Pressure Diuresis – 9 Sample Student "Essays"

Below please find assembled consecutively in one document the brief analyses submitted by nine students in Mammalian Physiology '08 to the Teach Yourself Pressure Diuresis exercise.

ALR October 22, 2008

Dr. Meyers

Pressure Diuresis and Pressure Natriuresis

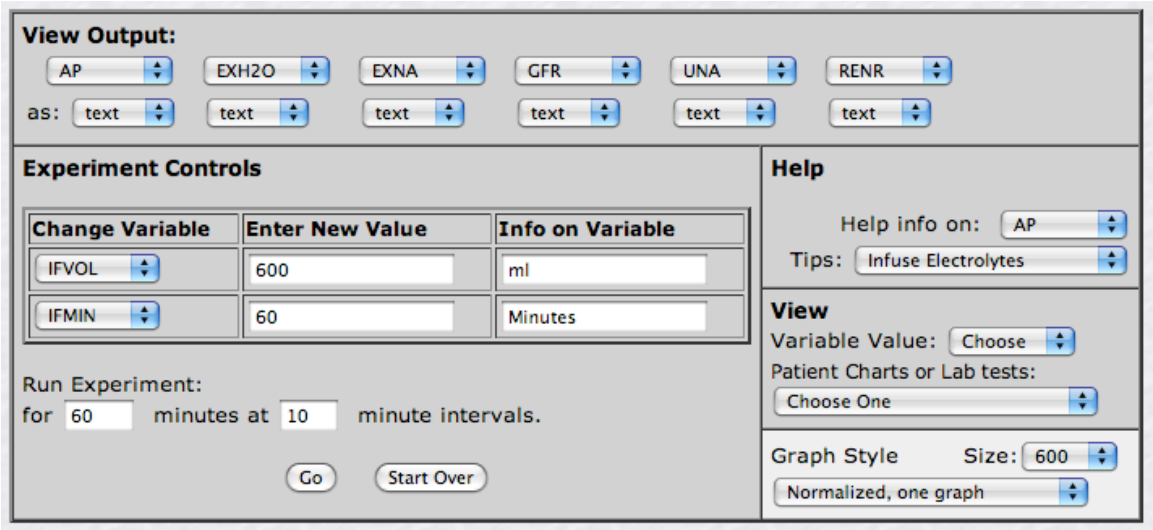
Pressure- diuresis was induced in web-human by infusing electrolytes to 600 mL over a 60-minute period. Pressure- Diuresis is occurring in this patient because over the 10 minute observation period because the value of urinary exrcretion of water (EXH2O) increased from 0.995 to 1.276; there was a general increase over time. Pressure- diuresis occurs because of a slight build up in arterial pressure, which occurred by inducing electrolytes. The mean arterial pressure increased from 99.69 mmHg to 102.2 mmHg. The diuresis occurred because of the increased mean arterial pressure because there was also an increase in GFR. The slight increase in GFR (from 123 to 139.7) can be accounted for by the increase in arterial pressure.

Similarly, pressure- natriuresis is occurring in this patient because over the 10 minute observation period, the urinary concentration of sodium increases from 116 to 148.5. Therefore, the amount of sodium being excreted from the body increases dramatically, indicating a natriuresis. The natriuresis is pressure indicated by the increase in arterial pressure and the increase in GFR explained above.

The three mechanisms that autoregulate the affects of the increase in pressure are also showed. The first is the increase from 123 to 139.7 in the GFR. The second mechanism that shows that pressure diuresis is occurring is the decrease in angiotensin II formation based on the renal vascular resistance. Angiotensin II increases the renal vascular resistance (RENR), so when the renal vascular resistance is decreased, it demonstrates that there is less angiotensin II in the blood. The third mechanism that occurs is a decrease in the percentage of the filtered load of sodium and water that is

reabsorbed. The values that indicate a decrease sodium and water reabsorption are the increases in sodium and water excretion. By the end of the observation period the reabsorption percentage went from 99.35% reabsorption of sodium to 0.8%, indicating that there was a huge decrease in sodium reabsorption.

DAY/HR	AP	EXH2O	EXNA	GFR	UNA	RENR
1-12:10 AM	99.69	0.9954	0.1155	123.0	116.0	0.8439E-01
1-12:20 AM	100.1	1.048	0.1269	126.8	121.0	0.8276E-01
1-12:30 AM	100.6	1.106	0.1417	130.1	128.2	0.8170E-01
1-12:40 AM	101.0	1.148	0.1534	132.6	133.6	0.8088E-01
1-12:50 AM	101.5	1.192	0.1656	135.1	138.9	0.8010E-01
1- 1:00 AM	101.8	1.232	0.1771	137.4	143.7	0.7937E-01
1- 1:10 AM	102.2	1.276	0.1895	139.7	148.5	0.7868E-01



CT Pressure Diuresis

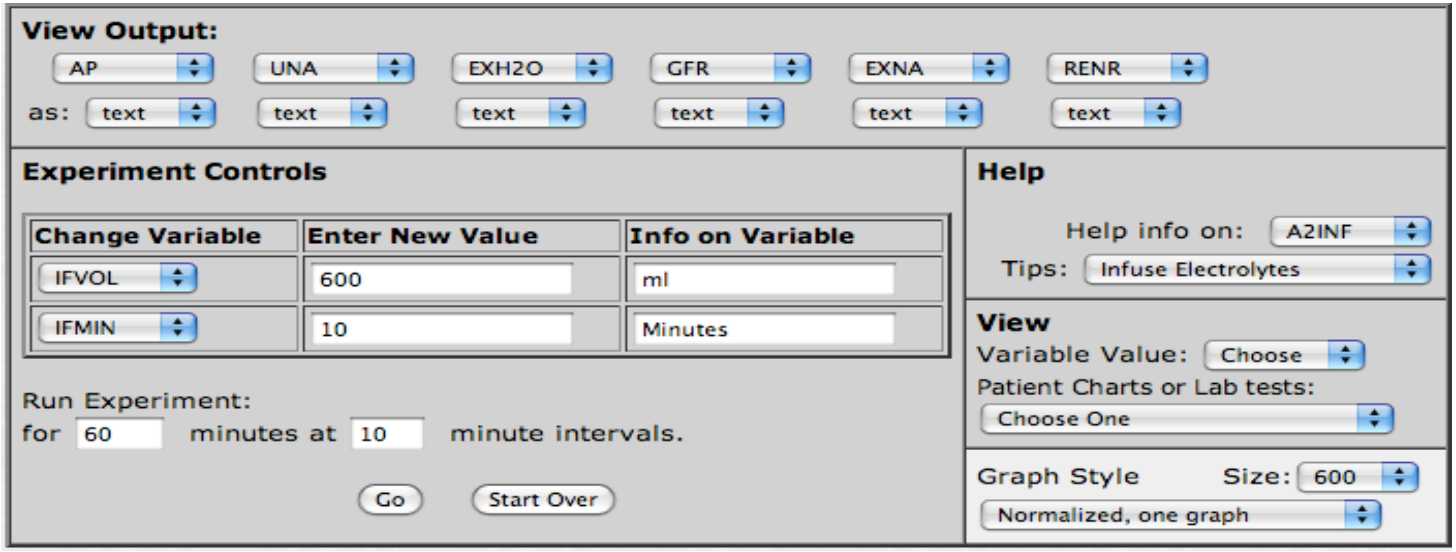
The infusion of electrolytes will increase the inter-arterial pressure as the ions are absorbed into the blood. This can be seen as the reason for the increase in AP from 99.64 up to 102.3 mmHg (see Fig 1). Accompanying the increase in pressure will be a slight increase in GFR, as autoregulation will not allow for this increase to be very large due to the massive increase of urine output if GFR greatly increased. This was the case as GFR increased from 122.9 to 138.3 ml/min. The increase in turn led to an increase in the mount of water excreted in the urine, or the volume flow of the urine. This value of excreted water increased from 0.99 up to 1.26 ml/min. This is strong evidence for the occurrence of a pressure diuresis.

The increase in pressure also decreases the percentage of the filtered load of sodium that is reabsorbed by the tubules. The mechanism for this process is an increase in peritubular capillary hydrostatic pressure, which will allow more secretion and less reabsorption. The increase in renal interstitial fluid hydrostatic pressure creates a back leak problem where sodium leaks into the tubular lumen and therefore reducing the reabsorption of water and sodium. The fact that the excretion of sodium, concentration of sodium in the urine and excreted water all increased (reabsorption decreased) is evidence for both pressure natriuresis and pressure diuresis. The raised levels of sodium in the urine and levels of excreted sodium specifically characterize the pressure natriuresis.

The increase in pressure should lead to a decrease of the vasoconstrictor Angiotensin II. Therefore, the constriction of the efferent arteriole becomes lessened and blood flow will increase. The decrease in Angiotensin II will also result in a decrease of sodium reabsorption. The renal resistance should also decrease as the Angiotensin II levels drop and constriction is decreased. The

large increase in renal blood flow and decrease in renal resistance are sufficient evidence to conclude that the increased arterial pressure resulted in a decrease of Angiotensin II (see Fig 2).

Figure 1: Indication of Pressure Diuresis and Natriuresis by Increases Levels of Water and Sodium Excretion

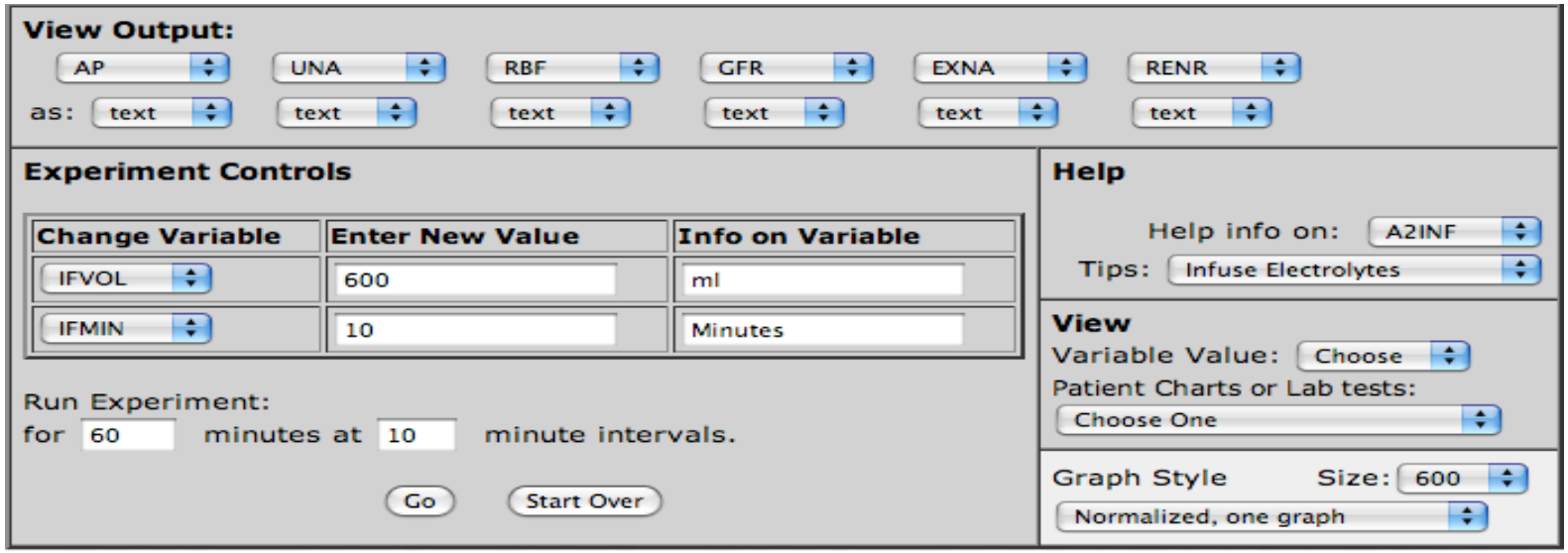


DAY/HR	AP	COL	O2DEBT	MFLOL	VENT	EXER
1-12:00 AM	99.78	5.438	0.000	1.100	5.675	0.000
1-12:05 AM	99.64	5.423	0.000	1.090	5.676	0.000

Total Infusion Volume (ml) ** Check IFMIN ** = 0.0000
Total Infusion Volume (ml) ** Check IFMIN ** = 600.00
Infusion Timespan (Minutes) = 10.000
Infusion Timespan (Minutes) = 10.000

DAY/HR	AP	UNA	EXH2O	GFR	EXNA	RENR
1-12:05 AM	99.64	115.9	0.9938	122.9	0.1151	0.8439E-01
1-12:15 AM	102.1	142.9	1.274	140.2	0.1821	0.7916E-01
1-12:25 AM	102.8	154.1	1.362	145.1	0.2098	0.7724E-01
1-12:35 AM	102.0	148.1	1.253	138.5	0.1856	0.7881E-01
1-12:45 AM	101.9	147.9	1.249	138.1	0.1847	0.7896E-01
1-12:55 AM	102.2	148.4	1.257	138.3	0.1865	0.7903E-01
1- 1:05 AM	102.3	148.4	1.259	138.3	0.1869	0.7910E-01

Figure 2: Indication of Decrease of Angiotensin II by Increased Renal Resistance and Renal Blood Flow



DAY/HR	AP	COL	O2DEBT	MFLOL	VENT	EXER
1-12:00 AM	99.78	5.438	0.000	1.100	5.675	0.000
1-12:05 AM	99.64	5.423	0.000	1.090	5.676	0.000

Total Infusion Volume (ml) ** Check IFMIN ** = 0.0000
Total Infusion Volume (ml) ** Check IFMIN ** = 600.00
Infusion Timespan (Minutes) = 10.000
Infusion Timespan (Minutes) = 10.000

DAY/HR	AP	UNA	RBF	GFR	EXNA	RENR
1-12:05 AM	99.64	115.9	1181.	122.9	0.1151	0.8439E-01
1-12:15 AM	102.1	142.9	1290.	140.2	0.1821	0.7916E-01
1-12:25 AM	102.8	154.1	1331.	145.1	0.2098	0.7724E-01
1-12:35 AM	102.0	148.1	1294.	138.5	0.1856	0.7881E-01
1-12:45 AM	101.9	147.9	1291.	138.1	0.1847	0.7896E-01
1-12:55 AM	102.2	148.4	1293.	138.3	0.1865	0.7903E-01
1- 1:05 AM	102.3	148.4	1293.	138.3	0.1869	0.7910E-01

EB October 22, 2008
Mammalian Physiology
Professor Meyers

Arterial Pressure Induced Pressure-Diuresis and Pressure-Natriuresis

The increase in mean arterial pressure has three main effects on the kidney system that will induce a pressure diuresis and a pressure natriuresis. The pressure diuresis, and the pressure natriuresis collectively result in an increase in urine output.

The patient was infused with 600 ml of electrolytes over 10 minutes, and evaluated over a period of one hour. A pressure diuresis is indeed occurring because the excreted H20 (EXH20) rises, and then slowly begins to normalize after the infusion of electrolytes. A natriuresis is also occurring because the urinary concentration of Na (UNA) also rises and slowly normalizes after the electrolyte infusion. (Table 1)

An increase in arterial pressure results in an increase in afferent arterial pressure into the glomeruli, this increases glomerular hydrostatic pressure, which increases GFR. The increase in GFR, although resisted via the glomerulotubular autoregulation system, still causes an increase in urine output because pressure diuresis and pressure natriures are induced. These results are shown in the decreased renal afferent arterial resistance, which causes an increase in glomerular hydrostatic pressure increasing GFR in table 1.

Total Infusion Volume (ml) ** Check IFMIN ** = 600.00
Infusion Timespan (Minutes) = 10.000
Infusion Timespan (Minutes) = 10.000
1.00000 Hours are 60.0000 Minutes

DAY/HR	AP	GFR	UNA	EXH2O	RAR	UPH
1-12:10 AM	99.69	123.0	116.0	0.9954	0.3301E-01	5.953
1-12:20 AM	102.1	140.2	142.9	1.274	0.3038E-01	6.112
1-12:30 AM	102.8	145.1	154.1	1.362	0.2998E-01	4.999
1-12:40 AM	102.0	138.5	148.1	1.253	0.3088E-01	6.088
1-12:50 AM	101.9	138.1	147.9	1.249	0.3086E-01	6.087
1- 1:00 AM	102.2	138.3	148.4	1.257	0.3090E-01	6.109
1- 1:10 AM	102.3	138.3	148.4	1.259	0.3093E-01	6.112

Table1

A rise in aterial pressure also causes an increase in peritubular capillary osmotic pressure, which causes a back up of H2O and Na into the tubular lumen, which leads to excess H2O and Na excretion. This process causes a decrease in Na absorption. Before infusion of Na, the patient's Na re-absorption is 99.36% of the filtered value. After the Na infusion, the Na re-absorption drops to 99.34%. The filtered loads, excreted loads, and amounts reabsorbed were calculated from the following data in Table 2.

DAY/HR	AP	PNA	GFR	EXH2O	UNA	EXNA
1-12:11 AM	99.54	144.1	122.8	0.9926	115.7	0.1149
1-12:11 AM	100.2	144.1	127.3	1.049	120.6	0.1266

Table2

The final factor associated with increased arterial pressure that affects the urine output through pressure diuresis and pressure natriuresis is reduced angiotensin II production. Angiotensin II is a vasoconstrictor, which ultimately causes a decrease in glomerular hydrostatic pressure, and a decrease in GFR. A decrease in angiotensin II dilates the renal arterioles and increases GFR. The decrease in angiotensin II is seen in the increase in renal blood flow (Table 3). As seen in Table 1, the renal afferent arterial resistance also decreases with a decrease in angiotensin II. Because angiotensin II also increases the secretion of H+, the decrease in angiotensin II also leads to an increase in urine pH. The increase in urine pH, however, may or may not be the result of other factors.

DAY/HR	AP	GFR	UNA	EXH2O	RBF	UPH
1-12:10 AM	99.69	123.0	116.0	0.9954	1181.	5.953
1-12:20 AM	102.1	140.2	142.9	1.274	1290.	6.112
1-12:30 AM	102.8	145.1	154.1	1.362	1331.	4.999
1-12:40 AM	102.0	138.5	148.1	1.253	1294.	6.088
1-12:50 AM	101.9	138.1	147.9	1.249	1291.	6.087
1- 1:00 AM	102.2	138.3	148.4	1.257	1293.	6.109
1- 1:10 AM	102.3	138.3	148.4	1.259	1293.	6.112

Table3

E G

Pressure Diuresis

DAY/HR	AP	COL	O2DEBT	MFLOL	VENT	EXER
1-12:00 AM	99.78	5.438	0.000	1.100	5.675	0.000
1-12:10 AM	99.69	5.425	0.000	1.089	5.673	0.000
DAY/HR	UPH	PPR	GFR	PH	EXBIC	BICARB
1-12:10 AM	5.953	7.333	123.0	7.410	-.4801E-01	23.69
1-12:11 AM	5.951	7.333	122.8	7.410	-.4796E-01	23.69

Total Infusion Volume (ml) ** Check IFMIN ** = 0.0000
Total Infusion Volume (ml) ** Check IFMIN ** = 600.00
Infusion Timespan (Minutes) = 10.000
Infusion Timespan (Minutes) = 10.000

DAY/HR	UNA	EXH2O	GFR	EXNA	RENR	AP
1-12:11 AM	115.7	0.9926	122.8	0.1149	0.8436E-01	99.54
1-12:16 AM	115.8	0.9934	122.9	0.1150	0.8438E-01	99.64
1-12:21 AM	142.9	1.274	140.2	0.1821	0.7916E-01	102.1
1-12:26 AM	174.9	1.645	151.8	0.2877	0.7738E-01	105.5
1-12:31 AM	154.1	1.362	145.1	0.2099	0.7724E-01	102.8
1-12:36 AM	150.1	1.286	140.4	0.1929	0.7832E-01	102.3
1-12:41 AM	148.1	1.253	138.5	0.1857	0.7880E-01	102.0
1-12:46 AM	148.1	1.253	138.3	0.1856	0.7896E-01	102.1
1-12:51 AM	147.9	1.250	138.1	0.1848	0.7896E-01	101.9
1-12:56 AM	148.2	1.255	138.3	0.1860	0.7900E-01	102.1
1- 1:01 AM	148.4	1.257	138.3	0.1865	0.7903E-01	102.2
1- 1:06 AM	148.6	1.261	138.4	0.1874	0.7908E-01	102.3
1- 1:11 AM	148.4	1.259	138.3	0.1869	0.7910E-01	102.3

Pressure diuresis is shown to occur as a result of a 600 mL infusion over the course of 10 minutes. Maximal increase in water excretion (from 0.9926 to 1.645 ml/min) is seen 15 minutes following the start of infusion. Pressure Natriuresis is shown to occur; maximal increase in sodium excretion (from 0.1149 to 0.2577 mEq/min) is seen to occur 15 minutes following the start of infusion. The sodium concentration of the urine is also shown to increase (from 115.7 to 174.9 mEq/min) which further supports the occurrence of pressure natriuresis.

Glomular Filtration Rate (GFR) is shown to increase (122.8 to maximum of 151.8 ml/min) which supports the role of increased GFR in the occurrence of pressure diuresis and natriuresis in response to increased arterial pressure.

Renal resistance is shown to decrease (0.08436 to 0.07724) which suggests a decrease in angiotensin II formation which acts to constrict efferent arterioles. Decreased levels of angiotensin II would result in decreased renal resistance due to decreased vascular constriction.

The percentage of sodium reabsorbed from the renal tubule decreases (99.4 to 99.0 %) following the 600 mL infusion. This suggests an increase in peritubular capillary hydrostatic pressure as well as an increase in renal interstitial fluid hydrostatic pressure as both of which are necessary to increase the amount of sodium excreted.

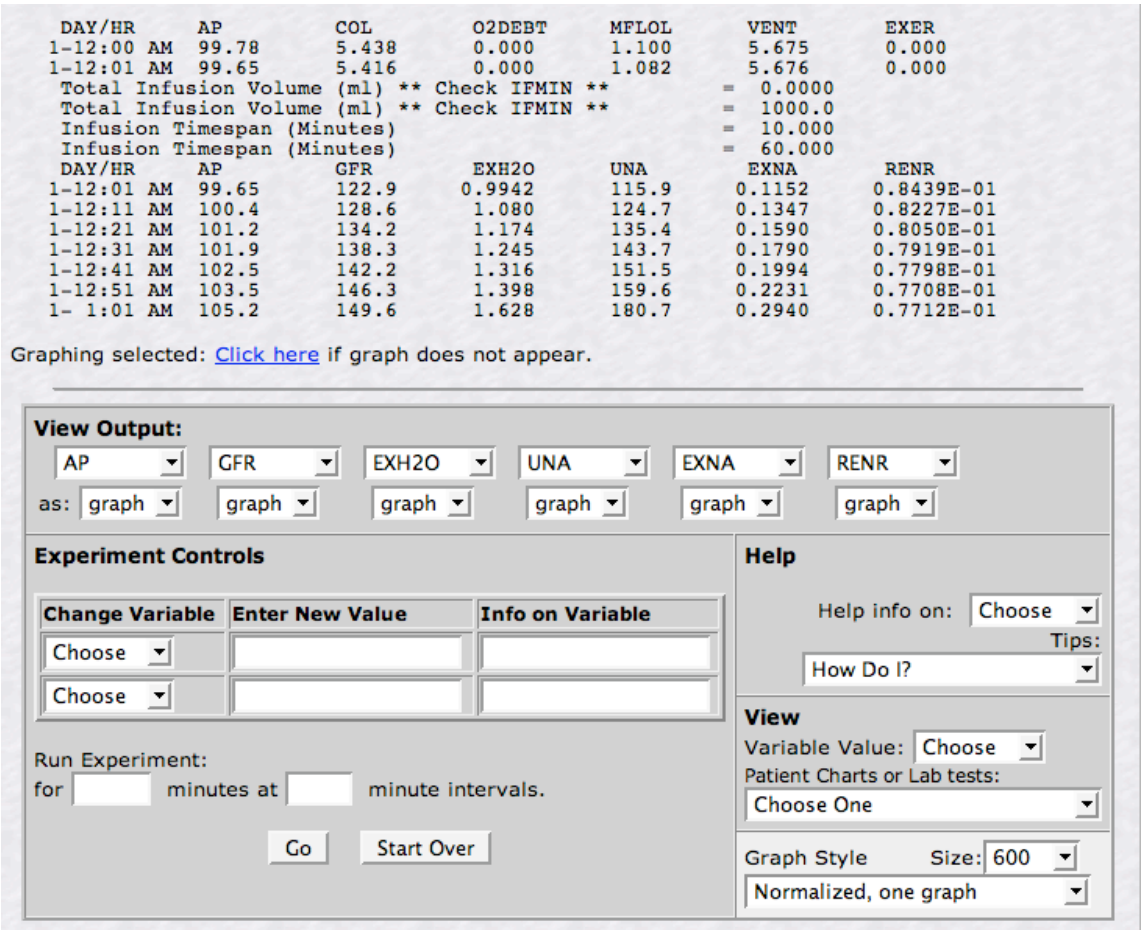
H P
Mammalian Physiology
Pressure Diuresis

A pressure diuresis and a pressure natriuresis were successfully induced by infusing the patient with1000 mL of electrolytes for a period of 60 minutes. The presence of diuresis is evident through the patient's increase in excretion of water (EXH2O) as a response to a slight increase in arterial pressure (AP increased from 99.65 to 105.2 mmHg in 60 min). Under normal conditions, an increase in blood pressure would not have an apparent effect in renal output due to various autoregulatory mechanisms. Therefore the increase in EXH2O from 0.994 to 1.628 mL/min indicates that the autoregulatory mechanisms are impaired. In addition, pressure natriuresis was shown through the patient's increase in excretion of sodium (EXNA) from 0.115 to 0.294 mEq/min as a response to a change in AP (more than twice the normal amount).

The lack of GFR autoregulation is shown through the patient's large increase in GFR with an increase in AP (122 to 149 mL/min). The impaired GFR autoregulation mechanism could be due to the decrease in angiotension II formation. Because angiotensin II preferentially constricts efferent arterioles, a decreased angiotensin II level lowers glomerular hydrostatic pressure while increasing renal blood flow. The increase in renal blood flow in turn leads to an increase in GFR. The decrease in angiotension II production can be seen through the patient's overall decrease in renal resistance (RENR) from 0.084 to 0.077 mmHg/ml/min, and his increase in blood flow from 1181 to 1364 mL/min). In addition, the decrease in angiotensin can also be witnessed through the decrease in aldosterone secretion. Since aldosterone increases potassium secretion, an

increase in aldosterone would lead to an increase in absorption. This is shown through the decrease in the patient's urine potassium level (decreased from 41.64 to 25.12 mEq/L).

Furthermore, another effect of increased renal arterial pressure that raises urine output is the decrease of the percentage of the filtered load of sodium and water that is reabsorbed by the tubules. The increase in the peritubular capillary hydrostatic pressure is shown through the drastic increase in urine sodium concentration (UNA) from 115 to 180 mEq/L, and also the increase in sodium excretion (EXNA) from 0.1 to 0.3 mEq/min. Both increased value showed a back-leak of sodium into the tubular lumen due to an increased renal interstitial fluid hydrostatic pressure as a response to a higher AP. The decreased net reabsorbance therefore led to an overall increase in the rate of urine output.



DAY/HR	AP	COL	O2DEBT	MFLOL	VENT	EXER
1-12:00 AM	99.78	5.438	0.000	1.100	5.675	0.000
1-12:01 AM	99.65	5.416	0.000	1.082	5.676	0.000

Total Infusion Volume (ml) ** Check IFMIN ** = 0.0000
Total Infusion Volume (ml) ** Check IFMIN ** = 1000.0
Infusion Timespan (Minutes) = 10.000
Infusion Timespan (Minutes) = 60.000

DAY/HR	AP	GFR	EXH2O	UNA	EXNA	RENR
1-12:01 AM	99.65	122.9	0.9942	115.9	0.1152	0.8439E-01
1-12:11 AM	100.4	128.6	1.080	124.7	0.1347	0.8227E-01
1-12:21 AM	101.2	134.2	1.174	135.4	0.1590	0.8050E-01
1-12:31 AM	101.9	138.3	1.245	143.7	0.1790	0.7919E-01
1-12:41 AM	102.5	142.2	1.316	151.5	0.1994	0.7798E-01
1-12:51 AM	103.5	146.3	1.398	159.6	0.2231	0.7708E-01
1- 1:01 AM	105.2	149.6	1.628	180.7	0.2940	0.7712E-01

Graphing selected: Click here if graph does not appear.

View Output:
AP | GFR | EXH2O | UNA | EXNA | RENR
as: graph | graph | graph | graph | graph | graph

Experiment Controls

Change Variable	Enter New Value	Info on Variable
Choose		
Choose		

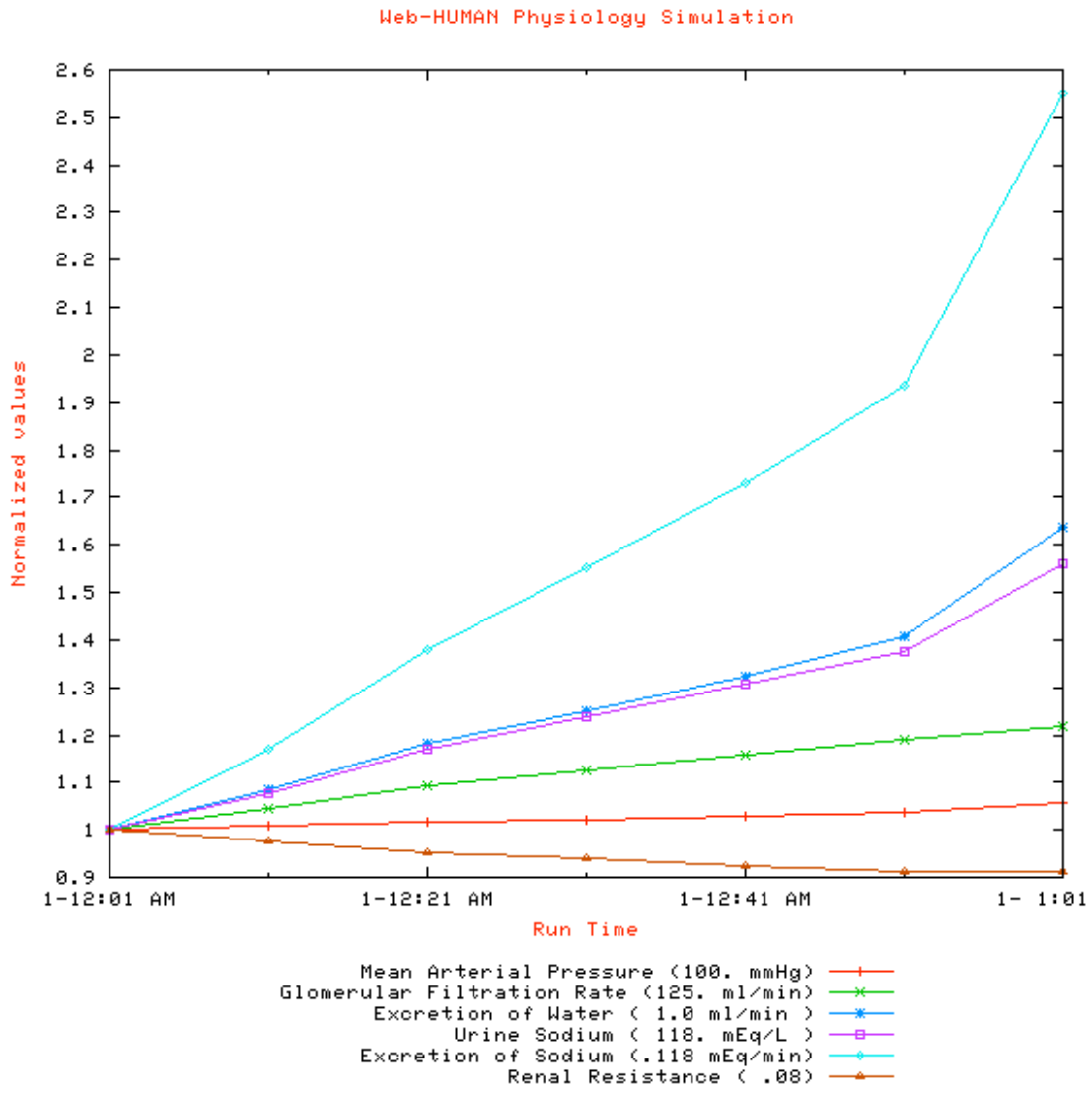
Run Experiment:
for minutes at minute intervals.
Go | Start Over

Help
Help info on: Choose
Tips:
How Do I?

View
Variable Value: Choose
Patient Charts or Lab tests:
Choose One

Graph Style Size: 600
Normalized, one graph

UK	RBF
41.64	1181.
39.30	1220.
36.26	1258.
33.88	1286.
31.73	1314.
29.60	1343.
25.12	1364.



J R
Pressure Diureisis

DAY/HR	AP	GFR	EXH2O	UNA	EXNA	PNA
1-12:10 AM	99.69	123.0	0.9954	116.0	0.1155	144.0
1-12:20 AM	105.4	150.6	1.586	169.2	0.2684	144.8
1-12:30 AM	107.2	157.6	2.003	201.0	0.4026	144.7
1-12:40 AM	104.1	147.6	1.514	173.9	0.2634	144.7
1-12:50 AM	104.4	147.1	1.518	174.2	0.2644	144.7
1- 1:00 AM	104.8	147.0	1.543	176.2	0.2719	144.6
1- 1:10 AM	105.1	146.8	1.566	177.9	0.2785	144.6
DAY/HR	AP	GFR	EXH2O	UNA	EXNA	RBF
1- 1:10 AM	105.1	146.8	1.566	177.9	0.2785	1351.
1- 1:10 AM	105.2	146.7	1.572	178.2	0.2802	1350.

To cause the increase in arterial pressure from 99.69-105 mmHg, necessary to induce a pressure diuresis and natriuresis, the patient was infused with 1L electrolytes for 10 min. The patient was then run for 50 minutes following that, while recording arterial pressure (AP), Glomerular Filtration rate (GFR), H20 excretion (EXH20), concentration of Na in the urine (UNA), excretion of urine (EXNA), plasma concentration of Na (PNA), and renal blood flow (RBF).

The patient's arterial pressure reached a high of 107.2 then steadied out at a 105.2 mmHg. We can see that a pressure-diuresis is occurring from the clear increase in the H20 excretion up to 2.003 ml/min at its highest. We also can see that a pressure-natriuresis

is occurring from the increase in Na excretion up to .4026 mEq/min from the normal at its highest.118 mEq/min. There is also a higher concentration of Na in the urine, 178.2 mEq/ml, than normal, 118 mEq/ml.

The first factor that is at play in the pressure diuresis and natriuresis is the slight increase in GFR, as seen here from 123 to 157.6 ml/min at its highest value.

The second factor causing the increase in urine output response to increased arterial pressure is a decrease in the percent of the filtered load that is reabsorbed. This is exhibited in our patient in the increase of Na excretion and concentration of Na in the urine, while the plasma urine remains the same. The % reabsorbed of filtered Na (calculated as ((GFR * PNA) – EXNA) /(GFR*PNA)) is down to 98 % from the expected 99.9 %.

The last factor in effect is a decrease in angiotensin II that in turn leads to a decrease in sodium reabsorption. Because angiotensin II is a known vasoconstrictor, we can measure the change in angiotensin II levels based on renal blood flow. A decrease in angiotensin II would therefore cause a vasodilation and in turn a higher renal blood flow. This is seen in our patient, with RBF increasing up to 1350 ml/min from a normal value of 1200 ml/min, confirming that angiotensin II levels have dropped.

JW
October 22, 2008
Pressure Diuresis Lab

The increased Arterial Pressure from 99.69 to 102.3 mm Hg indicates the primary cause for both a Pressure-Diuresis and a Pressure-Natriuresis. A Pressure-Diuresis is seen to be occurring when 600 ml of electrolytes are infused over a ten-minute period. The increased water loss from time 0 is from 0.9954 ml/min to 1.259 ml/min. The increase of Na+ excretion (0.1155 mEq/min to 0.1869 mEq/min) indicates a Pressure-Natriuresis is also occurring.

Table 1. Arterial Pressure, Urine excretion and Sodium excretion in a patient with 600 mL Electrolytes infused over a 10 minute period.

DAY/HR	AP	EXH2O	EXNA
1-12:10 AM	99.69	0.9954	0.1155
1-12:20 AM	102.1	1.274	0.1822
1-12:30 AM	102.8	1.362	0.2098
1-12:40 AM	102.0	1.253	0.1856
1-12:50 AM	101.9	1.249	0.1847
1- 1:00 AM	102.2	1.257	0.1865
1- 1:10 AM	102.3	1.259	0.1869

The first mechanism involved with the Pressure-Diuresis and a Pressure-Natriuresis is an increased GFR (from 144 L/24H to 199.152 L/24H) due to the impairment of GFR autoregulation. The increased GFR can be seen in table 2. The second mechanism is a decreased amount of the filtered load of NA+ that is reabsorbed. The decrease in the percentage of Na+ that is reabsorbed is from 99.3% (normal) to 99.06% (after 60 minutes). These values were determined by calculating the Filtered load and Excreted load and comparing the percent reabsorbed initially to the percent reabsorbed finally.

Table 2. Renal Na+ Reabsorption Calculations

	GFR L/24H	PNA mEq/L	Filtered Load mEq/L	EXH2O L/24H	UNA mEq/L	Excreted Load mEq/L	Amount reabsorbed	% Reabs.
initial	144.0	176.976	25484.5	1.44	116.0	169.04	25317.46	99.3
final	199.152	144.4	28757.6	1.81	148.4	269.04	28488.51	99.06

The third mechanism involved is the reduction of Angiotensin II. The angiotensin II restricts the Efferent Arteriole. However, the lack of Angiotensin II causes a decrease in resistance as shown by the data below (Table 3). This lack of resistance indicates a lack of Angiotensin II. The increase in blood flow also indicates the decreased resistance.

Table 3. Renal resistance and renal blood flow.

DAY/HR	RBF	RENR
1-12:10 AM	1181.	0.8439E-01
1-12:20 AM	1290.	0.7916E-01
1-12:30 AM	1331.	0.7724E-01
1-12:40 AM	1294.	0.7880E-01
1-12:50 AM	1291.	0.7896E-01
1- 1:00 AM	1293.	0.7903E-01
1- 1:10 AM	1293.	0.7910E-01

K S Pressure Diuresis

In order to determine the mechanisms of pressure-natriuresis and pressure-diuresis to counteract increased arterial pressure, an otherwise normal model was infused with 600 ml of electrolytes over 10 minutes and then observed for another 50 minutes at 10 minute intervals (figure 1).

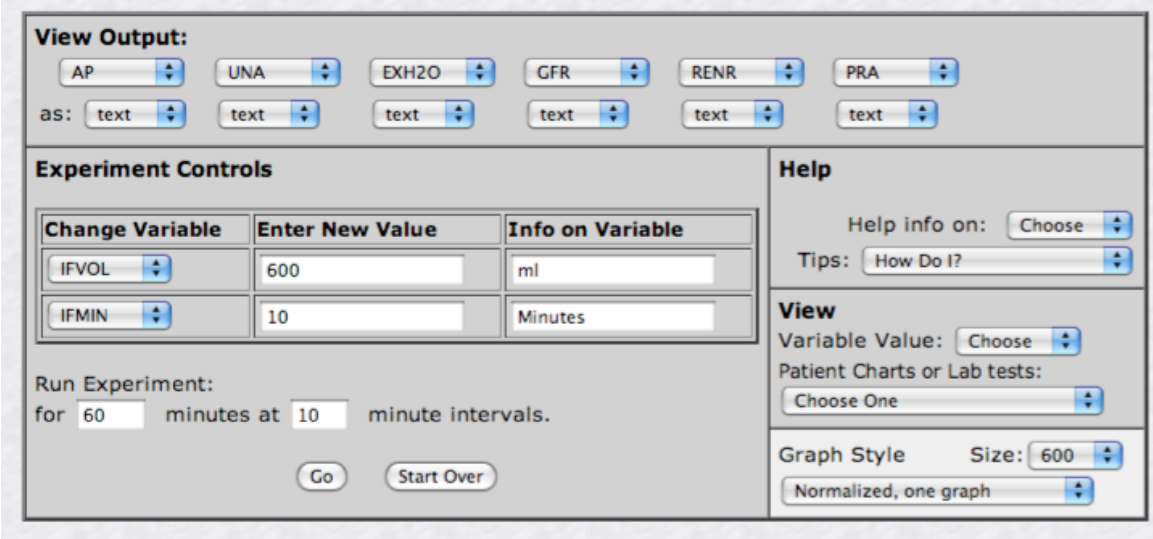


Figure 1

It can be determined that a pressure-diuresis is occurring by looking at the increase in excreted water was a result of the electrolyte infusion. While the normal value of excreted urine is 0.9954 ml/minute, this value increases to 1.27 ml/min at the time of the infusion, and increases even further to 1.36 ml/min ten minutes after the infusion (figure 2). This is a result of the function of the kidney to excrete excess water and return arterial pressure to normal, or pressure-duiresis.

Likewise, it can be determined that pressure-natriuresis is occurring by looking at the values of sodium concentration in the urine before and after the infusion. Before the infusion, the value of sodium in the urine is 116.0 mEq/L while at the time of the infusion this value increases to 142.9 mEq/L and increases again 10 minutes after the infusion to 154.1 mEq/L(figure 2) which is evidence that the kidney is excreting sodium to attempt to counteract the increased arterial pressure.

One of the three mechanisms that is involved in the process is an increase in GFR which can be seen by the value increasing from 123.0 ml/min to 140.2 ml/min at the time of infusion and then again to 145.1 ml/min at ten minutes after the infusion (figure 2).

The second mechanism is a decrease in the percentage of the filtered load of sodium and water that is reabsorbed by the tubules. The evidence for this mechanism working can be seen in the lack of change of concentration of sodium in the plasma that is filtered, but an increase in the sodium concentration of the urine (figure 2). This would indicate that

although the same amount is being filtered, more is being excreted, which means that less is being reabsorbed.

Another of the three mechanisms is a decrease in angiotensin II which leads to a decrease in renal vascular resistance. This is evidenced in the value increasing from 0.08439 mm Hg/ml/min to 0.07916 mm Hg/ml/min at the time of the infusion to 0.07724 mm Hg/ml/min at ten minutes after the infusion (figure 2). The function of angiotensin II is to increase vasoconstriction, therefore this evidenced decrease in vasoconstriction (resistance) points to a decrease in angiotensin II.

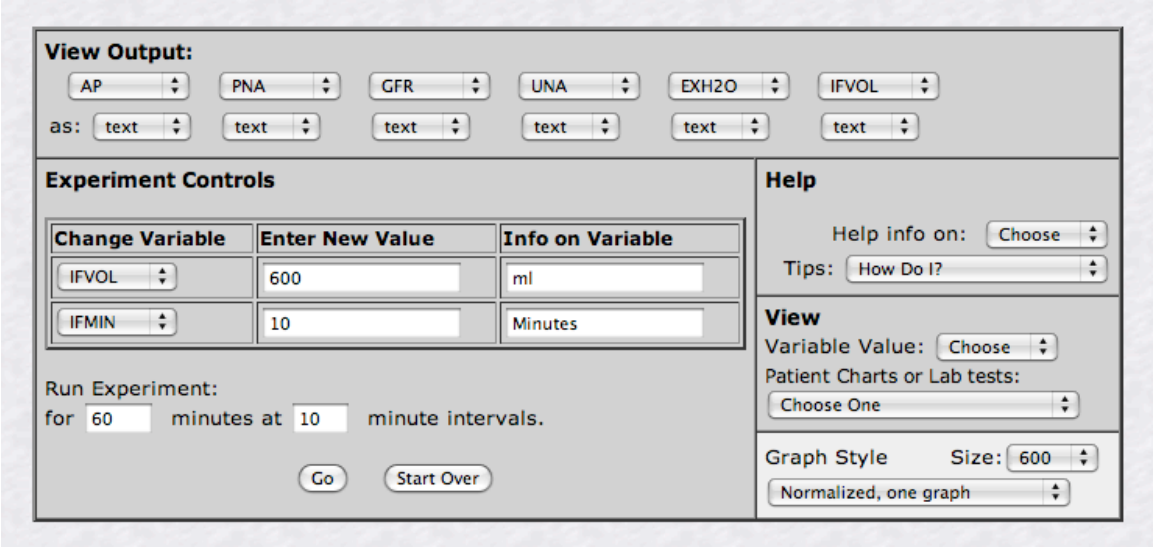
```
Total Infusion Volume (ml) ** Check IFMIN **          =  0.0000
Total Infusion Volume (ml) ** Check IFMIN **          =  600.00
Infusion Timespan (Minutes)                           =  10.000
Infusion Timespan (Minutes)                           =  10.000
 DAY/HR      AP          UNA         EXH2O       GFR         RENR         PNA
1-12:10 AM  99.69       116.0      0.9954       123.0     0.8439E-01   144.0
1-12:20 AM  102.1       142.9       1.274       140.2     0.7916E-01   144.5
1-12:30 AM  102.8       154.1       1.362       145.1     0.7724E-01   144.5
1-12:40 AM  102.0       148.1       1.253       138.5     0.7880E-01   144.4
1-12:50 AM  101.9       147.9       1.249       138.1     0.7896E-01   144.4
1- 1:00 AM  102.2       148.4       1.257       138.3     0.7903E-01   144.4
1- 1:10 AM  102.3       148.4       1.259       138.3     0.7910E-01   144.4
```

Figure 2

R N

Pressure Diuresis

An experiment can be set up by the following parameters:



Infusion of 600 mL over a 10 minute period gives the following results:

DAY/HR	AP	COL	O2DEBT	MFLOL	VENT	EXER
1-12:00 AM	99.78	5.438	0.000	1.100	5.675	0.000
1-12:05 AM	99.64	5.423	0.000	1.090	5.676	0.000

Total Infusion Volume (ml) ** Check IFMIN ** = 0.0000
Total Infusion Volume (ml) ** Check IFMIN ** = 600.00
Infusion Timespan (Minutes) = 10.000
Infusion Timespan (Minutes) = 10.000

DAY/HR	AP	GFR	UNA	EXH2O	ALDO	UK	EXNA	RAR	RBF
1-12:05 AM	99.64	122.9	115.9	0.9938	7.977	41.64	0.1151	0.3299E-01	1181.
1-12:15 AM	102.1	140.2	142.9	1.274	6.849	34.43	0.1821	0.3038E-01	1290.
1-12:25 AM	102.8	145.1	154.1	1.362	5.794	31.03	0.2098	0.2998E-01	1331.
1-12:35 AM	102.0	138.5	148.1	1.253	5.839	32.38	0.1856	0.3088E-01	1294.
1-12:45 AM	101.9	138.1	147.9	1.249	5.880	32.57	0.1847	0.3086E-01	1291.
1-12:55 AM	102.2	138.3	148.4	1.257	5.917	32.64	0.1865	0.3090E-01	1293.
1- 1:05 AM	102.3	138.3	148.4	1.259	5.954	32.83	0.1869	0.3093E-01	1293.

After the infusion, as a direct result of an increase in electrolyte infusion, the arterial pressure increases slightly to increase urine output. One effect this has on the kidney is the increase in GFR from the initial value of 122.9 to 145.1 mL/min. This increases the volume of urine excreted.

Another effect of the increased renal arterial pressure is the decrease in the reabsorption of water and sodium by the tubules. This is indicated by several factors. The sodium concentration in the plasma remains relatively constant, and the GFR increases only slightly. However, the excretion of sodium increases, from the initial value of 0.1151 to 0.2098 ml/min. The sodium concentration increases as well, which gives an overall increase in excreted load. The initial excreted load is 115.2 mEq/min compared

to an almost doubled value of 205.9 mEq/min after infusion. This results in a lower reabsorption after the infusion. There is also an increase in the excretion of water from 0.9938 mL/min to 1.362 after 10 minutes of infusion as well as an increase in the sodium concentration in the urine.

Angiotension II increases sodium and water reabsorption using three different techniques. One technique is directly stimulating sodium transport across luminal and basolateral surfaces of the epithelial cell membrane of the tubules. An increase in angiotension II would increase sodium reabsorption by increasing the secretion of aldosterone. Aldosterone regulates not only sodium reabsorption but also potassium secretion. After the infusion of electrolytes, there is a dramatic decrease in aldosterone from 7.977 ng/dl to 5.794 ng/dl. This is reflected in the decreased sodium reabsorption and potassium concentration in urine.

Angiotensin II also constricts the efferent arterioles, which reduces the hydrostatic pressure in the peritubular capillary. This also reduces renal blood flow, which causes an increase in the colloid osmotic pressure in the peritubular capillaries. Both of these reductions lead to an overall increase in tubular reabsorption. After infusion of the electrolytes, the renal blood flow increased, an indication, along with the lowered aldosterone levels, that angiotensin II levels are reduced. The implication is that less sodium and water are reabsorbed.

All the experimental results signifiy there is lower reabsorption of sodium and water in the patient. Lower reabsorption back into the body indicate more excretion of the substances out. This can be referred to as pressure-natriuresis and pressure-diuresis.

Because the experiment extended past the infusion, the kidney levels return to autoregulation after infusion.