Transureteral Phonocatheter Recording of the Renal Arterial Pulse

The Phonorenogram

By HERBERT L. TANENBAUM, M.D., and WILLIAM S. KISER, M.D.

IN 1929 Forssmann introduced a ureteral catheter into his antecubital vein and initiated the era of cardiac catheterization. This technic has led to a greater understanding of cardiovascular physiology. Our debt to the urologists for the use of their catheter is long overdue. We have been interested in applying cardiac catheterization technics via retrograde ureteral catheterization in the study of renal vascular disease. Many patients with hypertension suspected of having renal vascular lesions undergo bilateral ureteral catheterization for split renal function tests. In light of the risk, discomfort, and expense of the procedure, it behooves the physician to obtain as much information from the study as possible without undue prolongation or additional risk. The studies to date are primarily concerned with demonstrating increased water or sodium reabsorption as secondary effects of renal ischemia. Experimental evidence suggests that one of the important stimuli for the development of renal hypertension is a reduction of renal arterial pulse pressure followed by liberation of renin from the ischemic kidney. Under these circumstances, renal pulse examination should be of interest in the study of hypertension. Katz explored this concept with photoelectric pulse recordings from direct fixation on the exposed kidney of dogs. He demonstrated that in dogs, as well as in human subjects, stenosis significant enough to initiate hypertension was associated with an altered kidney pulse. Previous experimental studies in our laboratory have explored this further by direct ureteral pressure recordings following renal artery constriction. In the dog, after constriction of the main renal artery or a segmental branch, a decrease in the mean hydrostatic pressure and the transmitted renal arterial pulse pressure in the respective ureter was recorded. The present study applies these observations by the recording of the transmitted renal arterial pulse in human

Figure 1

Schematic drawing showing the position of the phonocatheter in the ureter in relation to the iliac and renal artery. Representative pulse tracings are shown at each site.

From the Cardiopulmonary Laboratory, Washington Hospital Center, Washington, D. C., and the Surgery Branch, National Cancer Institute, Bethesda, Maryland.

Supported in part by Research Grant HE-8563-01 from the National Heart Institute, U. S. Public Health Service.
Essentially be high-frequency paired at ter. The iliac e.p.s.
Both amplifiers were made of the middle (barium
wenit general or saddle-block anesthesia. Follo-
the With
were
smooth and phoInocatheter signed preamplifier-
Figure 1, phono- catheter- made through the D.C. input of either a photo-
graphic multichannel recorder † or a direct-writing
electrocardiographic amplifier. The frequency re-
sponse of the phonocatheter ⁹ has been shown to
be essentially constant over a frequency range of
180 to 20,000 c.p.s. The frequency response of the
preamplifier has a flat response from 30 to 10,000
c.p.s. Both low-frequency pulse displacement and
high-frequency sounds may be recorded and com-
pared at the same amplification setting.

As illustrated in figure 1, pulse and sound rec-
orderings were made at different levels in the ure-
eter. The iliac artery pulse was recorded at the
level of the middle and lower third of the ureter.
With the phonocatheter in the renal pelvis, it was
possible to record the pulsations of the respective
renal artery. Gentle manipulation allowed move-
ment of the catheter tip in exploring the renal pel-
vis. Simultaneous electrocardiographic or pulse
determinations correlated the recorded pulse with
the heart rate.

Results
The iliac and renal artery pulse recordings were
comparatively analyzed as to relative amplitude and the presence or absence of
bruits. Recorded deflections both in systole and in diastole are often noted. Representative
tracings of four patients are presented with
associated split renal function and arterio-
graphic studies.

Case 1. Figure 2 demonstrates the renal ar-
tery pulsations in a hypertensive patient with
no supporting evidence of renal artery disease. The pulsations are noted to follow the
QRS complex of the electrocardiogram and are of comparable amplitude.

Case 2. By contrast, G.W. is a 52-year-old
man with hypertension of 5 years’ duration un-
controlled by medical therapy. A bruit was
heard over the right side of the abdomen and
interpreted as arising from the renal artery.
Split renal function determinations (table 1)
indicated ischemia of the right kidney. Anato-
mic confirmation of a stenotic lesion of the
right main renal artery is noted on the renal
arteriogram (fig. 3). It is of interest that there

* American Electronic Laboratories, Inc., Colmar,
Pennsylvania.
† Electronics for Medicine, White Plains, New York.
‡ Sanborn Twin-Viso, Waltham, Massachusetts.

Figure 2
Equal renal artery pulse tracings in a hypertensive pa-
tient without renal artery occlusive disease. Simultane-
ous electrocardiographic recording.

Figure 3
Arteriogram of patient G.W. (case 2), demonstrating
marked irregularity of the abdominal aorta and occlu-
sion of the right main renal artery at its origin. Also
noted is an apparent narrowing of the left main renal
artery.
Table 1

Differential Renal Function Studies

<table>
<thead>
<tr>
<th>Patient</th>
<th>Vol., ml./min</th>
<th>Urine Cr., mg./ml</th>
<th>Urine PAH, mg./ml</th>
<th>Urine sodium, mEq./L</th>
<th>GFR, ml./min</th>
<th>RPF, ml./min</th>
</tr>
</thead>
<tbody>
<tr>
<td>G.W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>0.15</td>
<td>1.54</td>
<td>1865.0</td>
<td>14.9</td>
<td>12.5</td>
<td>83.2</td>
</tr>
<tr>
<td>Left</td>
<td>6.4</td>
<td>0.20</td>
<td>173.8</td>
<td>53.2</td>
<td>74.6</td>
<td>324.7</td>
</tr>
<tr>
<td>L.W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>0.7</td>
<td>0.365</td>
<td>548.7</td>
<td>119.0</td>
<td>31.1</td>
<td>124.0</td>
</tr>
<tr>
<td>Left</td>
<td>3.7</td>
<td>0.143</td>
<td>233.7</td>
<td>114.0</td>
<td>62.5</td>
<td>305.0</td>
</tr>
<tr>
<td>R.L.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>0.7</td>
<td>0.320</td>
<td>345.0</td>
<td>159.0</td>
<td>10.8</td>
<td>113.0</td>
</tr>
<tr>
<td>Left</td>
<td>5.4</td>
<td>0.205</td>
<td>245.0</td>
<td>125.0</td>
<td>60.8</td>
<td>648.0</td>
</tr>
</tbody>
</table>

Values are average of three consecutive 10-minute collection periods with 4 per cent urea, PAH, ADH, 0.9 per cent saline infusion.

Cr., creatinine; PAH, para-aminomipiric acid; GFR, glomerular filtration rate; RPF, renal plasma flow.

was also noted an apparent narrowing of the left renal artery as well. The phonocatheter pulse recording (fig. 4) shows a distinct difference of the pulse with a marked diminution in amplitude on the right side. Also, a systolic bruit was recorded over the right iliac artery and not in the region of the renal artery. At surgery, a gradient of 100 mm. Hg was recorded across a stenotic lesion of the right main artery. There was no gradient or anatomic constriction of the left renal artery. The bruit noted on physical examination was found to arise from plaques in the lower abdominal aorta and not from the renal artery constriction. Following a right nephrectomy, the patient has been normotensive.

Comment: The pulse recordings correlated with the physiologic studies and pathologic findings. The artifactual narrowing of the left renal artery on the renal angiogram was questioned by the normal pulse from that side. Finally, it should be pointed out that not all
abdominal bruits in hypertensive patients originate from stenotic renal arteries. In this patient, the bruit recorded over the iliac artery and not from the renal pelvis suggested its origin from the aorta as was confirmed at surgery.

Case 3. L.W. is a 49-year-old woman with hypertension of 6 years’ duration. The split renal function study (table 1) suggests ischemia of the right kidney. Renal arteriography (fig. 5) demonstrates bilateral medial hyperplasia, more marked on the right, and a small aneurysm of the renal artery on the right. The right kidney was 2 cm. smaller than the left. In spite of bilateral disease, the renal pulse tracings (fig. 6) showed a greater diminution of the renal artery pulse on the right side. At surgery, a gradient of 115 mm. Hg was recorded across the stenotic right renal artery. Following nephrectomy, the patient has been normotensive.

Comment: Although both renal function studies and angiography demonstrated right-sided impairment, there was evidence of bilateral disease. The pulse tracings supported the finding of more significant involvement on the right. No bruits were recorded and none were noted at surgery.

Case 4. R.L. is a 21-year-old man with severe hypertension (280/130) of 3 years’ duration. An intravenous pyelogram showed atrophy of the right kidney and enlargement of the left kidney. Renal angiograms demonstrated stenosis of the left main renal artery. Split renal function tests prior to this study confirmed the impression of ischemia of the left kidney. At surgery, a stenotic lesion of the left main renal artery was resected and an end-to-end anastomosis was performed. A biopsy of the right kidney showed chronic pyelonephritic-nephrosclerotic changes. Postoperatively, the blood pressure has been modified, but is not normal. A right nephrectomy is now being considered. Table 1 and figure 7 present the postoperative studies. Note the marked increase in renal plasma flow on the vascular repaired left side. The renal artery pulse is likewise significantly increased in amplitude and sharpness. The pulse recording from the atrophic right side is relatively normal in appearance.

Comment: One of the obvious problems that arises from this study is whether the pulse from an atrophic pyelonephritic kidney could be recorded to distinguish the smaller kidney with parenchymal disease from a primary vascular lesion. The normal pulse from the small kidney in this patient supports the integrity of the main renal vascular supply. Further studies are needed to establish the validity of this observation.
Discussion

One of the principal methods of vascular investigation has been the direct palpation of accessible pulsating vessels. Assessment of such features as relative amplitude, pulse pressure, rate of pressure change, and the presence or absence of thrills is of great value and is easily obtainable from superficial vessels. Such information should be of equal importance in patients with renal artery disease if the renal arterial pulse could be palpated in the intact subject. Phonocatheters have been widely used with cardiac catheterization as a means of recording intracardiac sounds, murmurs, and vascular bruits. As demonstrated, this same technic can be used to record the pulse from the iliac and renal arteries indirectly through the ureter and the tracings compared to the contralateral side. The “pulses” recorded in this study probably represent low-frequency mechanical impacts on the ureter and renal pelvis by the respective iliac and renal arteries.

There are certain obvious limitations. Bilateral vascular disease would affect both pulses. As noted in case 3, however, one side may be relatively more stenotic and amenable to repair. Further, small branch or parenchymal arterial lesions may be missed as indeed they sometimes are by all present technics including aortography. The recording of a normal pulse from an atrophic pyelonephritic kidney (case 4) is of interest, suggesting this technic as a means of assessing the main vascular arterial supply without recourse to arteriography. Also, what may be significant stenosis by angiography may not be significant physiologically and any associated hypertension may be a coincidence rather than a consequence of the apparent renal artery lesion. The artifactual narrowing noted in case 2 was questioned by the normal pulse recorded from that side.

Abdominal bruits described on clinical examination have been stressed as an important sign in evaluating patients with hypertension. However, one must not be misled as to the origin of the bruit. Many hypertensive patients have vascular disease and arteriosclerotic plaques involving the abdominal aorta that generate bruits with no involvement of the renal artery. The origin of these sounds may be clarified by the recordings over the iliac and renal artery.

More data must be collected to ascertain whether anatomic variations of the renal pelvis-renal artery relationship may make it impossible to record the transmitted renal artery pulse. Large amounts of perirenal fat may dampen the pulse tracing. This has not been the case in the patients and controls studied to date.

Until reliable and readily available blood angiotensin measurements are perfected, there is no shortcut to the selection of patients who will be improved by surgery. Since many aspects of this condition remain obscure, full investigation is advisable before an operation is carried out. The indirect recording of the renal artery and iliac artery pulsation may add important information during the performance of a split renal function study without undue prolongation or additional risk.

Finally, studies are now in progress to use this technic to study beat-to-beat changes in the renal artery pulse during infusion of vasoressor and other pharmacologic agents. Prolonged clearance studies may miss transient changes that may be noted with continuous monitoring of the pulse during the collection period.
Summary
The renal artery and iliac artery pulses were recorded indirectly during split renal function studies in hypertensive patients suspected of having renal vascular lesions. A phonocatheter was easily passed up each ureter and the pulses were recorded at the level of the crossing of the iliac artery and again in the renal pelvis. In patients with renal vascular occlusion, there was a distinct diminution in the amplitude of the transmitted renal arterial pulse as compared to the unoccluded side. The recording of vascular bruits from each side was helpful in localizing their origin. Selected cases confirmed at surgery were presented.

Acknowledgment
We wish to thank Dr. Eugene Braunwald and Dr. A. G. Morrow of the National Heart Institute for their encouragement and advice.

References

Abdominal Aneurysm
Abdominal aneurysm, first diagnosed clinically by Vesalius, was recorded post-mortem by his contemporaries Falloppius and Ballonious . . . . .
Harvey in the De Motu Cordis commented on the difference in the pulse on the two sides. MacDonnell in 1850 and W. H. Walshe (1812-92) in 1853 drew attention to the condition of the pupil in aortic aneurysm; in 1854 Stokes recognized laryngeal paralysis . . . . Tracheal tugging was first, and in remarkably brief terms, described by W. S. Oliver in 1878.—Sir Humphry Davy Rolleston. The Harveian Oration. Great Britain, Cambridge University Press, 1928, p. 59.
Transureteral Phonocatheter Recording of the Renal Arterial Pulse: The Phonorenogram
HERBERT L. TANENBAUM and WILLIAM S. KISER

Circulation. 1964;29:688-693
doi: 10.1161/01.CIR.29.5.688

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1964 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/29/5/688

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/