Comparison of Systemic and Renal Hemodynamics Measured by Doppler Ultrasonography in Canine Experimental Hypovolemia

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ABSTRACT. The aim of this study was to examine renal hemodynamics at the hypovolemic and recovery phases in two different hypovolemic shock models using Doppler ultrasonography, and to compare this with systemic hemodynamics. In experiment 1, the hypovolemic phase was induced in 6 mongrel dogs by removing arterial blood at 30 ml/kg for 60 min. In the recovery phase, this blood was reinfused at 30 ml/kg over 60 min. In experiment 2, hypovolemia was induced in 12 beagle dogs by rapid blood removal until blood pressure decreased to 40 mmHg and was maintained at this pressure for 30 min. Six of the dogs were then infused with 20 ml/kg hydroxyethyl starch over 5 min, and the other 6 were infused with 60 ml/kg lactated Ringer's solution also over 5 min. Parameters for systemic and renal hemodynamics were measured by using a polygraph and the Doppler method, respectively. The decrease of diastolic blood flow, resulted in an increase of vessel resistance, and was detected in the hypovolemic kidney by the Doppler method. The rapid and large volume infusion of resuscitation fluids was effective for the recovery of both systemic circulation and renal blood flow, however this induced an increase of kidney vessel resistance, a result of the autoregulation mechanism of the kidney. The changes in these parameters at the main renal artery and interlobar artery were similar. — KEY WORDS: canine, Doppler ultrasonography, hypovolemic state, renal blood flow, resuscitation.

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The primary causes of acute renal failure can be categorized as ischemic or nephrotoxic [4]. Prerenal azotemia is relatively common, particularly in dogs that have undergone surgical stress caused by anesthesia and operation [4, 6]. Prerenal azotemia may eventually compromise renal perfusion and thereby precipitate ischemic tubular damage and necrosis. The pathophysiologic mechanism leading to irreversible cell damage in the progression of prerenal azotemia to acute ischemic renal failure have been described in recent years, and the importance of the prevention of renal hypoperfusion has been emphasized [4]. It is therefore instructive to understand renal hemodynamics, in order to clarify the mechanism of the pathophysiology in renal failure.

Current methods used to estimate renal function include clearance and radioisotope tests as well as electromagnetic flowmetry and angiography. However, these methods are very complex and/or invasive for clinical applications [1, 2, 8, 20]. On the other hand, Doppler ultrasonography can be used to estimate renal hemodynamics in the non-invasive evaluation of renal grafts [12, 23, 29, 37] and some renal diseases [7, 22, 24, 25, 29, 36, 40]. Miyamoto *et al.* reported on a quantitative measurement of the renal blood flow by Doppler ultrasonography in the normal dog [19]. Doppler ultrasonography therefore, offers the possibility to study the pathophysiology of ischemic renal disease in real-time.

In this experiment, we studied renal hemodynamics using Doppler ultrasonography at the hypovolemic phase, and at the recovery phase from hypovolemia, in two different hypovolemic shock models and compared the results with the systemic hemodynamics.

MATERIALS AND METHODS

Experimental animals: Six adult mongrel dogs (4 males and 2 females; 2–9 years old), weighing 9–13 kg were used in experiment 1. Twelve adult beagles (7 males and 5 females; 2–7 years old), weighing 7–12 kg were used in experiment 2. These dogs were evaluated as clinically normal.

Anesthesia: After induction of the general anesthesia with atropine sulfate (0.03 mg/kg, i.m.), flunitrazepam (0.03 mg/kg, i.v.) and thiamylal sodium (approximately 15 mg/kg, i.v.), the dogs were endotracheally intubated, and anesthesia was maintained with isoflurane. The breathing movements were arrested using vecronium bromide (0.05 mg/kg, i.v., every 30 min), and were changed to mechanical ventilation with a ventilator (KV-2N, Kimura Ikakiki Co., Tokyo, Japan) at an inspired oxygen concentration (FIO_2) of 1.0 and a frequency of 16 breaths/min to achieve an arterial carbon dioxide tension ($PaCO_2$) of 35–45 mmHg. Heparin (500 U/kg, i.v.) was given every 30 min.

Hemorrhagic shock model:

Experiment 1: Hypovolemia was induced in 6 mongrel dogs during a 60 min period by removing arterial blood at 30 ml/kg/hr into a CPD blood transfusion bag (SC-207, TERUMO Co., Tokyo, Japan). The dogs were then observed over a 60-min period in the hypovolemic state. After that, blood was reinfused from the sterile bags at 30 ml/kg/hr for 60 min. Following reinfusion these dogs were again observed for 60 min.

Experiment 2: Hypovolemia was induced in 12 beagles by removing blood until the mean arterial pressure (MAP)

decreased to 40 mmHg and this pressure was then maintained for a 30-min period by adjusting the height of the CPD bag. At the end of this period, 6 of the dogs were then infused with 20 ml/kg hydroxyethyl starch solution (Hespander® Inj. Kyorin Co., Tokyo, Japan) over 5 min (HES Group), and the other 6 were infused with 60 ml/kg lactated Ringer's solution also over 5 min (LR group). Following infusion, the dogs were observed for 60 min.

Hemodynamic response: Heart rate (HR) and MAP were monitored continuously throughout the experiment using a polygraph (System 360, Nippondenki Sannei, Tokyo, Japan). For the measurement of MAP and cardiac output (CO), the ordinary catheterization technique was employed. CO was measured by the thermodilution method using a cardiac output computer (COM-2, Baxter, Santa Ana CA, U.S.A.).

Renal hemodynamic response: Renal hemodynamic response was examined in the main renal artery and in the interlobar artery using the Doppler method described by Miyamoto *et al.* [19]. The ultrasonographic equipment used was HITACHI EUB-565A (Hitachi Med. Co., Tokyo) with either a 7.5 MHz electronic linear probe (EUP-L33S, Hitachi Med. Co.) or 7.5 MHz electronic sector probe (EUP-S33, Hitachi Med. Co.). The measurements were done at the end of the expiratory phase. The diameter of the renal artery was measured in systole by B-mode echo plane and the measurement by pulsed Doppler was done at the same point. The interlobar artery was visualized using color Doppler and the measurement done by pulsed Doppler. The measurements of pulsed Doppler were recorded by video tape recorder (VTR). The calculation of the pulsed Doppler method was done from VTR images, and the angle between the Doppler beam and the vessel was corrected. The sample point size was 1 mm in length. The measurements included systolic maximal velocity (Vmax), diastolic minimal velocity (Vmin), pulsatility index (PI), resistance index (RI) and renal blood flow (RBF) in the main renal artery as well as PI and RI in the interlobar artery [5, 9, 17, 18, 35].

Measuring schedule:

Experiment 1: HR, MAP, CO and Doppler ultrasonography were measured immediately before bleeding and 30, 60, 90, 120, 150, 180, 210 and 240 min after bleeding.

Experiment 2: HR, MAP, CO were measured immediately before bleeding and 30, 35, 65 and 95 min after the MAP was stabilized at 40 mmHg. The Doppler measurement was done immediately before bleeding and 15, 35, 65 and 95 min after the MAP had stabilized at 40 mmHg.

Statistical analysis: Student's paired *t*-test was used to compare the baseline and post-treatment data in each group. Comparison among 2 groups was analyzed by Student's *t*-test according to variance equivalence in experiment 2. For non-normally distributed data, Welch's *t*-test was used. A difference was considered statistically significant when the *p*-value was below 0.05.

RESULTS

Experiment 1

Heart rate, MAP, CO and RBF (Fig. 1): HR started to increase at 60 min after bleeding and showed a significantly higher peak level at 120 min, then returned to the pre-bleeding level after the reinfusion of blood. MAP decreased significantly and quickly, reaching a minimum level of 55 mmHg at the end of bleeding, and then rose to a level higher than the pre-bleeding levels at 60 min after the reinfusion of blood. CO showed a significantly low trough at the end of bleeding and then increased, peaking at the end of the infusion of blood. After that it declined, but remained higher than the pre-bleeding level. The RBF volume showed a transient increase at 30 min after the start of bleeding. It decreased significantly to a minimum value of 74.5% at 60 min, and then increased to a maximum value of 117% at 150 min, returning to the baseline level at 240 min.

Systolic maximal velocity and Vmin (Fig. 2): The value of Vmax showed a minimum at 60 min, and showed two peaks at 30 min in the bleeding stage and at 150 min in the recovery stage. The value of Vmin decreased to its minimum at 60 min, and then recovered mildly. These changes were not significant.

Pulsatility index and RI (Fig. 3): The values of PI and RI showed two peaks at 60 and 180 min. The values of PI and RI in the main renal artery tended to be higher than in the interlobar artery. These changes were not significant.

Experiment 2

Heart rate, MAP, CO and RBF (Fig. 4): HR in both

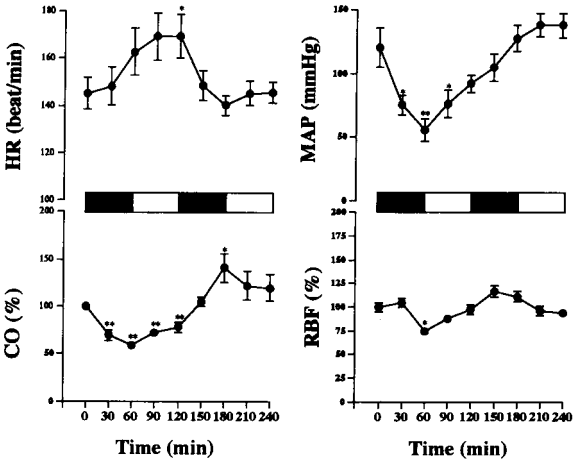

Fig. 1. Changes of the heart rate (HR), mean arterial pressure (MAP), cardiac output (CO) and renal blood flow (RBF) in experiment 1, in which a blood volume of 30 ml/kg/hr was removed. ■ Hypovolemic phase, □ Maintenance phase, ■ Recovery phase, *: $p<0.05$, **: $p<0.01$ compared with the prebleeding value.

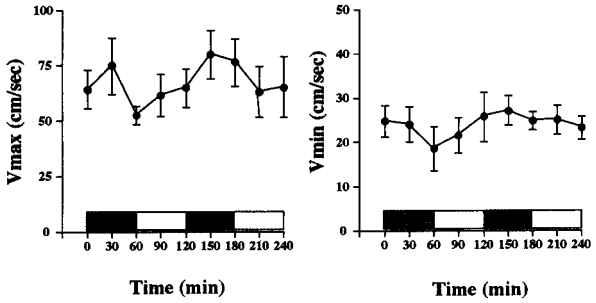

Fig. 2. Changes of the systolic maximal velocity (Vmax) and diastolic minimal velocity (Vmin) in experiment 1. For key see Fig. 1.

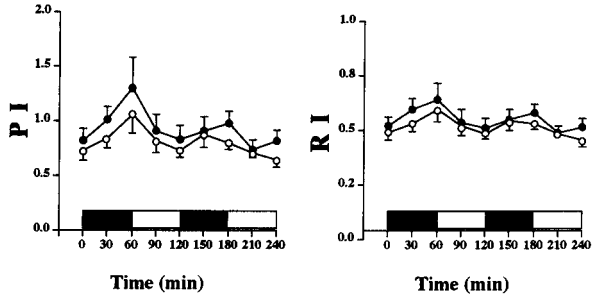

Fig. 3. Changes of the pulsatility index (PI) and resistance index (RI) of main renal artery and interlobar artery in experiment 1. ●: The value of the main renal artery; ○: The value of the interlobar artery. For key see Fig. 1.

groups showed maximum levels at the end of the hypovolemic phase. It decreased to the pre-bleeding level at the end of infusion, and then recovered mildly. The changes in MAP in both groups were similar. MAP decreased rapidly until 40 mmHg and was maintained at this pressure for 30 min. It had increased to near the level of the pre-bleeding value at the end of infusion, and remained almost at that level until the end of the experiment. CO in both groups showed a statistically significant minimum value (HES group, 36%; LR group, 50%) at the end of the hypovolemic phase, and a maximum values (Hes group, 150%; LR group, 211%) at the end of the infusion. It then decreased to the pre-bleeding value. The LR group had higher levels than the HES group at the end of the reinfusion. The RBF volume in both groups decreased in the hypovolemic phase (Hes group, 70%; LR group, 56%), then increased to a maximum value (Hes group, 213%; LR group, 153%) at the end of the infusion. The RBF volume of the HES group showed higher values than in the LR group.

Systolic maximal velocity and Vmin (Fig. 5): Vmax in both groups decreased in the bleeding stage, but had increased significantly at the end of the infusion. It then decreased mildly to a level which was higher than the pre-bleeding value. Vmin in both groups decreased significantly in the bleeding phase, but had increased significantly at the end of infusion. In the recovery phase in both groups, a level higher than the pre-bleeding level was maintained.

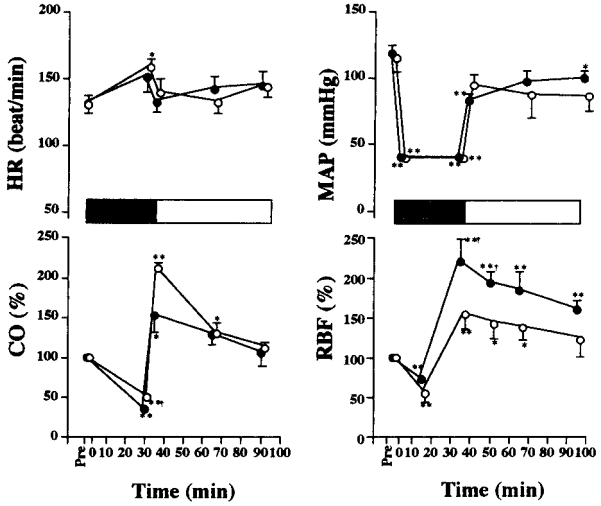

Fig. 4. Changes of HR, MAP, CO and RBF in experiment 2, in which blood was removed until the arterial pressure reached 40 mmHg and was maintained at that pressure over 30 min. ■ Hypovolemic phase, ■ Recovery phase, □ Maintenance phase, ●: The value of the HES group. ○: The value of the LR group. *: $p<0.05$, **: $p<0.01$ compared with the prebleeding value; †: $p<0.05$ compared with two groups, HES group and LR group.

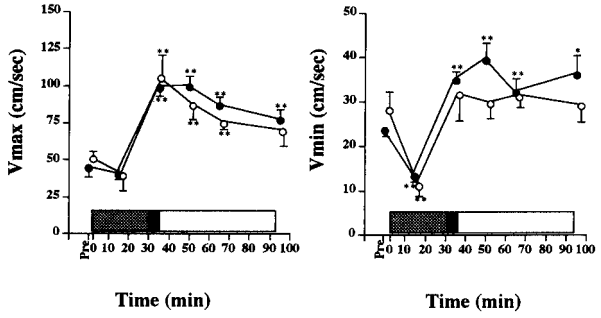

Fig. 5. Changes of Vmax and Vmin in experiment 2. For key see Fig. 4.

Pulsatility index and RI (Fig. 6): The changes of PI and RI in both groups and between the main renal artery and the interlobar artery were similar. The PI and RI increased significantly in the bleeding stage, but recovered mildly to a level which was higher than the pre-bleeding value. The changes of PI and RI in the main renal artery tended to be greater than in the interlobar artery.

DISCUSSION

Normal renal blood flow accounts for about 20% of total CO. Renal diseases such as acute renal failure might induce critical renal hypoperfusion. It is therefore, important to evaluate the renal hemodynamic as early as possible to avoid further damage to the kidney [4, 13, 14]. Doppler ultrasonography offers the possibility of non-invasive and

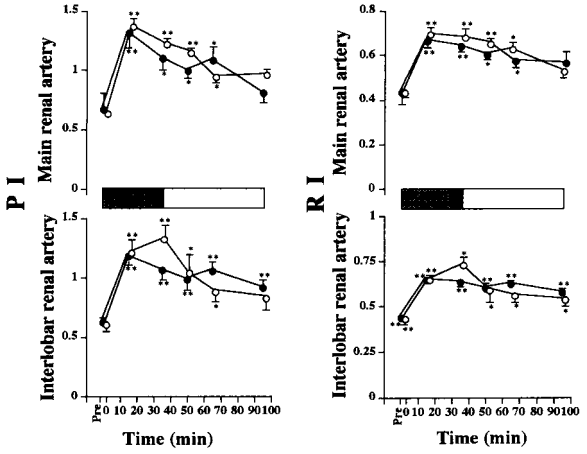

Fig. 6. Changes of PI and RI of main renal artery and interlobar artery in experiment 2. For key see Fig. 4.

real time evaluation of renal hemodynamics.

In this study, we used two different models of hypovolemia. One was a fixed level of hypotension for a preset period of time (Wiggers' model) [39], and the other utilized a single withdrawal of a certain percentage of total blood [32, 33, 38].

In humans, the examination of renal blood flow by ultrasonography done in the main renal, segmental, interlobar and interlobular arteries has been reported [18, 29, 36, 40]. The peripheral evaluation in the interlobar and the interlobular arteries reflect kidney function better [36]. In this experiment, renal blood flow in the main renal and interlobar arteries was evaluated. The approach to the main renal artery was easier in dogs, and the measurement error induced by the beam angle was minimal. We usually adopted the measuring point in the peripheral interlobar artery rather than in the segmental artery, because the intralobular artery was difficult to detect. Totsuka *et al.* reported that Vmax, Vmin and Vmin/Vmax were suitable for renal functional indices [36]. In our experiment, the measurement in the interlobar artery was chosen as PI and RI without angle correction. Since the angle between the Doppler beam and blood flow was about 60 degree, the measurement error induced by the beam angle was large. The changes of PI and RI in the main renal artery were similar to those in the interlobar artery, however the values of the main renal artery were higher than those of the interlobar artery. The main renal artery reflects the total renal vascular resistance, and in ischemic disease it is difficult to detect the interlobar blood flow in the peripheral artery. Therefore, the measurement in the main renal artery might be more appropriate than peripheral measurement.

In the model of experiment 1 in which a certain blood volume (30 m*l*/kg/60 min) was withdrawn, renal blood flow increased transiently during the bleeding period, whereas MAP and CO decreased according to the degree of bleeding. Schoenberg *et al.* reported that renal vasculature did not respond immediately to hemorrhage with vasoconstriction [32, 33]. A gradual renal vasoconstriction started slowly in response to hypotension [32, 33]. The recovery of MAP, CO and RBF was caused in this experiment by the reinfusion of a certain volume of blood (30 m*l*/kg/60 min). In this recovery condition, the autoregulation mechanism for peripheral blood pressure in the kidney may have acted earlier, because MAP increased to approximately 90 mmHg immediately before the recovery of blood circulation [11, 27, 35]. The increase of the Vmax was recognized as the recovery of the renal blood flow. There was almost no change in Vmin. It is known that the resistance of two kinds of arteriole, afferent glomerular arteriole and efferent glomerular arteriole, is responsible for the maintenance of the glomerular filtration pressure [3, 11, 15, 27, 34]. The increase of MAP was suspected to have been caused by the recovery of the vessel resistance, and the regaining of the vessel resistance was caused by the limitation of the diastolic blood flow.

In the model of experiment 2, in which low blood pressure was caused by rapid bleeding, there was a remarkable decrease in Vmin. It was thought that the renal vessels induced vasoconstriction in the rapid hypovolemic state, and the increase of vascular resistance caused by vasoconstriction resulted in a decrease of Vmin. During rapid bleeding, there was a transient disappearance of the diastolic blood flow. Only systolic blood flow was detected by the Doppler method. Norris and Barnes reported that an increase of vascular resistance, caused by the injection of microspheres into the renal artery, induced a decrease of diastolic blood flow [21]. In the study of acute rejection response to renal grafting, it was recognized that the decrease of the diastolic blood flow resulted from the increase of peripheral vascular resistance caused by interstitial edema [16, 23, 31]. Therefore, it was suspected that the response of the kidney to hypovolemia resulted in the increase of vascular resistance which caused a decrease in the diastolic blood flow. The recovery of MAP, CO and RBF was realized in experiment 2, by the infusion of a large volume of fluid (HES; 20 m*l*/kg/5 min, LR; 60 m*l*/kg/5 min). From the renal hemodynamics, it was suspected that the high level of the vessel resistance was caused by the rapid infusion while the peripheral vessels were still constricted after the hypovolemic state.

In the comparison of experiments 1 and 2, it was observed that the changes of renal hemodynamics were comparatively mild and maintained in experiment 1 in which a certain blood volume was withdrawn, although the changes of renal hemodynamics in experiment 2 were large.

In experiment 2, hydroxyethyl starch (20 m*l*/kg/5 min) and the LR solutions (60 m*l*/kg/5 min) were infused. The initial dose recommended for peracute resuscitation with HES in the dog is 20 m*l*/kg as a bolus infusion. Two to 4 times that amount is necessary when isotonic crystalloids are infused to achieve the same effect [10, 30]. Although there was a difference in the dosage and kind of the fluid, rapid resuscitation of the circulation was achieved leading to a successful to recovery of MAP, CO and RBF. The

RBF volume of the HES group showed higher values than in the LR group, although the CO volume of the LR group showed higher values than in the HES group. Prough *et al*. reported that the infusion of HES was superior to the infusion of non-colloid fluid for the recovery of renal blood flow [26]. The CO volume was measured in the pulmonary artery. The LR group with a large volume infusion showed higher values than the HES group. However, it was indicated that the infusion of HES was more effective on the recovery of RBF than in the large volume infusion of LR solutions.

In conclusion, the decrease of diastolic blood flow in the kidney in a hypovolemic state was recognized and detected by Doppler ultrasonography. The decrease of diastolic blood flow resulted in an increase of renal vessel resistance. The changes of renal vessel resistance between the main renal artery and interlobar artery were similar. The examination of blood flow in both the vessels might be beneficial in the detection of hypovolemia in the kidney. On the improvement of the circulation, the rapid and large volume injection of resuscitation fluids was efficient at recovering renal blood flow, although this induced an increase in kidney vessel resistance, a result of the autoregulation mechanism of the kidney.

REFERENCES

1. Abildgaard, U. 1989. Hemodynamic and functional changes during renal venous stasis in dog kidneys. *Dan. Med. Bull.* 36: 212–222.
2. Aukland, K. 1980. Methods for measuring renal blood flow: Total flow and regional distribution. *Ann. Rev. Physiol.* 42: 543–555.
3. Auklad, K. and Jien, H. 1987. Renal autoregulation: models combining tubuloglomerular feedback and myogenic response. *Am. Physiol. Soc.* 252: F768–F783.
4. Binns, S. H. 1994. Pathogenesis and pathophysiology of ischemic injury in case of acute renal failure. *Compend. Contin. Educ. Pract. Vet.* 16: 31–43.
5. Cazenave, C. R., Sievers, K. W., Kaude, J. V., Williams, J. L., Bush, D., and Wright, P. G. 1989. Pulsatile flow index for quantitative measurement of blood flow with duplex ultrasound. An experimental study. *Eur. J. Radiol.* 9: 42–43.
6. DiBartola, S. P. 1980. Pathophysiology and management. *Compend. Contin. Educ. Pract. Vet.* 11: 952–958.
7. Dodd, G. D., Kaufman, P. N., and Bracken, R. B. 1991. Renal arterial duplex Doppler ultrasound in dogs with urinary obstraction. *J. Urol.* 145: 644–646.
8. Finco, D. R., Tabaru, H., Brown, S. A., and Barsanti, J. A. 1993. Endogenous creatinine clearance measurement of glomerular filtration rate in dogs. *Am. J. Vet. Res.* 54: 1575–1578.
9. Fung, L. C. T., Steckler, R. E., Khoury. A. E., McLorie, G. A., Chait, P. G., and Churchill, B. M. 1994. Intrarenal resistive index correlates with renal pelvis pressure. *J. Urol.* 152: 607–611.
10. Griffel, M. and Kaufman, B. S. 1992. Pharmacology of colloids and crystalloids. *Crit. Care Clin.* 8: 235–253.
11. Guyton, A. C., Manning, R. D., Hall, J. E., Norman, R. A., Young, D. B., and Pan, Y. 1984. The pathogenic role of the kidney. *J. Cardiovasc. Pharmacol.* 6: S151–S161.
12. Kageyama, T., Muto, M., Endo, T., Watanabe, T., Mishina, M., Wakao, Y., Suzuki, T., and Takahashi, M. 1995. Evaluation of transplanted kidney by color Doppler imaging in dogs—renal blood flow and jet phenomenon of urine from the ureteral orifice. *Jpn. J. Vet. Anesth. Surg.* 26: 29–37 (in Japanese with English summary).
13. Lane, I. F., Grauer, G. F., and Fettman, M. J. 1994. Acute renal failure. Part I. Prevention, and strategies for protection. *Compend. Contin. Educ. Pract. Vet.* 16: 15–29.
14. Lane, I. F., Grauer, G. F., and Fettman, M. J. 1994. Acute renal failure. Part II. Diagnosis, management, and prognosis. *Compend. Contin. Educ. Pract. Vet.* 16: 625–645.
15. Lerman, L. O., Bentley, M. D., Fiksen-Olsen, M. J., Strick, D. M., Ritman, E. L., and Romero, J. C. 1995. Pressure dependency of canine intrarenal blood flow within the range of autoregulation. *Am. Physiol. Soc.* 268: F404–F409.
16. London, N. J., Aldoori, M. I., Lodge, V. G., Bates, J. A., Irving, H. C., and Giles, G. R. 1993. Reproducibility of Doppler ultrasound measurement of resistance index in renal allografts. *Br. J. Radiol.* 66: 510–513.
17. Mastorakou, I., Robbins, M. E. C., and Bywaters, T. 1993. Resistance and pulsatility Doppler indices: how accurately do they reflect changes in renal vascular resistance. *Br. J. Radiol.* 66: 577–580.
18. Mastorakou, I., Lindsell, D. R. M., Piepoli, M., Adamopoulos, S., and Ledingham, J. G. G. 1994. Pulsatility and resistance indices in intrarenal arteries of normal adults. *Abdom. Imaging* 19: 369–373.
19. Miyamoto, T., Hagio, M., Mwanza, T., Kobayashi, T., Okumura, M., and Fujinaga, T. 1995. Quantitative measurement of canine renal arterial blood flow using Doppler ultrasonography. *J. Vet. Med. Sci.* 57: 785–788.
20. Moore, C. D. and Gewertz, B. L. 1982. Measurement of renal blood flow. *J. Surg. Res.* 32: 85–95.
21. Norris, C. S. and Barnes, R. W. 1984. Renal artery flow velocity analysis: a sensitive measure of experimental and clinical renovascular resistance. *J. Surg. Res.* 36: 230–236.
22. Patriquin, H. B., Oregan, S., Robitaille, P., and Paltiel, H. 1989. Hemolytic-uremic syndrome: intrarenal arterial Doppler patterns as a useful guide to therapy. *Radiology* 172: 625–628.
23. Platt, J. F., Ellis, J. H., and Rubin, J. M. 1991. Renal transplant pyelocaliectasis: role of duplex doppler US in evaluation. *Radiology* 179: 425–428.
24. Platt, J. F., Rubin, J. M., and Ellis, J. H. 1991. Acute renal failure: possible role of duplex Doppler US in distinction between acute prerenal failure and acute tubular necrosis. *Radiology* 179: 419–423.
25. Platt, J. F., Rubin, J. M., and Ellis, J. H. 1994. Diabetic nephropathy: evaluation with renal duplex Doppler US. *Radiology* 190: 343–346.
26. Prough, D. S., Whitley, J. M., Taylor, C. L., Deal, D. D., and DeWitt, D. S. 1991. Small-volume resuscitation from hemorrhagic shock in dogs: effect on systemic hemodynamics and systemic blood flow.
27. Regan, M. C., Young, L. S., and Geraghty, J. 1995. Regional renal blood flow in normal and desease states. *Urol. Res.* 23: 1–10.
29. Rigsby, C. M. 1988. Renal duplex sonography. pp. 201–245. *In*: Clinical Application of Doppler Ultrasound (Taylor, K. J. W., Rigsby, C. M., Burns, P. N., and Wells,P. N. T. eds.), Raven Press, New York, U.S.A.
30. Rudloff, E. and Kirby, R. 1994. Hypovolemic shock and re-

suscitation. *In*: Emergency Medicine (Kirby, R. and Crowe, D. T. eds.), *Vet. Clin. North Am. Small Anim. Pract.* 24: 1015–1039.
31. Saarinen, O., Ahonen, S. J., and Edgren, J. 1994. Reversed diastolic blood flow at duplex Doppler. *Acta Radiol.* 35: 10–14.
32. Schoenberg, M. H., Lundberg, C., Gerdin, B., Smedegard, G., Messmer, K., and Arfors, K. E. 1985. Hemorhagic shock in the dog. I. Correlation between survival and severity of shock. *Res. Ex. Med.* 185: 21–33.
33. Schoenberg, M. H., Lundberg, C., Gerdin, B., Smedegard, G., Messmer, K., and Arfors, K. E. 1985. Hemorhagic shock in the dog. II. Studies on central hemodynamic and regional blood flow. *Res. Ex. Med.* 185: 469–482.
34. Stein, J. H. 1990. Regulation of the renal circulation. *Kidney Int.* 38: 571–576.
35. Terry, J. D., Granger, S. H., Chen, B. C., Rysavy, J. A., Lefkowiz, D. M., Oldemeyer, B., Pettinger, W. A., and Lee, H. C. 1993. Adjusted resistive index: a method to estimate rapidly renal blood flow: preliminary validation in hypertensives. *J. Ultrasound Med.* 12: 751–756.
36. Totsuka, D., Boku, S., Sugisaki, T., Kubota, K., Kawauchi, A., and Kamiya, K. 1990. 2-D Doppler echographic measurement of renal segmental and interlobar arteries in renal lesions: correlation between the renal blood velocity and the creatinine clearance. *Jpn. J. Med. Ultrasonics.* 17: 672–679 (in Japanese with English summary).
37. Une, S., Taura, Y., Yasuda, M., Nakama, S., and Ejima, H. 1994. Monitoring of blood flow using Doppler ultrasound in canine renal allografts. *J. Jpn. Vet. Med. Assoc.* 47: 268–269 (in Japanese with English summary).
38. Vivaldi, E., Macinelli, S., and Gunther, B. 1983. Experimental hemorrhagic shock in dogs: standadization. *Res. Exp. Med.* 182: 127–137.
39. Wiggers, C. J. 1950. Physiology of Shock. Commonwelth Fund, New York.
40. Yura, T., Yuasa, S., Sumikawa, T., Takahashi, N., Aono,M., Kunimune, Y., Fujioka, H., Miki, S., Takamitsu, H., and Matsuo, H. 1993. Doppler sonograhic measurement of phasic renal artery blood flow velocity in patients with chronic glomerulonephritis. *J. Ultrasound Med.* 4: 215–219.