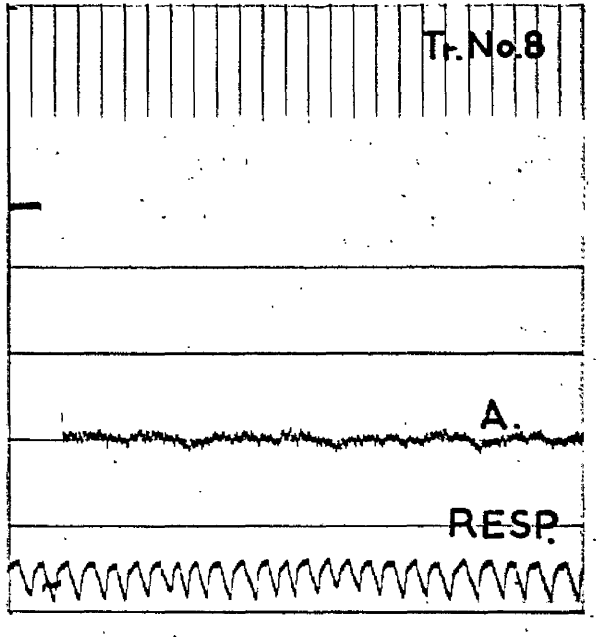




No 33 (A14) 26/1/62



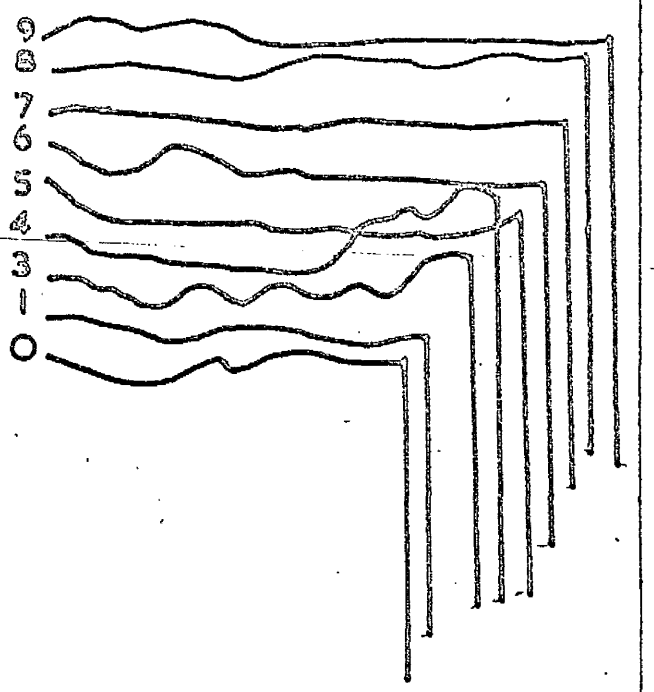




A.14 (No.33) 19.1.62 Auto.Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg. (Tr.No)
Basal	9	33 - 40 (0)
5	12	35 - 38 (1)
10	-	- (2)
15	16	34 - 43 (3)
20	23	39 - 49 (4)
25	20	42 - 48 (5)
30	30	44 - 48 (6)
35	30	42 - 46 (7)
40	35	43 - 48 (8)
45	30	50 - 55 (9)

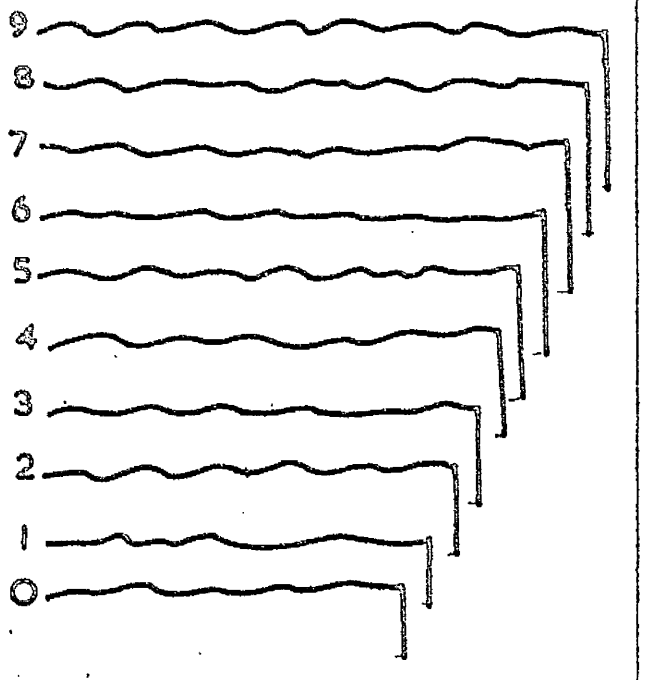
Pressure Tracing
20 sec



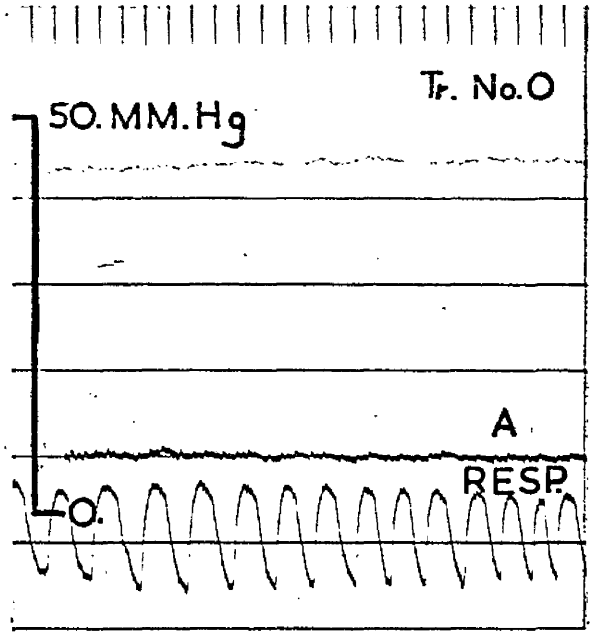
A.14 (No.33) 26.1.62 Auto.Kidney Osmotic Diuresis

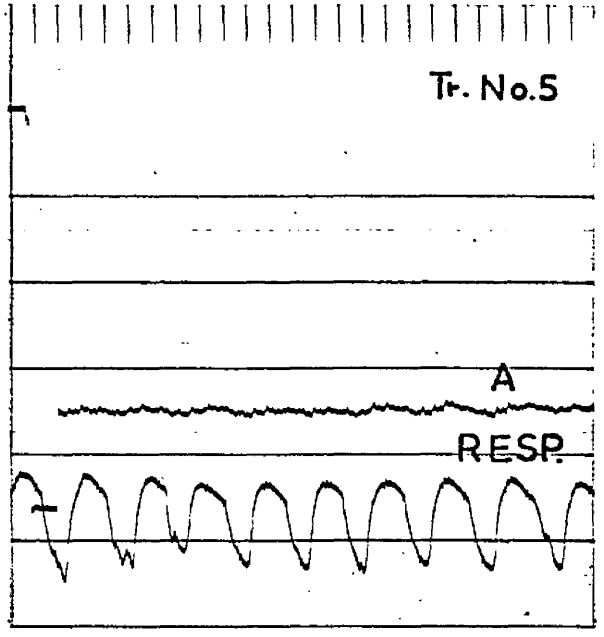
Time (Mins)	Urine/5 min Auto.Kid.	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	7 - 9 (0)
5	0	9 - 10 (1)
10	5	10 - 12 (2)
15	8	11 - 13 (3)
20	13	11 - 14 (4)
25	22	14 - 18 (5)
30	25	16 - 19 (6)
35	31	16 - 19 (7)
40	31	17 - 20 (8)
45	37	18 - 21 (9)

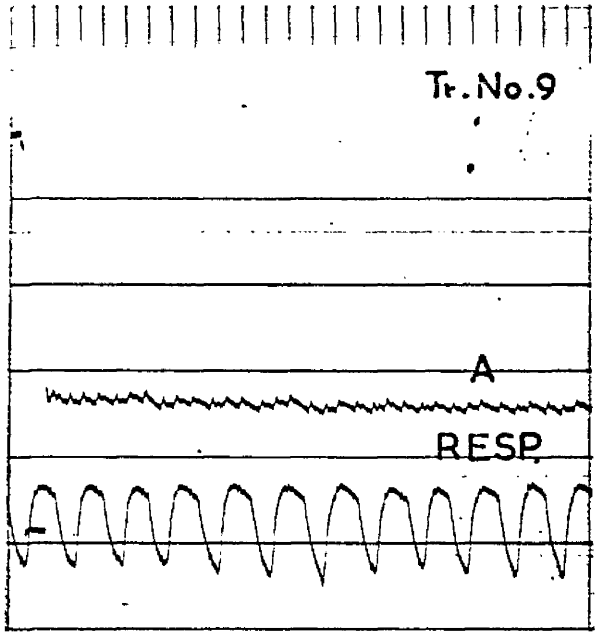
Pressure Tracing
20 sec



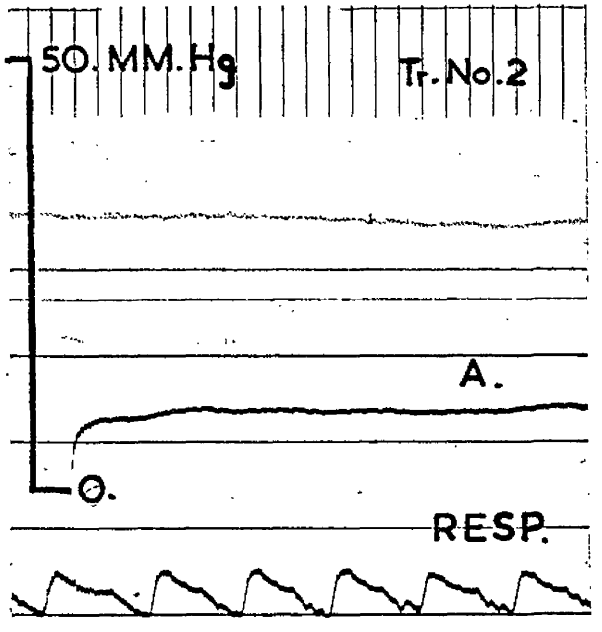
No 33 (A14) 2/2/62

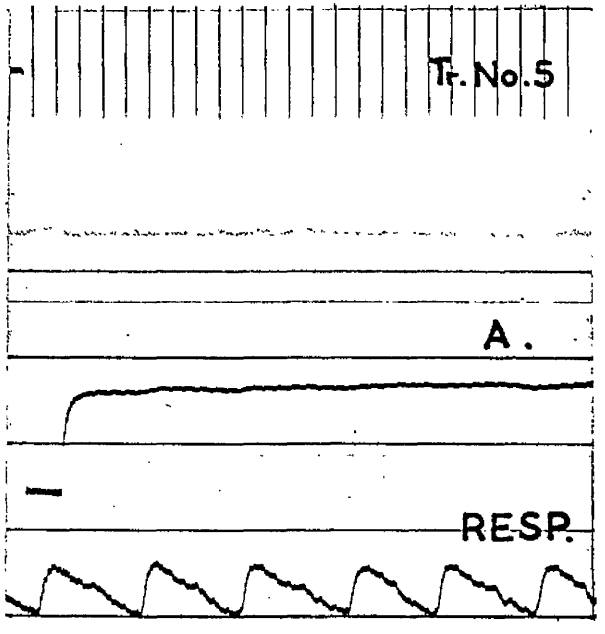


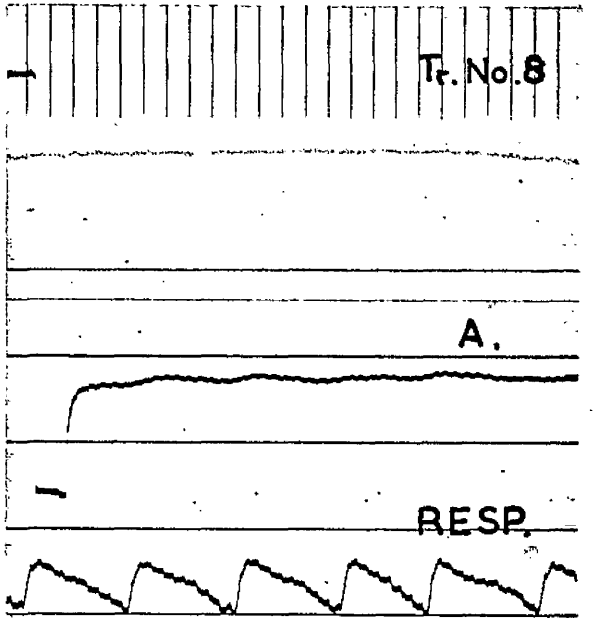




No 33 (A14) 9/2/62



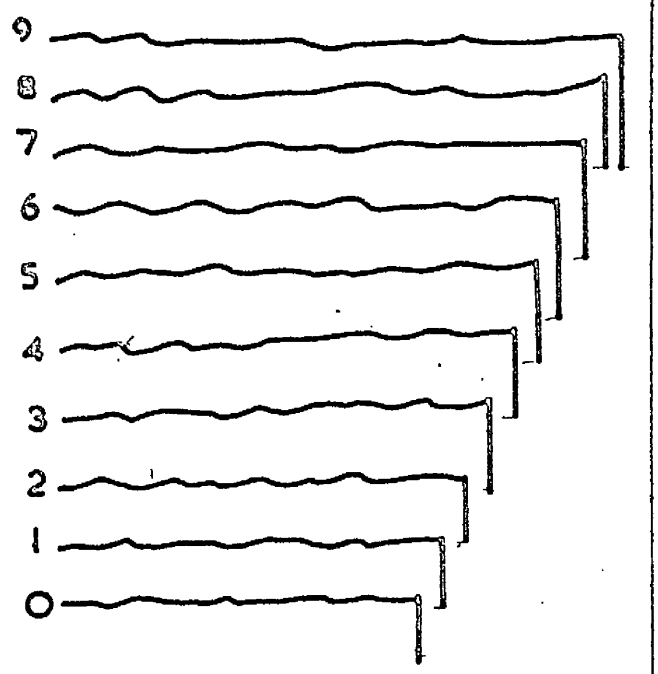




A.14 (No.33) 2.2.62 Auto.Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	6 - 7 (0)
5	2	8 - 9 (1)
10	22	7 - 9 (2)
15	10	9 - 11 (3)
20	18	9 - 11 (4)
25	18	10 - 12 (5)
30	23	12 - 15 (6)
35	30	12 - 15 (7)
40	24	8 - 11 (8)
45	22	15 - 18 (9)

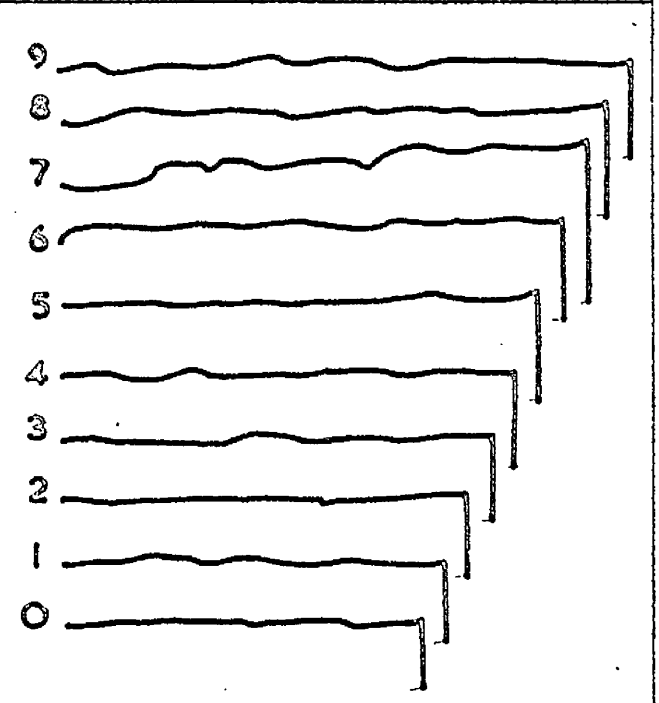
Pressure Tracing
20 sec



A.14 (No.33) 9.2.62 Auto.Kidney Osmotic Diuresis

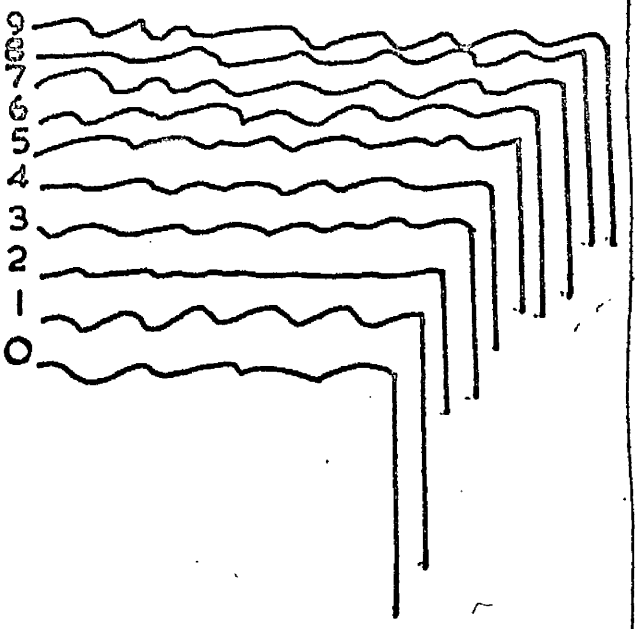
Time (Mins)	Urine/5 min Auto.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	7 - 9 (0)
5	2	9 - 10 (1)
10	2	10 - 11 (2)
15	5	10 - 11 (3)
20	12	10 - 11 (4)
25	15	12 - 13 (5)
30	21	11 - 13 (6)
35	22	13 - 20 (7)
40	8	11 - 14 (8)
45	0	10 - 12 (9)

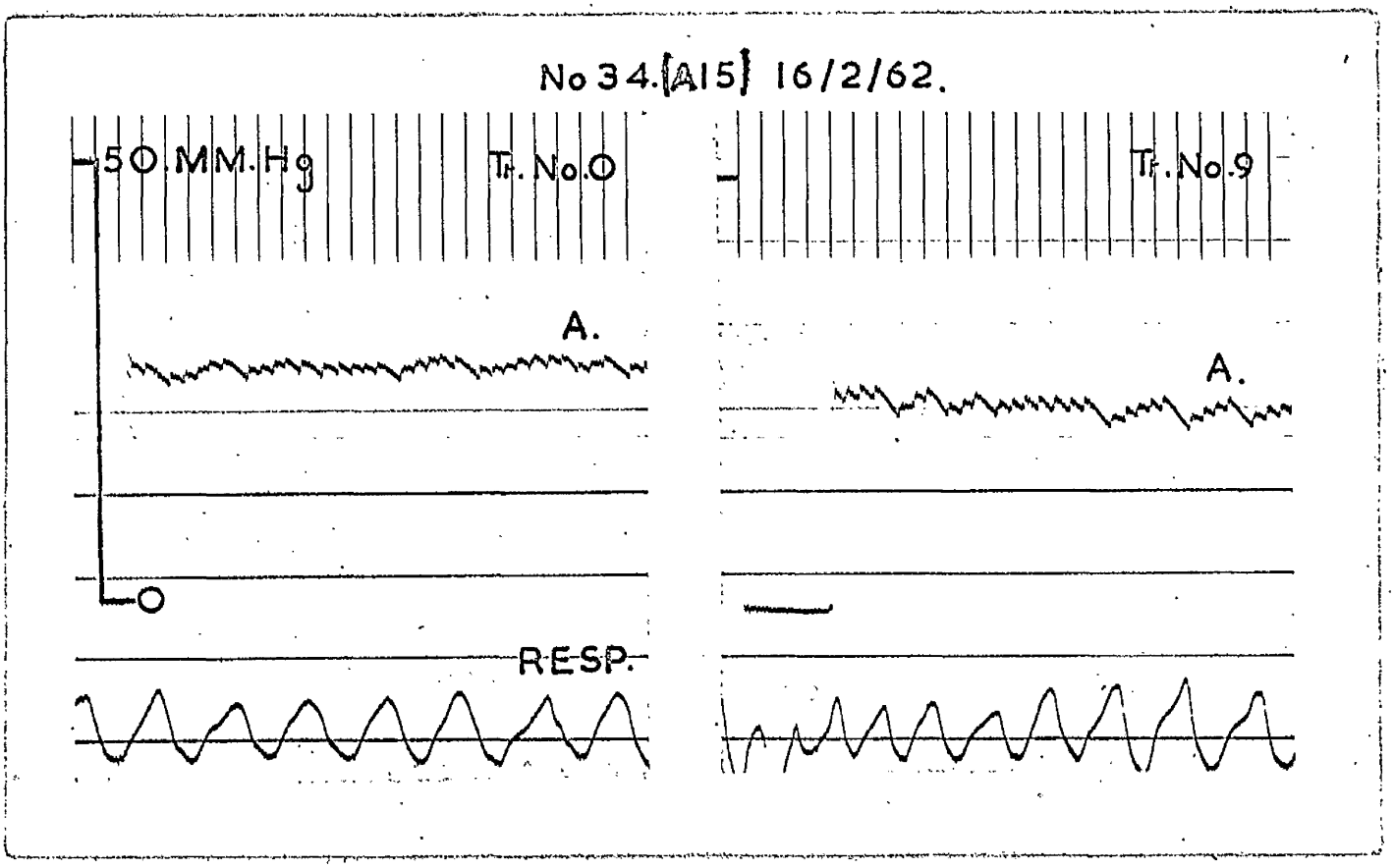
Pressure Tracing
20 sec



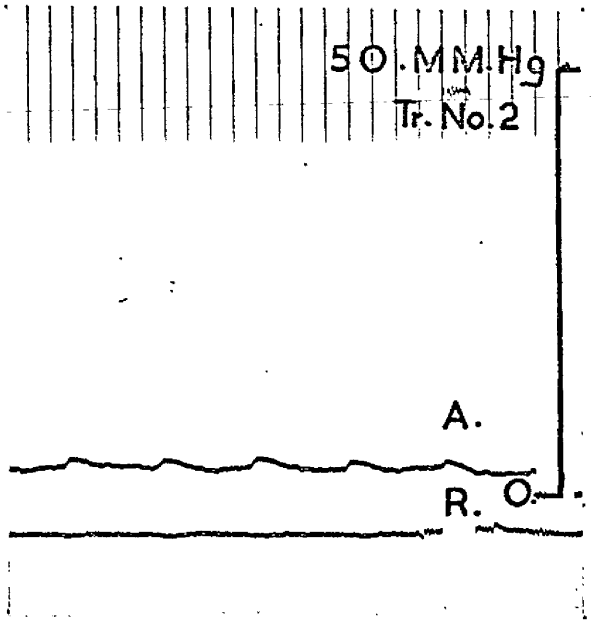
A.15 (No.34) 16.2.62 Auto kidney Osmotic Diuresis

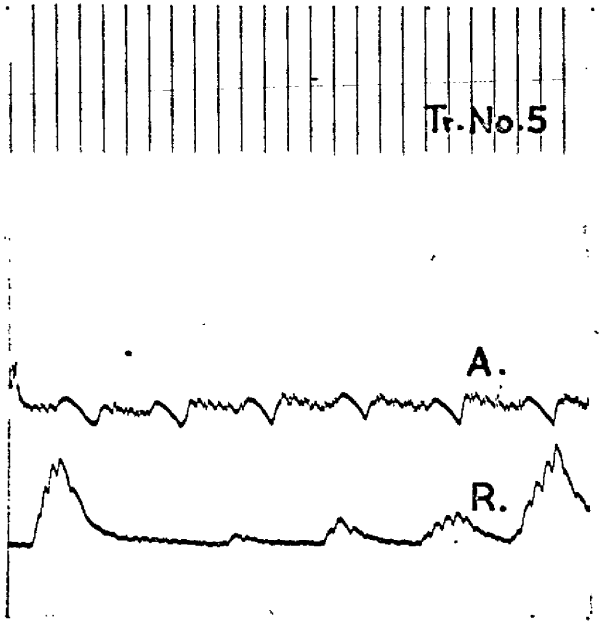
Time (Mins)	Urine/5 min	Pelv. Press mm Hg. (Tr.No)	Pressure Tracing ——— 20 sec ———
Basal	- mls	27 - 31 (0)	
5	5	28 - 32 (1)	
10	12	17 - 18 (2)	
15	5	20 - 23 (3)	
20	17	18 - 22 (4)	
25	10	20 - 22 (5)	
30	11	23 - 26 (6)	
35	12	23 - 28 (7)	
40	14	23 - 26 (8)	
45	13	25 - 29 (9)	

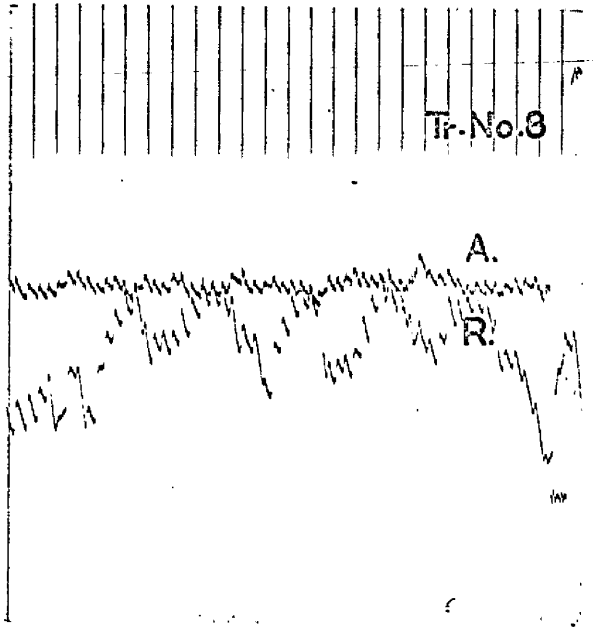




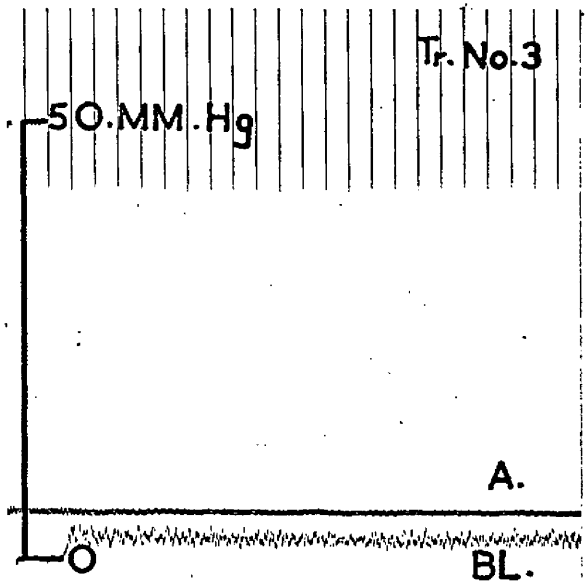
No.43 (A22) 25/5/62

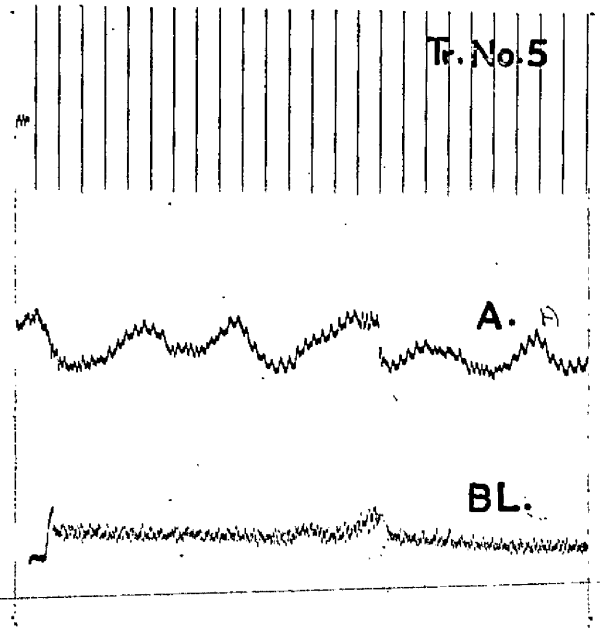


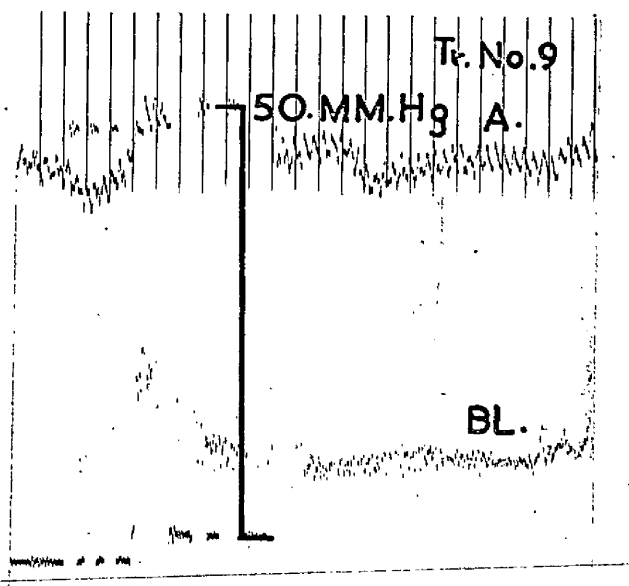




No.43 (A22) 31/5/62



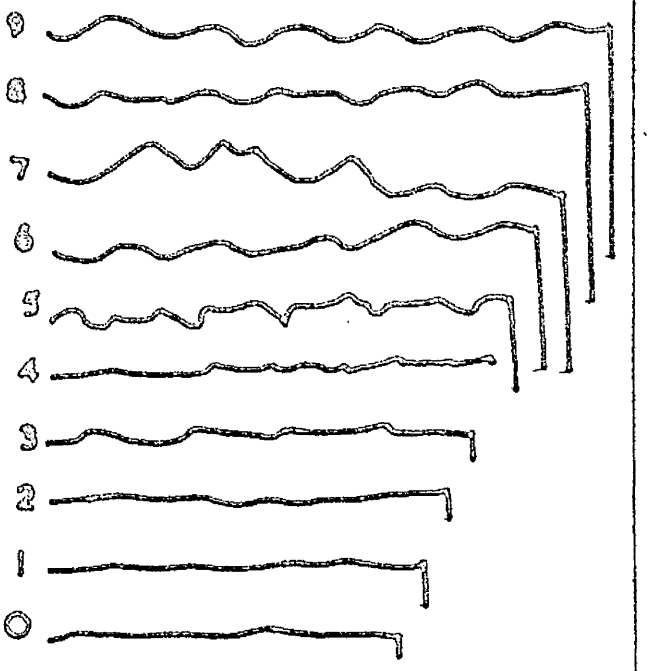




A.22 (No.43) 25.5.62 Auto Kidney Osmotic Diuresis

Time (Mins)	Blad.Press mm Hg.	Pelv. Press mm Hg. (Tr.No)
Basal	5	3 - 4 (0)
5	5	3 - 5 (1)
10	6	2 - 5 (2)
15	7	2 - 6 (3)
20	7	2 - 2 (4)
25	8	6 - 11 (5)
30	8	12 - 19 (6)
35	11	20 - 28 (7)
40	15	23 - 27 (8)
45	14	25 - 30 (9)

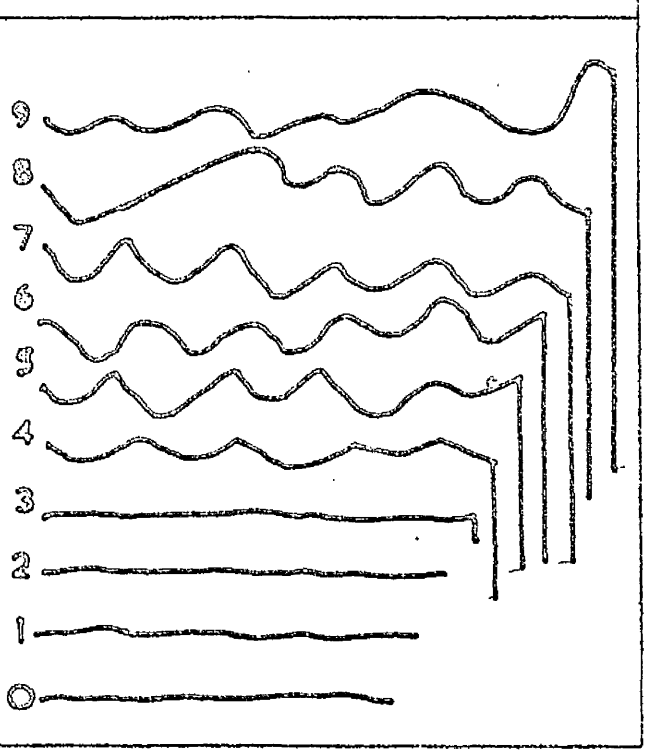
Pressure Tracing — 20 sec



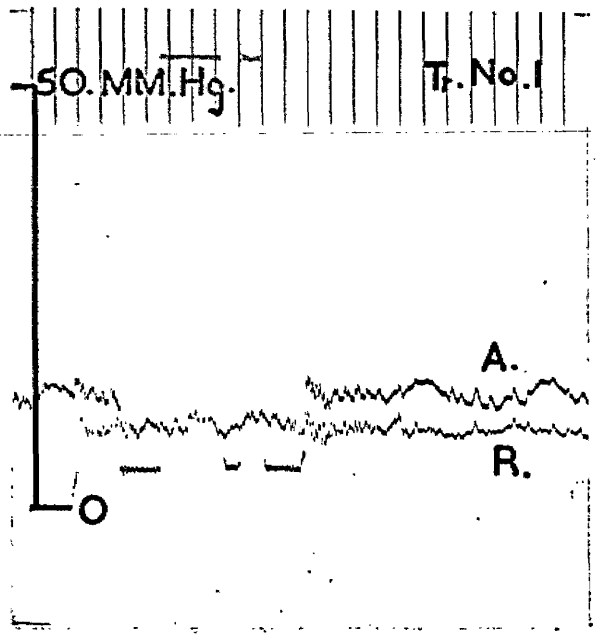
A.22 (No.43) 31.5.62 Auto Kidney Osmotic Diuresis

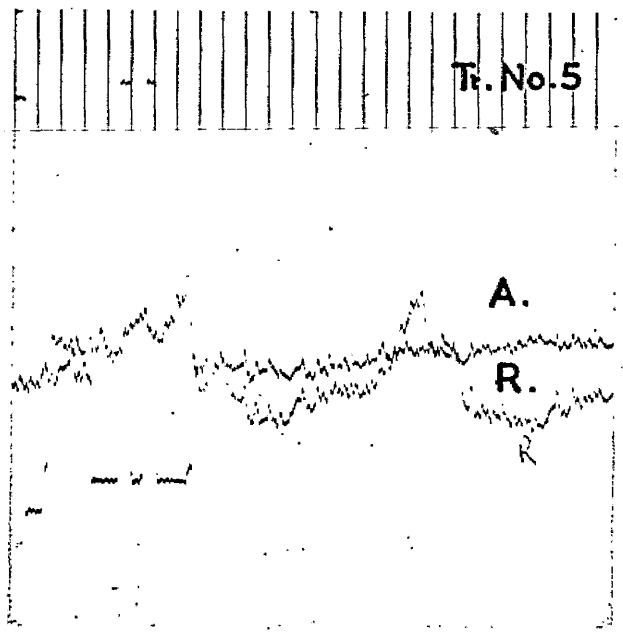
Time (Mins)	Blad.Press mm.Hg	Pelv. Press mm Hg. (Tr.No)
Basal	0	0 (0)
5	0	0 (1)
10	0	0 (2)
15	3	3 (3)
20	3	15 - 20 (4)
25	4	19 - 24 (5)
30	5	24 - 32 (6)
35	6	31 - 38 (7)
40	9	31 - 42 (8)
45	9	40 - 49 (9)

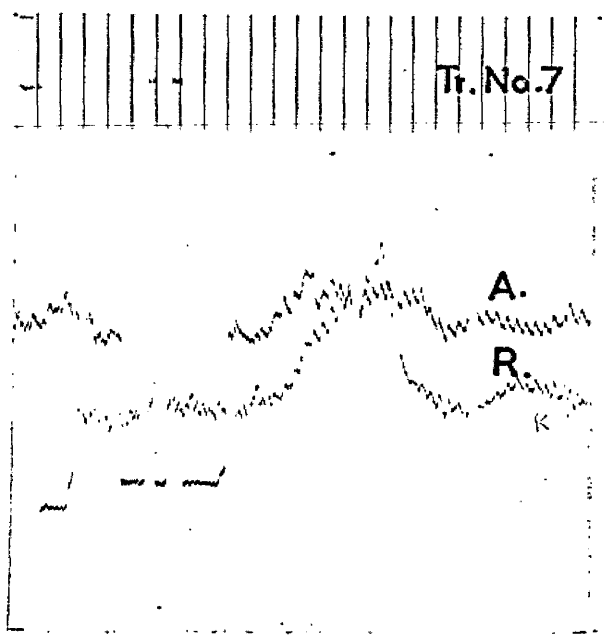
Pressure Tracing — 20 sec



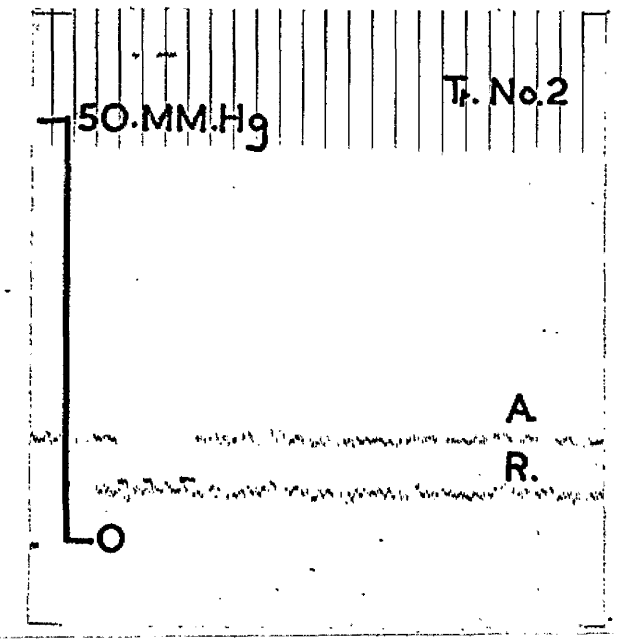
No.43 (A22) 25/6/62

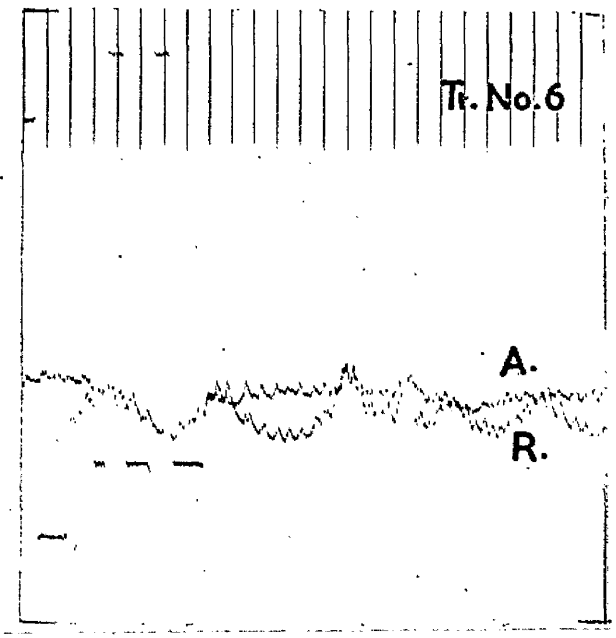


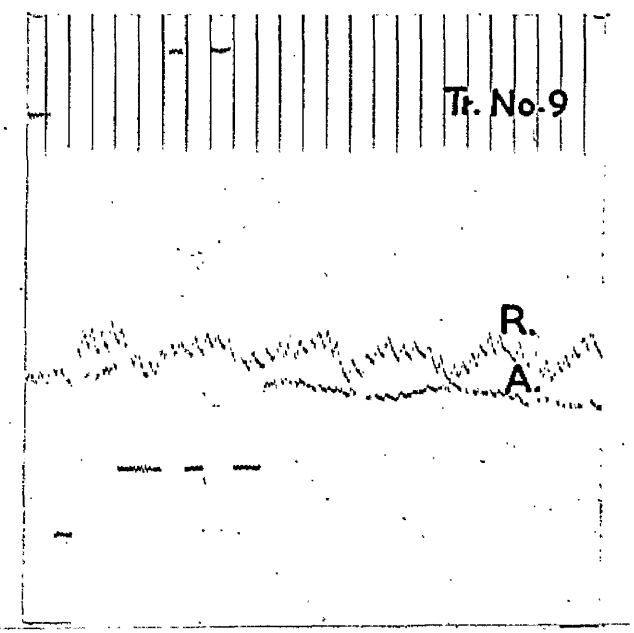




No.43 (A22) 16/8/62



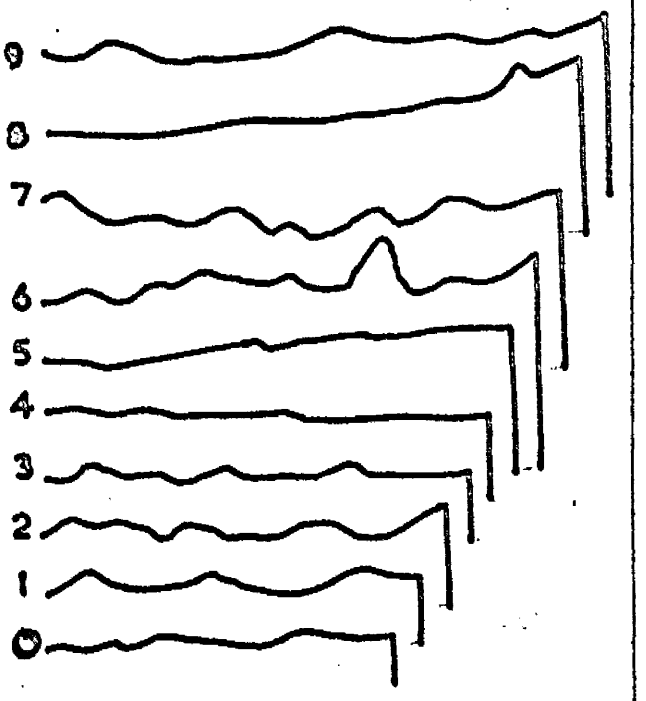




A.22 (No.43) 25.6.62 Auto Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto & R.Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	5 - 7 (0)
5	12	6 - 10 (1)
10	30	7 - 11 (2)
15	39	6 - 10 (3)
20	46	10 - 12 (4)
25	52	12 - 18 (5)
30	68	19 - 27 (6)
35	78	15 - 22 (7)
40	96	11 - 20 (8)
45	72	16 - 21 (9)

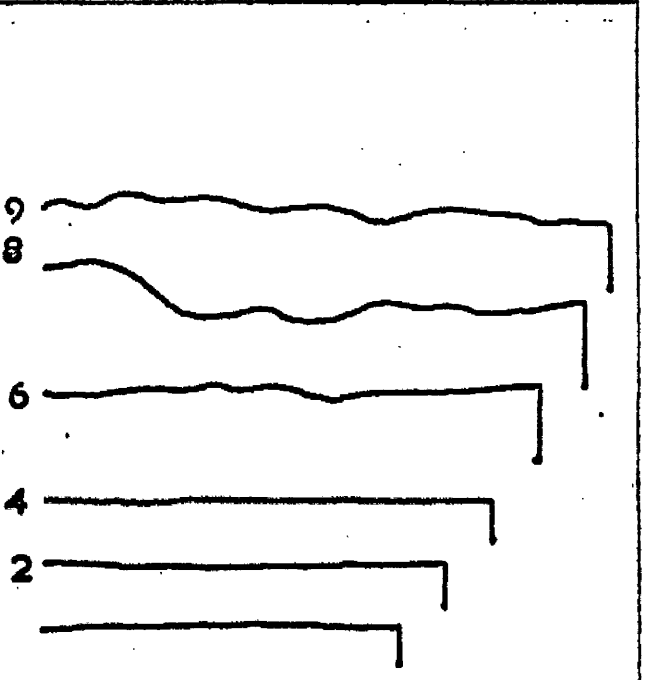
Pressure Tracing — 20 sec



A.22 (No.43) 16.8.62 Auto Kidney - Ureteric Implant Osmotic Diuresis

Time (Mins)	Urine/5 min. Auto & Rt. Kid.	Pelv. Press mm Hg. (Tr.No)
Basal	- mls.	5-6 (0)
5	18	4-6 (1)
10	28	4-6 (2)
15	33	4-6 (3)
20	39	4-6 (4)
25	55	4-6 (5)
30	65	6-10 (6)
35	70	13-18 (7)
40	88	8-15 (8)
45	103	7-11 (9)

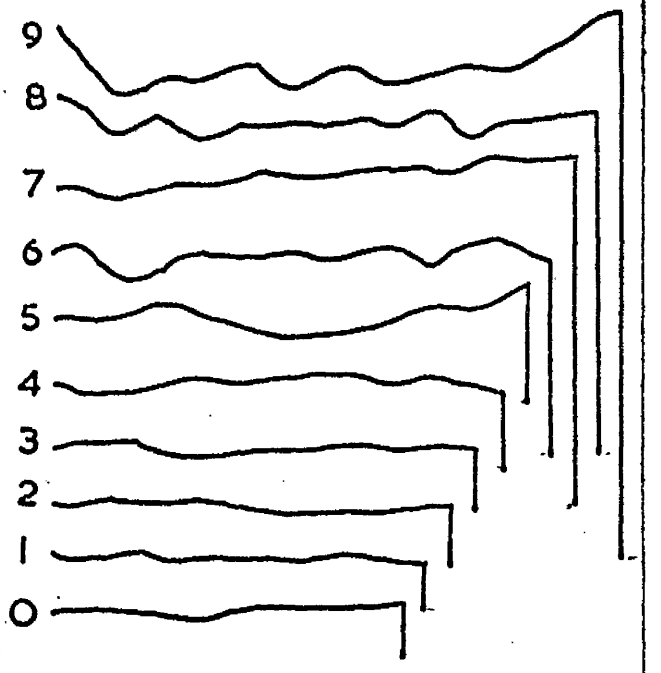
Pressure Tracing — 20 sec

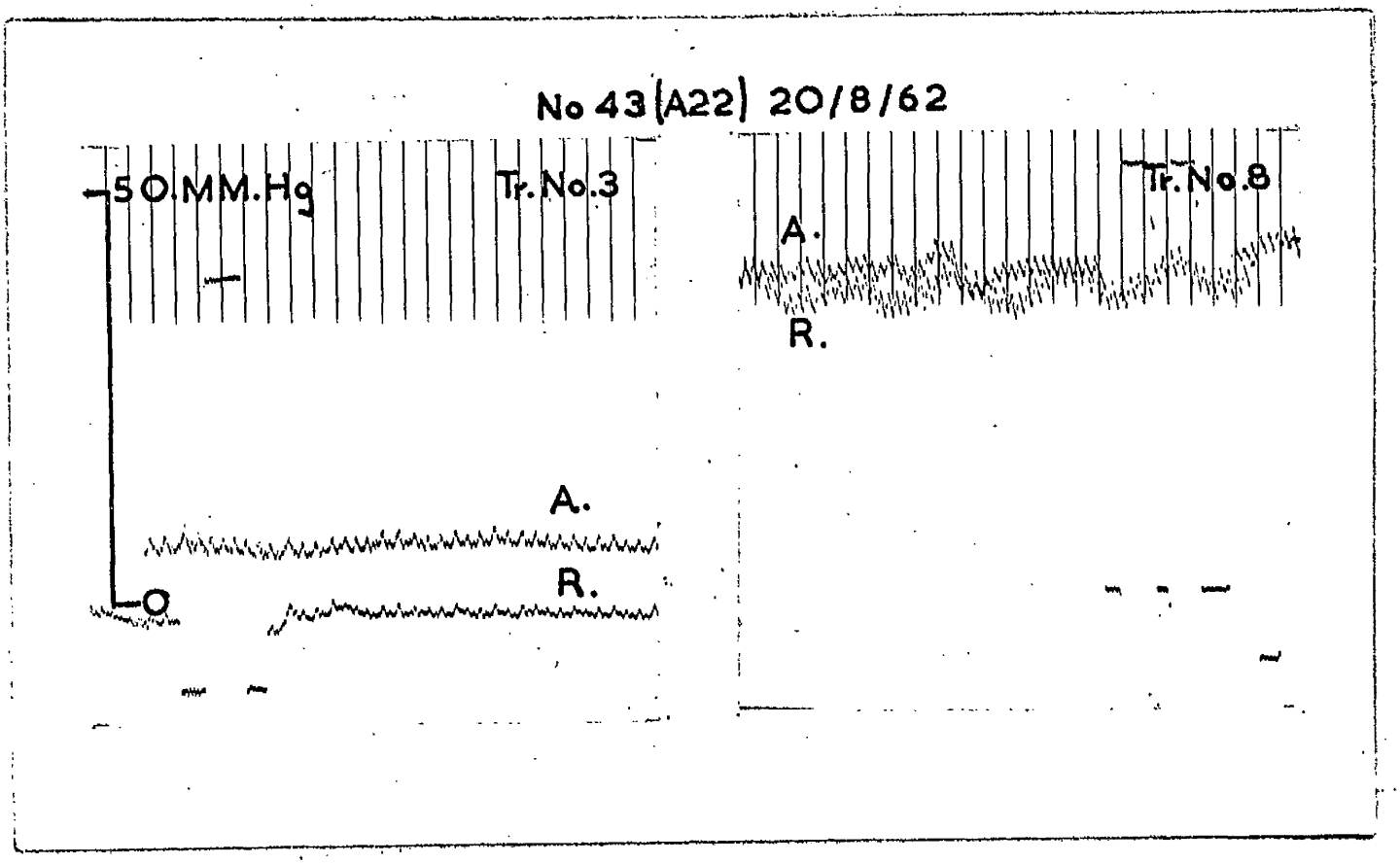


A 22 (No.43) 20.8.62 Auto Kidney - Ureteric Implant Osmotic Diuresis

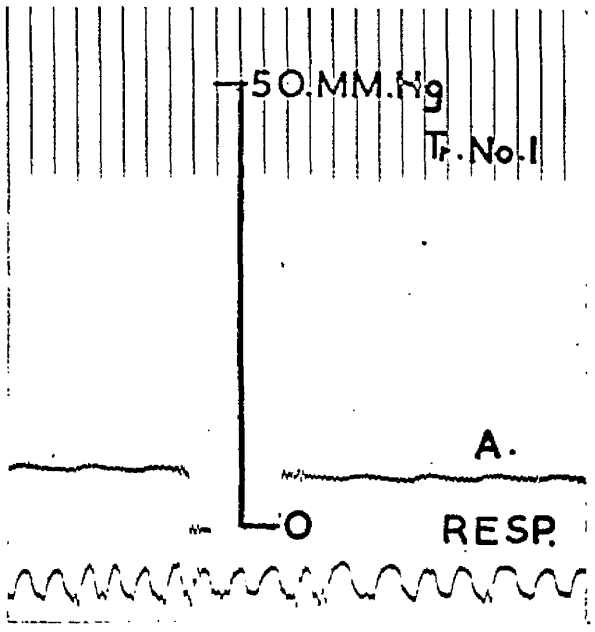
Time (Mins)	Blad. Press. mm. Hg.	Pelv. Press mm Hg. (Tr.No)	
Basal	6	4-6	(0)
5	7	5-7	(1)
10	6	5-7	(2)
15	8	5-7	(3)
20	10	8-12	(4)
25	14	7-14	(5)
30	18	19-26	(6)
35	32	34-41	(7)
40	33	36-44	(8)
45	48	55-65	(9)

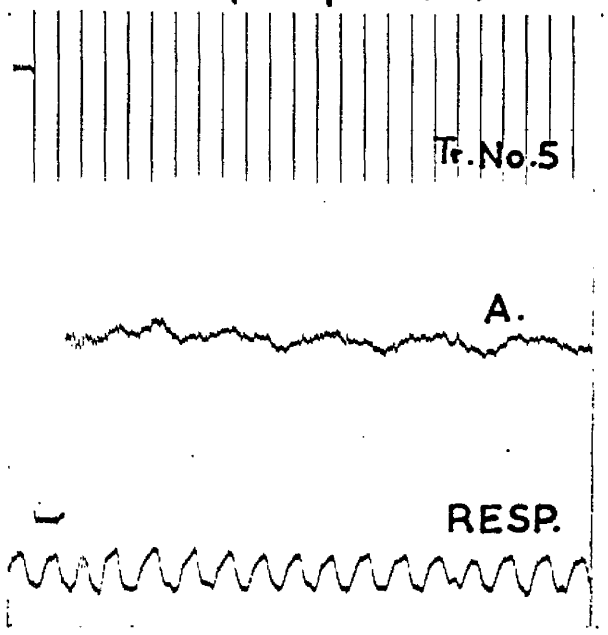
Pressure Tracing
20 sec

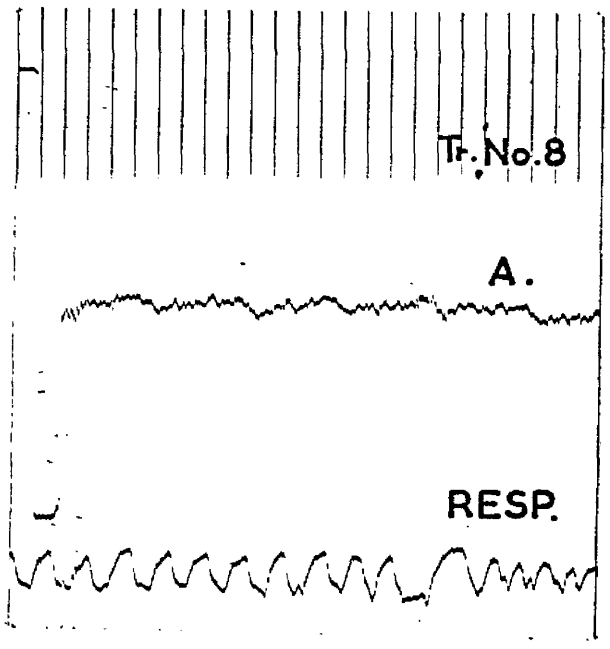




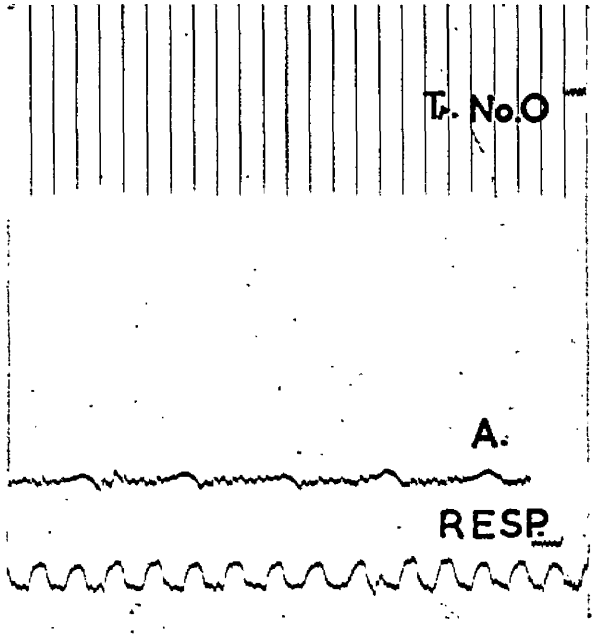
No.44 (A23) 18/6/62

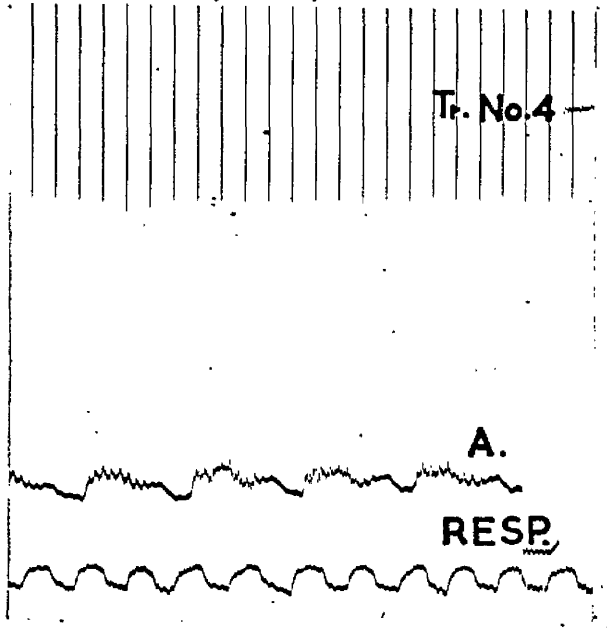


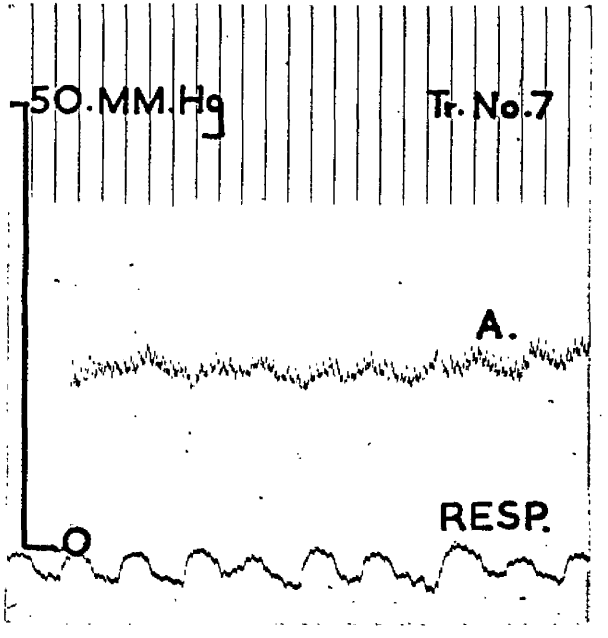




No.44 (A23) 3/7/62



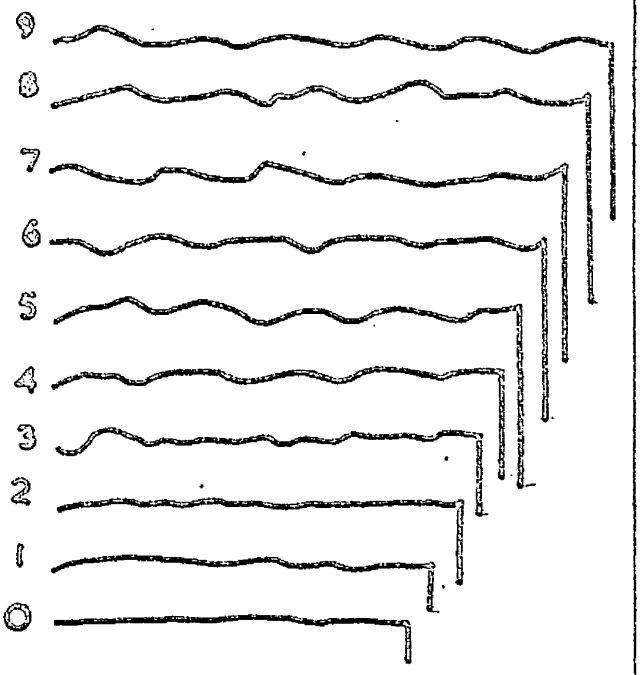




A.23 (No.44) 18.6.62 Auto Kidney Osmotic Diuresis

Time (Mins)	Urine/5 min Auto Kid	Pelv. Press mm Hg. (Tr.No)
Basal	- mls	5 - 6 (0)
5	2	5 - 8 (1)
10	3	9 - 11 (2)
15	9	8 - 11 (3)
20	20	11- 13 (4)
25	32	19 - 24 (5)
30	15	19 - 22 (6)
35	26	20 - 25 (7)
40	24	23 - 26 (8)
45	25	20 - 25 (9)

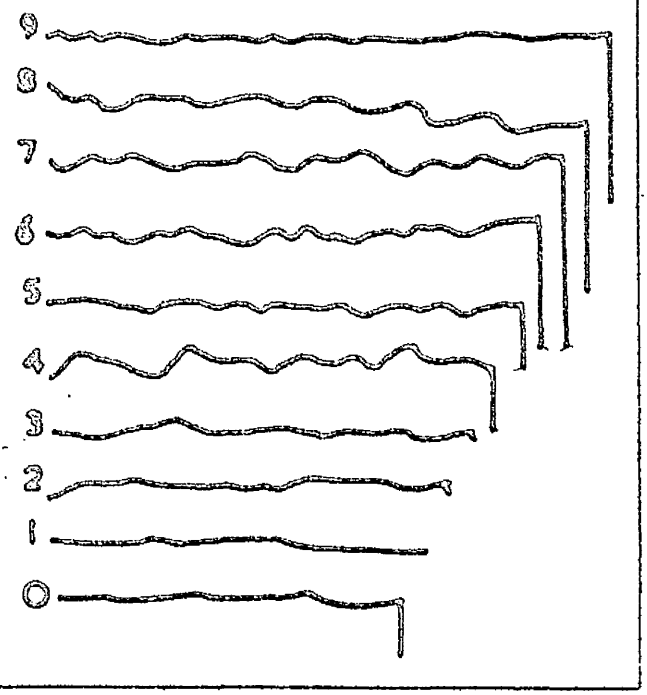
Pressure Tracing — 20 sec



A.23 (No.44) 3.7.62 Auto Kidney Osmotic Diuresis

Time (Mins)		Pelv. Press mm Hg. (Tr.No)
Basal	- mls	6 - 8 (0)
5	2	0 - 1 (1)
10	5	2 - 4 (2)
15	11	2 - 4 (3)
20	17	7 - 12 (4)
25	25	7 - 10 (5)
30	33	12 - 16 (6)
35	31	20 - 24 (7)
40	28	19 - 25 (8)
45	21	20 - 21 (9)

Pressure Tracing — 20 sec



ILLUSTRATIONS

FIG:	CAPTION
1	Permanent nephrostomy tube
2	Section of kidney showing tip of manometric tube in renal pelvis.
3	Dog in Pavlov stand during pressure recording experiment
4	Dog lying in trough during pressure recording experiment
5	Stockinette coat rolled to display manometric tube
6	Effect of respirations on pressure record
7	Effect of pulse beat on pressure record.
8	Irregular waves produced by muscular movements.
9	Excretion pyelogram illustrating normal appearance of renal pelves in right and left (autotransplanted) kidneys.
10	Two minute recording from renal pelves Dog 8, illustrating low basal pressures. Calibration 50 mm.Hg. Signals denote second intervals.

FIG:	CAPTION:
11	Illustrates regular phasic contraction waves in right (R) pelvis during diuresis
12A	Maximum and minimum pelvic pressures related to urine flow rates during diuresis - dog 20.
12B	Maximum and minimum pelvic pressures related to urine flow rates during diuresis - dog 20.
12C	Recordings of the pressures in both renal pelves - dog 20
13A	Pelvic pressures related to increase in bladder pressure during osmotic diuresis
13B	Recordings of the pressures in the bladder and renal pelves
14	Acute increase in bladder pressure during diuresis results in rise in renal pelvic pressure
15	At comparable urine flow rates, alterations in bladder pressure are directly reflected by changes in intrapelvic pressure.
16	The pulse wave produces a greater deflection in the pelvic pressure trace during diuresis.

FIG:	CAPTION:
17	Illustrates the fall in pelvic pressure caused by catheter drainage of the bladder during diuresis
18	Pelvic pressure in normal (N) and autotransplanted (A) kidneys restored to original levels by refilling bladder.
19	Pelvic pressures related to urine flow rate and bladder pressure during water diuresis
20	Two minute recording from pelvis of autotransplanted kidney, Dog 22, illustrating low basal pressure.
21	Obstruction at uretero-vesical anastamosis after renal transplant results in high basal and diuretic pelvic pressures.
22	Descending pyelogram shows hydronephrosis after renal transplantation
23	Temporary nature of increase of pelvic pressure after kidney transplant.
24A	Pelvic pressure in the autotransplanted kidney related to urine flow rate and bladder pressure during osmotic diuresis
24B	Recordings of the pelvic pressures in the autotransplanted kidney.

FIG:	CAPTION:
25	Descending pyelogram, Dog 31, shows normal upper urinary tract in autotransplanted kidney.
26	Descending pyelogram, Dog 31, shows hydronephrosis, basal pelvic pressure 8-12 mm.Hg.
27	Illustrates changes in pelvic and ureteric pressures following acute ureteric obstruction.
28A	High resting pelvic pressure after uretero-colic anastamosis further increased by osmotic diuresis
28B	Recordings of the pressures in the renal pelvis.
29	Illustrates the gradual fall in basal and maximal diuretic pressures in the renal pelvis after uretero-colic anastamosis.
30	Radiograph of injected post-mortem specimen showing stenosis at the uretero-colic anastamosis.
31	Illustrates rapid fall in pelvic pressure at end of saline infusion.
32	High pelvic contraction waves produced by prolonged infusion.

FIG:		CAPTION:
33		Direct infusion of left renal pelvis at 7 ml/min (Tr.1) and 12 ml/min (Tr.2) raise pressure 8 and 10 mm.Hg respectively. Infusion at 4 ml/min (Tr.3) at beginning of standard diuresis produces 4 mm.Hg. increase in pressure whereas the same rate at mid point of standard diuresis produces 8 mm.Hg. increase
34		Pyelograms made in Dog 45 during the infusion experiments illustrated in Fig.33. Less contrast obtained during diuresis but no significant alteration in pelvic contour.
35		"Ureteric" waves recorded from left manometric tube in extrarenal pelvis.
36		Tracing of pyelogram indicating position of tips of manometric tubes in pelvis (P) and pelvi-ureteral (PU) zone.
37	A	Pelvic (P) and pelvi-ureteral (PU) basal pressure traces similar
	B	Pelvi-ureteral (PU) basal pressure trace of "ureteric" type
	C	At high urine flow rates similar wave form and pressures in pelvis (P) and pelvi-ureteral (PU) zone
38		Change in form of wave at pelvi-ureteral zone caused by higher rate of infusion.

FIG.	CAPTION
39	Different forms of ureteric wave recorded from left ureter
40A	Pelvic and ureteric pressures recording during diuresis
40B	Common wave pattern in pelvis and ureter at high urine flow rates
41	Illustrates the effect of increased intro-abdominal pressure on pelvic and ureteric pressures.
42	Illustrates satisfactory position of ureteric T-tube
43	T-tube rotated causing hydronephrosis