Shock Wave Injury to the Kidney in SWL: Review and Perspective

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Abstract. Shock wave lithotripsy (SWL) is a first-line option for treatment for urinary calculi—particularly effective for the removal of uncomplicated stones from the upper urinary tract. The success of lithotripsy is tempered, however, by the occurrence of acute injury that has been reported to progress to long-term complications. SW trauma to the kidney is a vascular lesion characterized by parenchymal and subcapsular bleeding. The acute lesion is dose-dependent, and typically localized to the focal volume of the lithotripter. Cavitation has been implicated in vessel rupture, but SW-shear has the potential to be a primary mechanism for damage as well. Possible chronic adverse effects of SWL may include new-onset hypertension, development of diabetes, and exacerbation of stone disease. If acute trauma could be reduced, it seems likely that serious long-term effects could be minimized, or even eliminated. Reducing the dose of SW’s needed for stone breakage is one option. Improved coupling improves stone breakage, and slowing SW rate significantly improves stone-free outcomes. Experiments with animals now show that treatment protocols can be designed to protect against tissue injury. Initiating treatment with low energy SW’s dramatically reduces lesion size, and reducing the rate of SW delivery virtually eliminates SW trauma altogether. SWL stands to gain from new advances in technology, as lithotripters become safer and more effective. Perhaps the greatest progress will be made when we have determined the physical mechanisms of SW action both for stone breakage and tissue damage, and have better characterized the biological response to SW’s—as this will provide the principles needed to achieve the best combination of safety and efficiency with whatever lithotripter is at hand.

Keywords: shock wave lithotripsy, kidney, trauma, kidney stones, animal models

PACS: *43.25.Cb, *43.40.Jc, 87.53.Tf, 87.19.Rr; 87.54.Hk

INTRODUCTION

It is well documented that renal injury occurs as a side effect of SWL, and considerable progress has been made in characterizing the acute effects of SW trauma, particularly in experimental animals. Still, we are just beginning to appreciate the causes of SW injury, the acoustic mechanisms involved, and the physiologic risk factors that appear to potentiate adverse effects. It is also clear that we have a fairly limited understanding of the long-term consequences of SW treatment. With this in mind, it seems reasonable to take stock of the field, redefine the problem of adverse...
effects, and assess what can be done to help make shock wave lithotripsy safer and more effective.

THE CHARACTERISTICS OF ACUTE SWL INJURY TO THE KIDNEY

Shock Waves Rupture Blood Vessels

Animal studies leave no doubt that SW’s can cause hemorrhage to the kidney vasculature [1-5]. The pig is the accepted model of renal injury in SWL [4-6]. Detailed morphological analysis of pig kidneys treated with a clinical dose of SW’s has shown that veins are particularly susceptible to SW injury, and a broad range of vessels—from vasa recta and cortical capillaries, to arcuate and intralobular vessels—are damaged by SW’s [2,3,7,8]. The vast majority of research in SWL injury has been conducted using the Dornier HM3 lithotripter, a water bath-style, spark-gap machine, but all lithotripters that have been studied create a vascular lesion [9].

Vascular Trauma in SWL Can Cause Parenchymal Hemorrhage and Subcapsular Hematomas

The occurrence of hematomas is a potentially significant finding. Certainly, mild subcapsular hematomas can be uncomplicating, but big bleeds can seriously compromise renal function, and can lead to acute renal failure [10-21]. Hematoma rates may depend on the type of lithotripter, and values between about 1 and ~12% can be found in the literature [reviewed in 7]. Even higher rates have been reported using CT or MR imaging [22,23]. There are also data suggesting that age is a risk factor for the development of perinephric hematomas in SWL, with the incidence of hematomas increasing about 2-fold per decade [13].

The Hemorrhagic Lesion in SWL Is Dose-dependent, and Proper Detection and Quantitation of Injury Requires a Rigorous Methodology

Lesion size, the volume of hemorrhagic tissue in the kidney, increases with SW number and with the power setting of the lithotripter [24-27]. It is often possible to see such trends by gross visual inspection alone, but quantitation of the histologic lesion gives a much better means to localize sites of hemorrhage, and determine the extent of damage. A method that has proven to be both sensitive and objective is to perform in situ perfusion fixation, followed by instillation of the vasculature with pigmented microfil to mark the patent vasculature [28]. The kidney is then embedded whole in paraffin, and serially sectioned at 40 μm. The cut face of the kidney is photographed—systematically selected representative sections—and areas of parenchymal hemorrhage are outlined and measured (morphometric segmentation) to determine lesion size. By this method the entire kidney is serially sectioned, areas of hemorrhage exclusively
within the parenchyma are segmented to exclude the renal pelvis and calyceal system, and the lesion is expressed as percent of functional renal volume (%FRV) [7].

Research on Renal Injury in SWL Requires an Intact, Physiologically Competent, Viable Animal Model

Patient studies have shown that SW’s trigger a rapid reduction in renal blood flow in both the treated and the contralateral kidney, and data suggest that the effect can be long-lasting [29,30]. The overall importance of this hemodynamic response has yet to be fully worked out, but vasoconstriction may be a critical event in the protective effect of initiating SWL treatment with low energy SW’s, as has been observed in pigs [31]. Observation of the responsiveness of the kidney to SW’s is also a reminder of the importance of using the appropriate animal model to assess SWL trauma. Some laboratories have reported using cadaver and slaughterhouse kidneys to assess for SW injury [32]. Unfortunately, no matter how freshly excised the kidney may be, it will not show a normal physiologic response. SW injury to the kidney is dominated by vascular trauma. If the vasculature of the model is not anatomically intact and physiologically competent, the damage that occurs upon treatment with SW’s cannot be expected to represent what occurs in SWL, and such a model has limited-to-no practical value.

EVIDENCE FOR LONG-TERM ADVERSE EFFECTS IN SWL, AND WHY CHRONIC INJURY IS A CONCERN

Acute Vascular Trauma to the Kidney in SWL Leads To Scarring and Loss of Functional Renal Mass

The acute vascular lesion caused by SW’s can progress to scar formation. Thus, parenchymal hemorrhage can result in a chronic loss of nephrons—a loss of functional renal mass. Chronic damage of this sort was first reported in a study with experimental animals, in which dogs that were treated using the Dornier HM3 lithotripter showed fibrosis after one month, with the severity of scarring dependent on the dose of SW’s applied [1]. A study in rabbits likewise showed a dose-dependent increase in scar formation one month after treatment, with a significant rise (nearly 10 fold higher) in scar volume with treatment at 2000 SW’s compared to 1000 SW’s [33]. The renal medulla may be particularly susceptible to SW damage, and a study in pigs has shown that treatment with a clinical dose of SW’s can lead to complete atrophy of renal papillae within the target site, observed at 3 months post-SWL [16]. Scar formation has also been reported in patients, in which Single Photon Emission CT was used to measure exclusion of Technicium-99 label from areas of poor vascular perfusion [34]. Patients were scanned before and 30 days following SWL, with a loss of marker uptake observed in half the subjects. The scars that developed in these patients measured larger (mean 19x15 mm) than the focal zone of the lithotripter (EDAP-PEL) that was used. These reports all suggest that lasting damage can occur as a
consequence of SWL but, clearly, more work is needed to characterize the long-term consequences of treatment, and to determine the relationship between acute injury and lasting damage.

New-onset Hypertension Has Been Reported, with Increased Risk for Older Patients

Hypertension is a potential consequence of SWL and there is evidence to suggest that the occurrence of new-onset hypertension in lithotripsy patients is related to the dose of SW’s they received [9]. Clearly this topic has stimulated spirited debate, but the implications posed by reports showing a link between SWL and hypertension cannot help but be cause for concern [2,7,9,35-42]. A prospective study by Janetscheck and colleagues shows an alarming increase in intrarenal resistive index in patients 60 years of age and older [36]. This finding implies that SW treatment for stone disease can have serious long-term effects, and that age may be a critical risk factor for significant adverse effects in SWL [41]. It is not clear precisely what mechanisms might be at play in the cellular response to SW’s that could lead to hypertension. However, the observation that SW’s can stimulate glomerular mesangial cell proliferation in pigs, still evident one month after treatment, suggests a condition that if unresolved could be a potential causative factor in the development of hypertension [43].

A Potential Link Has Been Proposed Between SWL and the Development of Diabetes

In a patient follow-up study, Krambeck and colleagues report an association between SW dose for treatment of upper urinary tract stones, and the development of diabetes mellitus [35, also see Dr. Krambeck’s paper in this volume]. Here, again, the potential cellular mechanisms at play leading to such a response are not known, but one would expect this involves direct SW damage to the pancreas. Patient studies have reported SW trauma to the pancreas, and detected increased serum and urinary amylase levels following SWL [44-47]. Clearly the pancreas lies along the acoustic axis during treatment of either kidney, but it is also true, although not widely appreciated, that SW’s targeted at urinary stones have the potential to cause significant trauma outside the SW-axis. A recent study in which gas-laden microspheres were injected into the circulation of pigs during SW administration has shown that SW pressures exceeding the threshold for vessel-damaging cavitation can occur a considerable distance off the geometric SW axis of the lithotripter [48]. That is, with abundant cavitation nuclei present in the vasculature, SW’s targeted at the lower pole of the left kidney caused extensive hemorrhage throughout the abdominal viscera, even the contralateral kidney. Thus, it should be recognized that SWL treatment of stones in the kidney and upper ureter carries the potential to deliver a damaging dose of SW’s to the pancreas.
Multiple Lithotripsy May Exacerbate Stone Disease

Epidemiological studies indicate that over the past three decades the percentage of calcium phosphate (CaP) in stones has been on the rise and, indeed, the data suggest a transition from calcium oxalate (CaOx) stones to CaP stones [49]. In looking for factors that might explain this, Parks and colleagues observed a striking positive correlation between the percent CaP in stones and the number of lithotripsy sessions per patient [50]. The number of SWL sessions was higher for CaP stone formers than for CaOx stone formers, and highest for patients whose stones contained brushite, even when corrected for number of stones and duration of stone disease. Brushite stones typically present a more complicated pathology than CaOx stones. Unlike CaOx stones, which appear to develop in association with papillary interstitial mineral, brushite stones develop from crystalline deposits that plug the papillary collecting ducts—causing further cell damage that can lead to advanced tissue alterations, such as tubular atrophy and papillary fibrosis [51]. The correlation between SWL and the occurrence of brushite stones is concerning, as this implies that for some patients, multiple sessions of SWL may lead to the development of a more advanced pathology involving stones (brushite) that generally do not respond well to subsequent SW treatment.

WHAT PHYSICAL MECHANISMS CAUSE SHOCK WAVE INJURY?

Cavitation Is Strongly Implicated in SW Trauma, But May Not Be the Only Cause of Injury

Cavitation within blood vessels is believed to play an important role in the vascular trauma that characterizes tissue injury in SWL [9,52]. This is shown by studies in which cavitation is enhanced by injecting micro-bubbles or gas-laden micro-carrier beads into the circulation during SWL, resulting in increased hemorrhage [48,53]. Also, strategies to suppress cavitation, such as using tandem SW’s or a phase-reversed waveform to interrupt bubble growth, dramatically reduce tissue damage [54,55].

The search to determine just how bubbles go about rupturing vessels has proven to be an interesting problem. Decades of research in physics and engineering on the disruptive force of inertial cavitation, has shown that when a cavitation bubble collapses it can generate a fluid micro-jet that delivers great force to a very small area [56]. This is the mechanism by which cavitation bubbles chip away at metal surfaces, such as the erosion that often occurs to ship’s propeller blades. If a cavitation bubble were to grow and collapse within a blood vessel, the force of its micro-jet would surely break the vessel wall. At question, however, is whether a cavitation bubble within a vessel can grow large enough to undergo such forceful collapse. Inertial cavitation typically involves bubbles that grow to a size of one to several millimeters in diameter before collapse. This is too large to occur within the micro-vessels of the kidney that are damaged during SWL. Zhong and colleagues tested the idea that vessel...
rupture could be caused by bubble expansion [52,57]. They set up an in vitro system in which a laser fiber was used to excite bubble expansion within the lumen of a segment of micro-dialysis tubing, and were able to photograph the rupture of this vessel phantom.

Whether it is bubble expansion, or bubble micro-jets, or possibly the secondary SW’s generated when bubbles collapse symmetrically that is responsible for vessel rupture, it is important to note that cavitation does not occur readily in circulating blood [53]. Also, studies using sensitive methods of cavitation detection have shown that it takes hundreds of SW’s to generate bubble activity in tissue within the living kidney [58]. This suggests that the occurrence of cavitation may be highly dependent on the intravascular micro-environment, and possibly the presence of minute particles that may serve as nuclei for cavitation bubble formation. It is not known what constitutes a natural cavitation nucleus in the circulatory system, but the fact that cavitation does not initiate readily suggests that the circulation is relatively free of such particles [53]. Thus, cavitation appears to be an important player in SW damage, but one wonders if other factors may be involved as well.

**Shear Has the Potential to Damage Tissue, Particularly at Fast SW Rate**

Cavitation may not be the driving force for SW injury. That is, bubble expansion (or collapse) may not be the primary mechanism responsible for tissue damage. In vitro studies have shown that when isolated cells are placed under static pressure in excess of the threshold for cavitation, SW’s induce cell lysis significantly higher than in untreated controls [59]. That is, in the absence of cavitation, SW’s still caused cell injury. Pigs treated with SW’s from an HM3 lithotripter fitted with a reflector insert that suppressed cavitation without significantly affecting pulse amplitude, exhibited dramatically reduced vessel damage compared to animals treated with the standard reflector, but still showed some bleeding involving vessels of the renal papillae [55]. That is, the region of the kidney that is the most sensitive to SW’s showed injury in the absence of cavitation. A study involving numerical modeling of tissue compartments in the kidney, presented in these proceedings [See Dr. Freund’s paper in this volume, pp. 356-359], suggests that stress can accumulate within the kidney parenchyma if the rate of SW delivery is faster than the displacement relaxation time of the tissue. The Freund model predicts that the magnitude of shear deformation of tissue will be different for different regions of the renal papilla, and that the region closest to the tip of the papilla, where the kidney is most sensitive to SW’s, will exhibit the greatest strain. The study suggests that vessel failure could be induced by shear, and that subsequent hemorrhage and pooling of blood could then create an environment conducive to cavitation, and further SW damage.
INJURY IS DOSE-DEPENDENT, AND THERE APPEARS TO BE A THRESHOLD FOR SIGNIFICANT SW TRAUMA

SW Damage Jumps Dramatically after ~1000 SW’s

SW injury to the kidney is dose-dependent [2,7,24-26]. Damage increases as more SW’s are delivered, and injury is greater at higher SW energy levels, but it is clear that whereas renal injury is cumulative, it is not linear. Studies in the pig in which the volume of hemorrhagic tissue was quantified, showed that a dose of 2000 SW’s at 24kV from the HM3 lithotripter produced a lesion measuring ~6% of functional renal volume [31]. Treatment with half that dose (1000 SW’s) at the same energy setting gave a lesion of only 0.2%FRV [7]. That is, the injury produced by 1000 SW’s was barely detectable, and doubling the dose increased the lesion about 30 fold. These data in the pig strongly suggest that there is a threshold for the initiation of injury that, if exceeded, can result in significant trauma. This level of acute injury is in agreement with data for evidence of long-term damage in rabbits, where the size of parenchymal scars measured one month following treatment increased from ~1.4% at 1000 SW’s to over 12% at 2000 SW’s [33].

An important challenge is that we do not know where the threshold for significant SW injury truly lies. Data for the HM3 localize the threshold only vaguely, and begin to define it for just one lithotripter, and for only one energy setting, at that. One would expect that the threshold for measurable injury would depend not only on the number of SW’s, and the power or kV of the pulses, but also on the acoustic signature of the particular lithotripter—the dimensions and energy density of the focal volume, and possibly even the rate of SW delivery. That is, since acoustic output is different for different lithotripters, the threshold for injury should be different, and should depend on treatment parameters. Further, just as kidney size and the health of experimental animals have been shown to affect the renal response to SW’s, the health status of the subject should also affect the threshold for injury [4,13,36,42,60].

Thresholds for SW Injury Could Be Determined in an Appropriate Animal Model

Clearly it would be valuable to know the number of SW’s that can be delivered safely at typical treatment settings, and this is a reasonable problem to address with a relevant animal model such as the pig. At the present time, the most clear-cut metric of SWL injury is lesion size, and the method to determine lesion volume is well defined [28]. Such data collected for popular lithotripters could give a reasonable approximation of the damage expected with a given dose. That is, it may be possible to determine the maximum dose that is safe for a particular lithotripter, and this information could be a useful guideline.
Cavitation Detection Could Be a Means to Monitor SW Damage in Real Time

As so many factors have the potential to affect the injury threshold for an individual patient, it would also be valuable to have a means to detect injury in the making. This is not out-of-the-question and, indeed, cavitation detection has been proposed as a means to do this [61]. The approach is to use acoustic monitoring such as focused directional transducers to listen for bubble noise, or B-mode ultrasound to visualize cavitation echogenicity during treatment. In concept, a robust cavitation signal originating within the kidney parenchyma would indicate that conditions are right for vessel damage, signaling the need to stop treatment. This makes good sense, but a problem encountered in early attempts was difficulty in localizing cavitation to the renal parenchyma. A recent study shows that using US simultaneously with passive cavitation detection gives precise localization, and suggests that with proper characterization of the signal, cavitation can be detected in the kidney parenchyma using diagnostic ultrasound alone [58]. Thus, it now seems feasible that ultrasound monitoring during SWL could be used to detect the occurrence of cavitation in tissue, and this information could help decision making during treatment.

TREATMENT STRATEGIES TO MAKE SWL SAFER AND MORE EFFECTIVE

New lithotripter technologies may eventually be developed to make SWL safer and more effective, but what can be done now to improve how lithotripsy is performed using the lithotripters we currently have at hand? The answer may be fairly simple—slow down, and treat at lower energy.

Stone Clearance Is Improved at 60 SW/min Compared to 120 SW/min

Several clinical studies have now reported that slowing the rate of SW delivery to 60 SW/min breaks stones more effectively, giving better outcomes than treatment at the typical rate of 120 SW/min [62-67]. This positive effect of slowing SW rate is seen with both electrohydraulic and electromagnetic lithotripters. The obvious advantage of improving the efficiency of SW action is that fewer SW’s should be needed for treatment [62]. A potential disadvantage is that slower rate can increase overall treatment time. How much an increase in treatment time may depend on how the treatment end point is determined (i.e. the quality of imaging used to assess breakage).

The idea that simply slowing SW rate can break stones better may be difficult at first to appreciate, but recent studies give clues to the physical mechanisms involved. Cavitation bubble collapse at the stone surface contributes to stone comminution, but bubble growth along the acoustic axis can interfere with delivery of shock wave energy to the stone. Cavitation is enhanced at fast SW rate because as bubbles collapse they spawn micron sized micro-bubbles that can persist between pulses. Such micro-
bubbles act as cavitation nuclei, seeding new cavitation initiated by subsequent SW's [68]. The shorter the interval between SW's, the more cavitation nuclei survive, and the greater the number of new cavitation bubbles that form. Bubble interference at fast rate does not appear to be a matter of blockage or deflection of the pulse, but instead involves transfer of energy from the tensile phase of the SW into bubble growth. That is, cavitation increases as SW's are delivered at faster rate, but the leading positive pressure phase of the SW is virtually unaffected, and it is the trailing negative wave that is reduced [69]. It is as yet not entirely clear how the reduction of negative pressure in the SW impacts stone breakage, but it has been predicted that this should reduce the amplitude of shear waves within the stone, as well as affect the dynamics of bubble cluster collapse at the stone surface [70,71].

**Step-wise SWL Protects against SW Injury**

One of the most exciting findings in lithotripsy research is the observation that treatment with a priming dose of low amplitude SW’s can have a dramatic tissue-protective effect. That is, initiating treatment at a low power setting before shifting to higher amplitude pulses results in a significant reduction in lesion size [31]. This observation comes from work using the pig model. The findings are significant because they suggest a potential treatment strategy to reduce adverse effects. How the observation came to light is also instructive, as the experimental design behind the study validates the importance of using an appropriate animal model for this sort of work. In studies to characterize SWL injury to the kidney using a clinical dose of SW’s from the Dornier HM3, one group of animals was treated with 2000 SW’s at 24 kV (120 SW/min) targeted to the lower renal pole. This dose created a lesion measuring ~6% functional renal volume (FRV). A pilot group of pigs received the same dose of SW’s to both renal poles. That is, a dose of 2000 SW’s at 24 kV was delivered first to the lower pole, then 2000 SW’s were targeted to the upper pole of the same kidney. The lesion in the lower pole was as expected, but there was virtually no damage to the upper pole. Treatment of the lower pole had protected the upper pole from injury. Subsequent experiments determined that as few as 100 SW’s delivered to one pole at 12 kV (28.8±10.8 MPa) prior to treatment of the same site with 2000 SW’s at 24 kV (41.4±5.3 MPa), reduced lesion size to only 0.3% FRV. Thus, “pre-treatment” with low energy SW’s protected the kidney from subsequent injury. The physiologic mechanism responsible for this protective effect has yet to be fully characterized, but assessment of renal hemodynamics shows that SW’s induce transient vasoconstriction in the treated kidney (and in the contralateral kidney as well). Increased vascular tone may make vessels less susceptible to damage due to cavitation or shear stress. The responsiveness of the living, intact kidney is, thus, an important feature of this animal model. The main result observed using the “protection protocol” in experimental animals—significantly reduced tissue damage—suggests that a similar, step-wise treatment protocol might reduce kidney injury in clinical SWL.
Treatment at Reduced SW Rate Minimizes SW Trauma while Improving Stone Breakage

Results from a study in pigs suggest that slowing the rate of SW delivery to 30 SW/min reduces lesion size to less than 0.1% FRV [72,73]. That is, slowing SW rate affords protection against kidney injury equivalent to that observed using the stepwise, “protection protocol”. Since stone breakage is also improved at slow SW rate, this finding opens the possibility that reducing the rate of SW delivery could be a means to improve both the safety and efficacy of SWL.

Improved Coupling Reduces the Number of SW’s Needed for Stone Comminution

Most modern lithotripters employ a dry treatment head that is coupled to the patient with a medium of high acoustic transmission such as gel or oil. Development of the dry head was an important advance in the evolution of the lithotripter, making it possible for lithotripters to be transportable, and substantially improving access to SWL. Unfortunately, it is difficult to achieve good coupling with a dry-head device. Typical protocols used to prepare a patient for treatment create air pockets at the coupling interface, and these defects can be a significant barrier to SW transmission. In vitro studies have shown that coverage by air pockets of just 2% of the coupling interface reduced stone breakage by 20-40% [74]. The quality of coupling is also highly variable, and it is conceivable that variability in coupling may contribute to variability in clinical outcomes.

Poor coupling may be a factor in adverse effects. When coupling is poor more SW’s are needed to break the stone. Though the pressure amplitude of the pulses is reduced, they still generate negative pressure that is well above the threshold for cavitation, thus they still have the capacity to injure blood vessels. Because poor coupling is less efficient, it takes more SW’s to break the stone, and delivery of more SW’s has the potential to cause more injury. A further difficulty concerning coupling is that the interface between the treatment head and the skin is not readily visible. One does not know when defects are present. However, recent in vitro tests [see Pishchalnikov et al. in these proceedings, pp. 368-371] show that it is possible to improve on the quality of coupling—that how the gel is applied, matters. Taken together, these observations suggest that coupling, an aspect of lithotripsy that is generally taken for granted, is problematic, but that with attention to detail, this aspect of treatment can be improved.

CONCLUSIONS

Basic research has done a great deal to characterize the acute effects of SW’s, and there has been considerable progress in understanding the physical mechanisms of SW action in stone breakage and tissue damage. Although we know that the vascular lesion in SWL can progress to scarring and loss of functional renal mass, we know
very little about the long-term consequences of acute SW trauma. Clinical findings such as the suggestion of new-onset hypertension in older patients, the apparent link between multiple lithotripsies and a transition to brushite stone disease, and the possibility that some patients may even develop diabetes as a result of treatment for their stones, seem reason enough to invest more effort in determining the causes and consequences of long-term adverse effects in SWL, and encourage us to find practical strategies to make lithotripsy truly safe.

ACKNOWLEDGMENTS

This work was funded by NIH grant DK-43881

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