World Journal of Urology (2019) 37:125–131
https://doi.org/10.1007/s00345-018-2378-4

INVITED REVIEW

Pressure matters: intrarenal pressures during normal and pathological conditions, and impact of increased values to renal physiology

Theodoros Tokas[1] · Thomas R. W. Herrmann[2] · Andreas Skolarikos[3] · Udo Nagele[1] · Training and Research in Urological Surgery and Technology (T.R.U.S.T.)-Group

Received: 27 April 2018 / Accepted: 12 June 2018 / Published online: 18 June 2018

Abstract

Purpose To perform a review on the latest evidence related to normal and pathological intrarenal pressures (IRPs), complications of incremented values, and IRP ranges during endourology.

Methods A literature search was performed using PubMed, restricted to original English-written articles, including animal, artificial model, and human studies. Different keywords were: percutaneous nephrolithotomy, PCNL, ureteroscopy, URS, RIRS, irrigation flow, irrigation pressure, intrarenal pressure, intrapelvic pressure and renal pelvic pressure.

Results Normal IRPs range from zero to a few cm H_2O. Pyelovenous backflow may occur at pressure range of 13.6–27.2 cm H_2O. During upper tract endourology, complications such as pyelorenal backflow, sepsis, and renal damage are directly related to increased IRPs. Duration of increased IRPs and concomitant obstruction are independent predictors of complication development.

Conclusions IRP increase remains a neglected predictor of upper tract endourology complications and its intraoperative monitoring should be taken into consideration. Further research is necessary, to quantify pressures generated during upper tract endourology, and introduce means of controlling them.

Keywords Percutaneous nephrolithotomy · PCNL · Ureteroscopy · URS · RIRS · Irrigation flow · Irrigation pressure · Intrarenal pressure · Intrapelvic pressure · Renal pelvic pressure

Introduction

Endourological upper tract treatment constitutes a main field in everyday urology. Retrograde intrarenal surgery (RIRS) and percutaneous nephrolithotomy (PCNL) constitute the main means of active renal stone treatment and are characterized by a huge variety of new techniques and instrumentation. To achieve better visibility during the procedure, irrigation flow (IF) and irrigation pressures (IPs) have to be increased. Nevertheless, consequent intraoperative increments in intrarenal pressures (IRPs) are able to deteriorate any procedure. Yet, only a few endourologists remain cognizant and are aware of normal and pathological IRP values. Furthermore, the impact of increased IRPs in perioperative complications and the ways to prevent them by controlling IRPs remain obscure.

✉ Theodoros Tokas
ttokas@yahoo.com

1 Department of Urology and Andrology, General Hospital Hall in Tirol, Milser Str. 10, 6060 Hall in Tirol, Austria

2 Department of Urology, Kantonspital Frauenfeld, Spital Thurgau AG, Frauenfeld, Switzerland

3 Second Department of Urology, Sismanoglio Hospital, Athens Medical School, Athens, Greece

Materials and methods

Evidence acquisition

A review of the literature was performed using PubMed. Original works restricted to the English language were identified. We included articles discussing IRP, IP, and IF. All experimental and observational studies were judged as eligible, but not restricted to controlled clinical trials, case series, case–control and cohort studies. Reviews, comments, and editorials were excluded. The literature search was conducted by the first author using the keywords, percutaneous nephrolithotomy OR PCNL, Ureteroscopy, OR

URS, OR RIRS, and then restricted with the keywords (AND) irrigation flow, OR irrigation pressure OR intrarenal pressure OR intrapelvic pressure OR renal pelvic pressure. This search identified 552 records (Fig. 1). After excluding duplicate references, 511 unique references were reviewed by title or abstract. A list of articles judged to be highly relevant by the junior (T.T.) and senior (U.N.) authors were distributed to the co-authors, to reach a final consensus on the articles included and the structure of the review. Eligible studies known to the authors, but not identified by the search were also evaluated for inclusion, adding an additional 17 unique records. A total of 90 unique references (experimental studies, controlled clinical trials, case series, case–control and cohort studies) were included in the qualitative synthesis. Due to study heterogeneity and the nonstandardized quality appraisal, a narrative synthesis was performed. The limitations of using a single database for review are taken into account [1]. Moreover, outcomes may be limited by study heterogeneity and selection bias. IRPs are measured in cm H_2O, mm Hg, or mbar. In this work, all pressures were converted to cm H_2O (1 cm H_2O = 0.73 mm Hg = 0.97 mbar), which is the most common IRP unit in the international literature. Due to the journal word and reference restrictions, the review was divided into two works. This first part deals with normal and pathological IRPs, as well as possible complications of increased IRP values.

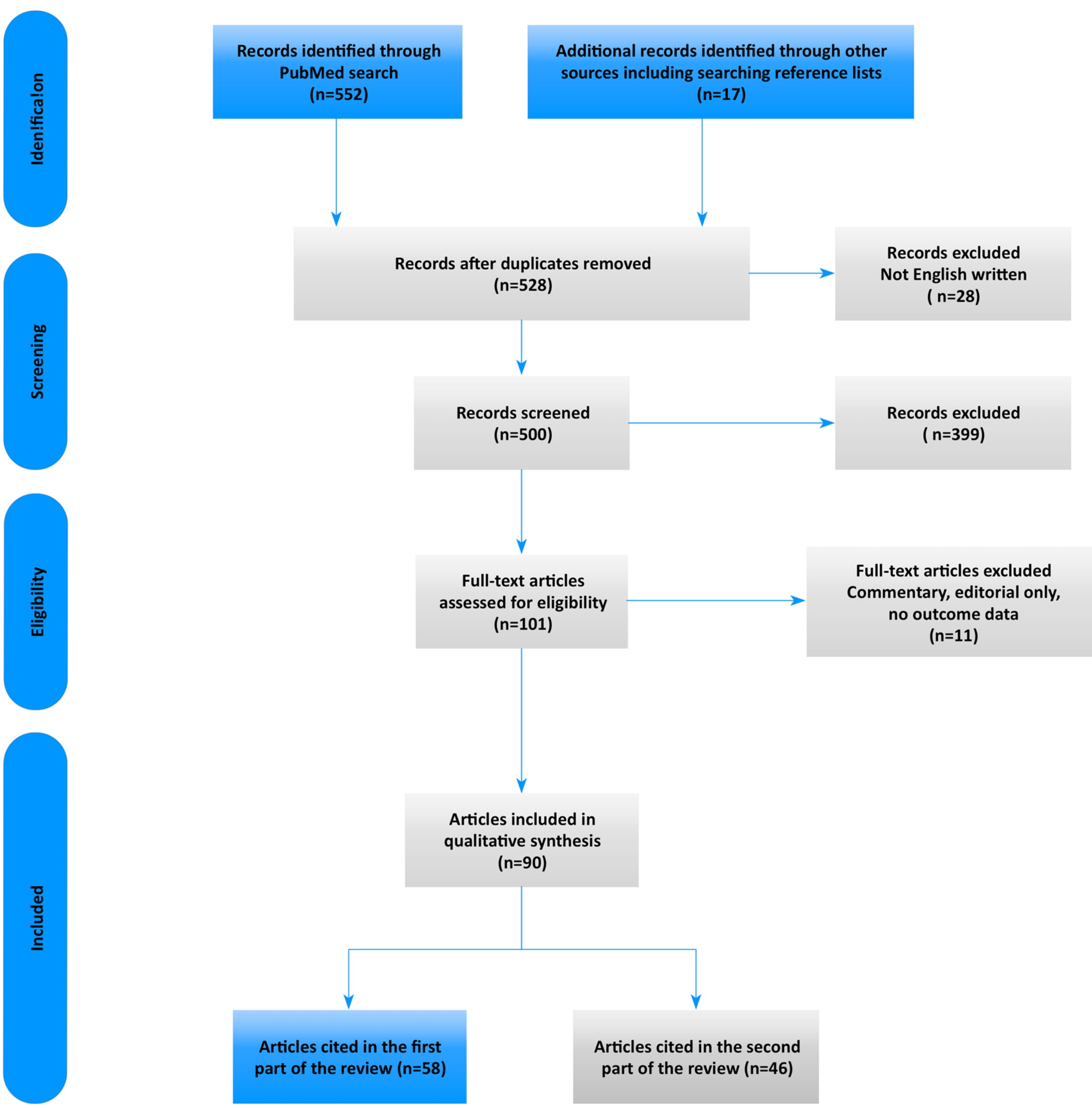

Fig. 1 Flowchart of the literature search

Outcomes

How to measure IRPs

By utilizing pressure transducers in animal and human studies, many researchers have recorded accurate intraluminal pressures in the renal pelvis and ureter [2–4]. However, it was Whitaker who laid the foundations by introducing an antegrade pressure measurement of the upper urinary tract, the Pressure Flow (PPF)—or Whitaker test [5]. The test aimed to identify obstruction as an established cause of urinary tract dilatation and was initially used in pediatric patients [5, 6]. Its basic principle is based on the antegrade perfusion of the whole upper urinary tract with saline or contrast medium, at a constant FR of 10 mL/min through a percutaneous needle or an, already existing, nephrostomy tube. In that way, the IRP, namely, the pressure needed to push the fluid through the urinary system at a fixed flow rate, is recorded. The resistance created by the tubing of the percutaneous needle is also taken into account. The site of access should always be above a suspected obstruction. The perfusion is maintained until the pressure stabilizes, and at this point, the FR down the ureter and into the bladder is also approximately 10 mL/min. In case of a complete obstruction, the pressure continues to rise with a rate depending on the FR and the degree of dilatation. The absolute or final pressure is related to the atmospheric pressure at a point outside the patients' body and at the kidney level. By subtracting the empty bladder pressure from the absolute pressure, we obtain the relative pressure, which is in fact the pressure drop across the ureteric orifice. Differential pressures ranging 12–15 cm H_2O are considered normal, whereas pressures of over than 20 cm H_2O indicate obstruction. Values ranging 15–20 cm H_2O are considered to be indeterminate (Table 1) [7].

IRP values during normal and obstructive conditions (Table 1)

The flow of a fluid through a rigid tube is influenced by the pressure gradient between the ends of the tube, the fluid viscosity, the length, and the diameter of the tube. The relationship of these variables is described by the Poiseuille equation (dP = 8VgL/pR4), where V represents the FR through the tube, g is the viscosity of the fluid, L is the length, and R the radius of the tube [8, 9]. External compression constitutes an additional factor that affects flow. However, in a collapsible tube, the transmural pressure is better expressed by the Laplace equation. Important factors are the difference between the internal and external pressures, external compression, wall tension, and wall thickness. According to the Laplace equation, the transmural pressure equals the tension per unit length over the radius. During the early filling phase, the pressure increases in a gentle curve up to a certain capacity which represents the inherent elasticity of the upper urinary tract. The rise of the curve is the indirect measure of the ureteral or pelvic wall compliance [9].

In human unobstructed kidneys, the IRPs at low urine FRs range from zero to a few cm H_2O [10]. During diuresis, IRPs may exceed 27.2 cm H_2O. In chronic kidney

Table 1 IRPs in different conditions

Condition	Values (cm H_2O)
Pressures during the PPF test	
Normal	12–15 [7]
Intermediate	15–20 [7]
Increased	>20 [7]
Human unobstructed kidney	0–2 [10]
Maximum IRP during diuresis	> 27.2 [11]
Chronic kidney obstruction	68–95.2 [11]
Minimum measured IRPs during obstruction	20 [12]
Mean basal IRP during hydronephrosis	12.1 [13]
IRPs at 50% bladder capacity (intravesical pressure: 8.9 ± 3.1 cm H_2O)	
Non-hydronephrotic kidneys	7.4 ± 1.1 [16]
Hydronephrotic kidneys	20.8 ± 2.1 [16]
Measured IRPs that can cause pyelovenous backflow	40.8–47.6 [11]
Minimum measured IRPs that can cause pyelovenous backflow	13.6–27.2 [17, 18]
Measured IRPs that can cause pyelosinous backflow/forniceal rupture	81.6–95.2 [21, 22] 272 [23]
Measured IRPs that can cause kidney injury and arterial blood flow reduction	20–40 [41]

IRPs intrarenal pressures *PPF* pressure flow test obstruction they range 68–95.2 cm H_2O, and consequently, the values decrease until the kidney atrophies [11]. However, values of 20 cm H_2O in cases of severe obstruction have also been recorded [12]. In hydronephrosis, mean basal IRPs of 12.1 cm H_2O have been presented [13] and the pressure reaches obstructive levels at flow rates > 10 mL/min [14, 15]. Of note, changes in intravesical pressure are reflected in IRP changes, as bladder pressure at 50% capacity reaches 8.9 ± 3.1 cm H_2O, and corresponding pelvic pressure is 20.8 ± 2.1 cm H_2O in hydronephrotic versus 7.4 ± 1.1 cm H_2O in non-hydronephrotic kidneys [16]. Hence, the urinary bladder should be continuously drained during endourological procedures to avoid additional increments of IRPs.

Complications of increased IRPs

Backflow and fluid absorption

Pyelorenal backflow may be defined as a condition in which the contents of the renal pelvis and calyceal system penetrate beyond their limits, either to the peripelvic sinus tissue (pyelosinous backflow), renal vein (pyelovenous backflow), collecting ducts and tubules (pyelotubular backflow), or renal interstitium (pyelointerstitial backflow). The last two conditions may be termed as intrarenal backflow. Hinman and Redewill [11] demonstrated in dogs, that pyelovenous backflow occurs at pressures 40.8–47.6 cm H_2O. Subsequent studies showed that pyelovenous backflow occurs at pressures as low as 13.6–27.2 cm H_2O [17, 18], and at 40.8–68 cm H_2O pyelovenous backflow becomes evident [19, 20]. Risk of pyelosinous backflow/forniceal rupture has been recorded at pressures of 81.6–95.2 cm H_2O in rabbits [21, 22] and at 272 cm H_2O in piglets [23]. Pre-existing factors such as low urine flow, ischemic damage, and vesicoureteral reflux lower the critical pressure for backflow [22, 24]. A serious complication of pyelovenous backflow is the excessive irrigation fluid absorption, which can be extra- or intra-vascular [25] and may result in fluid overload, electrolyte imbalance and cardiovascular instability [26].

Reported fluid absorption during PCNL ranges 50–2200 ml [17, 27, 28]. In one study, fluid absorption occurred in 78% of patients, and in 28% of the cases, absorbed volumes were in excess of 1 L [27]. Renal pelvic perforation results in passage of large volumes of irrigating fluid into the retroperitoneum [29]. The acute absorption syndrome occurs in case of a rapid irrigating fluid absorption due to direct intravascular absorption through opened veins, or after opening of the intraperitoneal space with peritoneal resorption [17, 30]. Additional intraoperative bleeding from opening veins, or lesions in the wall of the renal pelvis can be a contributing factor and at the same time a warning sign for irrigation fluid absorption. More specifically, fluid may be absorbed either directly into the opened veins or from a perinephric accumulation of irrigating fluid [31].

On the other hand, fluid absorption during RIRS usually remains low, mainly due to the smaller instrument caliber and the small irrigation channel, which result in a reduced FR. However, by increasing the flow to maintain optimal view, some form of high-pressure irrigation should be utilized resulting, indeed, to a risk of fluid extravasation in case of a wall defect [32]. In a study recruiting 23 patients, and by measuring fluid input (irrigation fluid and contrast medium) and output (irrigation fluid, contrast medium, and urine output), calculated fluid absorption reached a mean value of 54 mL (range 4–137 mL) [33]. Of note, authors used pressurized irrigation, and there may be variations in fluid absorption according to whether unpressurized irrigation, syringe irrigation, or mechanically pressurized irrigation is utilized.

Infectious complications

During endourological procedures, urinary tract infections, including sepsis, are probably directly related to IRP and backflow, highlighting the role of antibiotics, especially for stone treatment. Irrigation volume appears to be an independent risk factor for SIRS [34]. During RIRS [34], SIRS occurs in 8.1% of the cases. Along with other factors, such as patient age and stone size, irrigation flow rate and increased irrigation volume with consecutive IRP increase are independently correlated with increased SIRS rates. Fever also complicates PCNL with an overall incidence of 10.8% [35]. Septic shock after PCNL has a low reported incidence (0.3–1%), but a high mortality (66–80%) [36]. Increased IRP constitutes a major predisposing factor for postoperative fever and sepsis [37]. High IP (272 cm H_2O) has been associated with a higher risk of SIRS (46%) compared to low IP (108.8 cm H_2O/11%) [38].

Kidney damage

From animal studies, it is proven that kidneys subjected to high pressures can be irreversibly damaged [20, 39]. IPs greater than 204 cm H_2O have been demonstrated to produce significant pathological changes in the kidneys of pigs compared with IPs less than 122.4 cm H_2O. Rupture of the collecting system has been noted at 448.8 cm H_2O [39]. Forniceal rupture with pyelosinous backflow has been associated with perirenal pseudocysts, retroperitoneal edema, fibrolipomatosis, perinephritic abscess, and perirenal hemorrhage [20]. High pressures can result in calyceal urothelium denudation, submucosal edema formation, and congestion [20, 39]. In the acute phase of increased pressures, the renal tubules show marked vacuolization and degeneration, as well as histological signs of metaplasia and pericalyceal vasculitis. These findings can be present even 4–6 weeks after a procedure [20, 39].

In a pig angiography kidney study [40], delays in arterial filling at increased IRPs were documented. Additionally, perfusate flow measurements demonstrated a corresponding fall in perfusion flow. In another porcine study [41], at pressures of 20–40 cm H_2O (Table 1), kidney injuries and arterial blood flow reduction were documented. High pressures can also induce renal oxidative damage and secondary loss of renal function due to insufficient venous flow and compression of microvessels. Renal venous outflow obstruction is more detrimental to kidneys than arterial obstruction due to ischemia/reperfusion injury [42, 43]. Pyelovenous backflow limits venous outflow to a certain extent, and renal microvessels become compressed by the perfusion pressure, which decreases the blood supply to the renal parenchyma. Both of these factors lead to ischemia/reperfusion damage of the kidney [44].

Other complications

Extravasation of irrigating fluid in the presence of infection is the most likely cause of ureteric stricture formation [45]. The reno-renal reflex characterizes potential changes in baseline IRP and diuresis in the contralateral kidney during conditions of increasing IRP and perfusion with isotonic saline in the ipsilateral renal pelvis. This reflex is supposed to allow either of the kidneys to share an extra load of the other one by increasing the contractile activity of the renal pelvis, thus assumedly assisting the regulation of urine flow [46]. The mean systolic and diastolic blood pressure levels are significantly increased during PCNL compared to post-procedure levels. Additionally, a tendency to hyponatremia and metabolic acidosis develops in addition to significant increases in renin, aldosterone and ACTH levels. These changes may be due to the invasive nature of the intervention to the kidney and the continuous irrigation with consecutive rises of IRPs [47, 48]. Additionally, urine appears to be a strong causative agent of fibrotic reaction, when escaping from its normal pathway [20, 49]. Moreover, intrarenal backflow may be responsible for development of a pathological site for stone growth by causing papillary damage, forming, in that way, a vicious circle [50]. High IRPs, probably due to forniceal or parenchymal ruptures, can lead to subcapsular hematomas [51] and even life-threatening perirenal bleedings [52]. Finally, high IRPs during RIRS for urothelial cancer could cause tumor seeding [53, 54]. Nevertheless, this hypothesis has not been yet confirmed by high-power literature evidence and further investigation is deemed necessary.

Role of different factors in complication development

Despite the values reported above, many experts postulate that during endourological procedures, different complications appear with much higher IRPs. These differences could be explained by several important confounders.

Duration of elevated IRPs

In pigs, renal cellular injury becomes evident within as little as 1 h at IRPs of 20 cm H_2O or greater [41]. During RIRS, approximately 1 mL of irrigation fluid is absorbed per minute [33]. Additionally, the procedure time is independently correlated with increased postoperative fever and SIRS rates [34, 55, 56]. During PCNL, the volume of fluid absorbed also increases with increased IRPs and procedure times [17]. Maximum fluid absorption appears after a total irrigation time of 30 min, and, after 30 and 90 min the volumes of irrigant absorbed are 153.8, and 1361.9 ml [27], respectively.

Furthermore, decrease in potassium levels is reported from the 30th to 120th irrigation minute and does not recover until 24 h later after the operation. An increase in Cl levels is reported at the 120th minute of irrigation and there is a decreasing trend of pH from the start to the 120th minute of irrigation, and 24 h after operation this trend attenuates [28]. Finally, although hemodynamic and electrolyte changes during PCNL remain stable, a trend towards metabolic acidosis is obvious as the irrigation time goes by [28]. Hence, current evidence suggests that overall procedure time should be limited to 2 h [28].

Role of obstruction, irrigation pressure, irrigation volume in kidney injury

Rats with severely obstructed kidneys are more likely to suffer acute kidney injuries by IPs starting from 81.6 cm H_2O [57]. In contrast, not obstructed kidneys suffer no renal injuries and less obstructed kidneys only suffer injuries at pressures of 136 cm H_2O. In a similar rabbit study [44], severely obstructed kidneys were more susceptible to oxidative damage and mitochondrial injury than mildly obstructed kidneys when subjected to kidney perfusion pressure > 81.6 cm H_2O. IPs of 136 cm H_2O cause oxidative damage in both obstructed and non-obstructed kidneys. Current recommendations suggest the maintenance of perfusion pressure during endourological procedures, between 68 cm H_2O and 408 cm H_2O to maintain a lower IRP limit that does not exceed the 30 cm H_2O that can cause kidney injury [58, 59].

Conclusion

In human unobstructed kidneys, IRPs at low urine FRs reach only a few cm H_2O. IRPs lower than 30 cm H_2O should be maintained during endourological procedures to avoid complications. Duration of increased IRPs, obstruction, perfusion pressure, and irrigation fluid volume are independent predictors of complication development. Current evidence regarding IRPs remains heterogeneous and there is a lack of proper designed and executed human studies, making IRPs a neglected predictor of upper tract endourology complications.

Author contributions TT: Data management, data analysis, manuscript writing. TRW Herrmann: Interpreting data. AS: Interpreting data. UN: Protocol/project development and interpreting data.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Human and animal rights This review does not involve human participants and/or animals.

References

1. Falagas ME, Pitsouni EI, Malietzis GA, Pappas G (2008) Comparison of PubMed, Scopus, Web of Science, and Google Scholar: strengths and weaknesses. FASEB J 22(2):338–342. https://doi.org/10.1096/fj.07-9492LSF
2. Struthers NW (1969) The role of manometry in the investigation of pelvi-ureteral function. Br J Urol 41(2):129–162
3. Rattner WH, Fink S, Murphy JJ (1957) Pressure studies in the human ureter and renal pelvis. J Urol 78(4):359–362
4. Weaver RG, Yelderman JJ (1967) The positive pressure catheter for measurement of intracavital pressures and pressure patterns. J Urol 98(6):718–720
5. Whitaker RH (1973) Methods of assessing obstruction in dilated ureters. Br J Urol 45(1):15–22
6. Whitaker RH (1979) An evaluation of 170 diagnostic pressure flow studies of the upper urinary tract. J Urol 121(5):602–604
7. Veenboer PW, de Jong TP (2011) Antegrade pressure measurement as a diagnostic tool in modern pediatric urology. World J Urol 29(6):737–741. https://doi.org/10.1007/s00345-011-0717-9
8. Pfitzner J (1976) Poiseuille and his law. Anaesthesia 31(2):273–275
9. Bullock KN (1983) The biomechanical principles of upper urinary tract pressure-flow studies. Br J Urol 55(2):136–139
10. Kiil F (1953) Pressure recordings in the upper urinary tract. Scand J Clin Lab Invest 5(4):383–384. https://doi.org/10.3109/00365515309094217
11. Hinman F, Redewill FH (1926) Pyelovenous back flow. J Am Med Assoc 87(16):1287–1293. https://doi.org/10.1001/jama.1926.02680160035011
12. Walzak MP Jr, Paquin AJ Jr (1961) Renal pelvic pressure levels in management of nephrostomy. J Urol 85:697–702
13. Kinn AC (1981) Pressure flow studies in hydronephrosis. Scand J Urol Nephrol 15(3):249–255
14. Lupton EW, Holden D, George NJ, Barnard RJ, Rickards D (1985) Pressure changes in the dilated upper urinary tract on perfusion at varying flow rates. Br J Urol 57(6):622–624
15. Pfister RC, Newhouse JH, Hendren WH (1982) Percutaneous pyeloureteral urodynamics. Urol Clin North Am 9(1):41–49
16. Fichtner J, Boineau FG, Lewy JE, Sibley RK, Vari RC, Shortliffe LM (1994) Congenital unilateral hydronephrosis in a rat model: continuous renal pelvic and bladder pressures. J Urol 152(2 Pt 2):652–657
17. Kukreja RA, Desai MR, Sabnis RB, Patel SH (2002) Fluid absorption during percutaneous nephrolithotomy: does it matter? J Endourol 16(4):221–224. https://doi.org/10.1089/089277902753752160
18. Stenberg A, Bohman SO, Morsing P, Muller-Suur C, Olsen L, Persson AE (1988) Back-leak of pelvic urine to the bloodstream. Acta Physiol Scand 134(2):223–234. https://doi.org/10.1111/j.1748-1716.1988.tb08483.x
19. Boccafoschi C, Lugnani F (1985) Intra-renal reflux. Urol Res 13(5):253–258
20. Jung HU, Frimodt-Moller PC, Osther PJ, Mortensen J (2006) Pharmacological effect on pyeloureteric dynamics with a clinical perspective: a review of the literature. Urol Res 34(6):341–350. https://doi.org/10.1007/s00240-006-0069-x
21. Thomsen HS, Dorph S, Olsen S (1982) Pyelorenal backflow in rabbits following clamping of the renal vein and artery: radiologic and microscopic investigation. Acta Radiol Diagn 23(2):143–148
22. Thomsen HS, Dorph S, Olsen S (1981) Pyelorenal backflow in normal and ischemic rabbit kidneys. Invest Radiol 16(3):206–214
23. Thomsen HS, Larsen S, Talner LB (1982) Pyelorenal backflow during retrograde pyelography in normal and ischemic porcine kidneys. A radiologic and pathoanatomic study. Eur Urol 8(5):291–297
24. Hodson CJ (1969) The effects of disturbance of flow on the kidney. J Infect Dis 120(1):54–60
25. Hahn RG (1991) Monitoring of TURP with ethanol. Lancet (London, England) 338(8782–8783):1602
26. Schultz RE, Hanno PM, Wein AJ, Levin RM, Pollack HM, Van Arsdalen KN (1983) Percutaneous ultrasonic lithotripsy: choice of irrigant. The Journal of urology 130(5):858–860
27. Malhotra SK, Khaitan A, Goswami AK, Gill KD, Dutta A (2001) Monitoring of irrigation fluid absorption during percutaneous nephrolithotripsy: the use of 1% ethanol as a marker. Anaesthesia 56(11):1103–1106
28. Xu S, Shi H, Zhu J, Wang Y, Cao Y, Li K, Wang Y, Sun Z, Xia S (2014) A prospective comparative study of hemodynamic, electrolyte, and metabolic changes during percutaneous nephrolithotomy and minimally invasive percutaneous nephrolithotomy. World J Urol 32(5):1275–1280. https://doi.org/10.1007/s00345-013-1204-2
29. Carson CC, Nesbitt JA (1985) Peritoneal extravasation during percutaneous lithotripsy. J Urol 134(4):725–727
30. Sinclair JF, Hutchison A, Baraza R, Telfer AB (1985) Absorption of 1.5% glycine after percutaneous ultrasonic lithotripsy for renal stone disease. Br Med J (Clinical research ed) 291(6497):691–692
31. Dimberg M, Norlen H, Hoglund N, Allgen LG (1993) Absorption of irrigating fluid during percutaneous transrenal lithotripsy. Scand J Urol Nephrol 27(4):463–467
32. Rao PN (1987) Fluid absorption during urological endoscopy. Br J Urol 60(2):93–99
33. Cybulski P, Honey RJ, Pace K (2004) Fluid absorption during ureterorenoscopy. J Endourol 18(8):739–742. https://doi.org/10.1089/end.2004.18.739
34. Zhong W, Leto G, Wang L, Zeng G (2015) Systemic inflammatory response syndrome after flexible ureteroscopic lithotripsy: a study

of risk factors. J Endourol 29(1):25–28. https://doi.org/10.1089/end.2014.0409
35. Seitz C, Desai M, Hacker A, Hakenberg OW, Liatsikos E, Nagele U, Tolley D (2012) Incidence, prevention, and management of complications following percutaneous nephrolitholapaxy. Eur Urol 61(1):146–158. https://doi.org/10.1016/j.eururo.2011.09.016
36. Michel MS, Trojan L, Rassweiler JJ (2007) Complications in percutaneous nephrolithotomy. Eur Urol 51(4):899–906. https://doi.org/10.1016/j.eururo.2006.10.020 **(discussion 906)**
37. Kreydin EI, Eisner BH (2013) Risk factors for sepsis after percutaneous renal stone surgery. Nat Rev Urol 10(10):598–605. https://doi.org/10.1038/nrurol.2013.183
38. Omar M, Noble M, Sivalingam S, El Mahdy A, Gamal A, Farag M, Monga M (2016) Systemic inflammatory response syndrome after percutaneous nephrolithotomy: a randomized single-blind clinical trial evaluating the impact of irrigation pressure. J Urol 196(1):109–114. https://doi.org/10.1016/j.juro.2016.01.104
39. Schwalb DM, Eshghi M, Davidian M, Franco I (1993) Morphological and physiological changes in the urinary tract associated with ureteral dilation and ureteropyeloscopy: an experimental study. J Urol 149(6):1576–1585
40. Thomsen HS, Dorph S, Larsen S, Talner LB (1983) Intrarenal backflow and renal perfusion during increased intrapelvic pressure in excised porcine kidneys. Acta Radiol Diagn 24(4):327–336
41. Fung LC, Atala A (1998) Constant elevation in renal pelvic pressure induces an increase in urinary *N*-acetyl-beta-D-glucosaminidase in a nonobstructive porcine model. J Urol 159(1):212–216
42. Li X, Liu M, Bedja D, Thoburn C, Gabrielson K, Racusen L, Rabb H (2012) Acute renal venous obstruction is more detrimental to the kidney than arterial occlusion: implication for murine models of acute kidney injury. Am J Physiol Renal Physiol 302(5):F519–F525. https://doi.org/10.1152/ajprenal.00011.2011
43. Park Y, Hirose R, Dang K, Xu F, Behrends M, Tan V, Roberts JP, Niemann CU (2008) Increased severity of renal ischemia-reperfusion injury with venous clamping compared to arterial clamping in a rat model. Surgery 143(2):243–251. https://doi.org/10.1016/j.surg.2007.07.041
44. Cao Z, Yu W, Li W, Cheng F, Rao T, Yao X, Zhang X, Larre S (2015) Oxidative damage and mitochondrial injuries are induced by various irrigation pressures in rabbit models of mild and severe hydronephrosis. PLoS One 10(6):e0127143. https://doi.org/10.1371/journal.pone.0127143
45. Boddy SA, Nimmon CC, Jones S, Ramsay JW, Britton KE, Levison DA, Whitfield HN (1989) Irrigation and acute ureteric dilatation—as for ureteroscopy. Br J Urol 63(1):11–13
46. Shafik A (1998) Response of the renal pelvis and ureter to distension of the contralateral renal pelvis and ureter: identification of the reno-renal pelvic reflex. World J Urol 16(5):359–364
47. Atici S, Zeren S, Aribogan A (2001) Hormonal and hemodynamic changes during percutaneous nephrolithotomy. Int Urol Nephrol 32(3):311–314
48. Mohta M, Bhagchandani T, Tyagi A, Pendse M, Sethi AK (2008) Haemodynamic, electrolyte and metabolic changes during percutaneous nephrolithotomy. Int Urol Nephrol 40(2):477–482. https://doi.org/10.1007/s11255-006-9093-6
49. Hodson CJ (1981) Neuhauser lecture. Reflux nephropathy: a personal historical review. AJR Am J Roentgenol 137(3):451–462. https://doi.org/10.2214/ajr.137.3.451
50. Thomsen HS (1984) Pyelorenal backflow. Clinical and experimental investigations. Radiologic, nuclear, medical and pathoanatomic studies. Dan Med Bull 31(6):438–457
51. Meng H, Chen S, Chen G, Tan F, Wang C, Shen B (2013) Renal subcapsular hemorrhage complicating ureterolithotripsy: an unknown complication of a known day-to-day procedure. Urol Int 91(3):335–339. https://doi.org/10.1159/000350891
52. Xu L, Li G (2013) Life-threatening subcapsular renal hematoma after flexible ureteroscopic laser lithotripsy: treatment with superselective renal arterial embolization. Urolithiasis 41(5):449–451. https://doi.org/10.1007/s00240-013-0585-4
53. Cutress ML, Stewart GD, Wells-Cole S, Phipps S, Thomas BG, Tolley DA (2012) Long-term endoscopic management of upper tract urothelial carcinoma: 20-year single-centre experience. BJU Int 110(11):1608–1617. https://doi.org/10.1111/j.1464-410X.2012.11169.x
54. Grasso M, Fishman AI, Cohen J, Alexander B (2012) Ureteroscopic and extirpative treatment of upper urinary tract urothelial carcinoma: a 15-year comprehensive review of 160 consecutive patients. BJU Int 110(11):1618–1626. https://doi.org/10.1111/j.1464-410X.2012.11066.x
55. Zhong W, Zeng G, Wu K, Li X, Chen W, Yang H (2008) Does a smaller tract in percutaneous nephrolithotomy CONTRibute to high renal pelvic pressure and postoperative fever? J Endourol 22(9):2147–2151. https://doi.org/10.1089/end.2008.0001
56. Dogan HS, Sahin A, Cetinkaya Y, Akdogan B, Ozden E, Kendi S (2002) Antibiotic prophylaxis in percutaneous nephrolithotomy: prospective study in 81 patients. J Endourol 16(9):649–653. https://doi.org/10.1089/089277902761402989
57. Cao Z, Yu W, Li W, Cheng F, Xia Y, Rao T, Yao X, Zhang X, Larre S (2013) Acute kidney injuries induced by various irrigation pressures in rat models of mild and severe hydronephrosis. Urology 82 (6):1453.e1459–1416. https://doi.org/10.1016/j.urology.2013.08.024
58. Landman J, Venkatesh R, Ragab M, Rehman J, Lee DI, Morrissey KG, Monga M, Sundaram CP (2002) Comparison of intrarenal pressure and irrigant flow during percutaneous nephroscopy with an indwelling ureteral catheter, ureteral occlusion balloon, and ureteral access sheath. Urology 60(4):584–587
59. Shao Y, Shen ZJ, Zhu YY, Sun XW, Lu J, Xia SJ (2012) Fluid-electrolyte and renal pelvic pressure changes during ureteroscopic lithotripsy. Min Invas Ther Allied Technol MITAT 21(4):302–306. https://doi.org/10.3109/13645706.2011.595419