International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2013): 6.14 | Impact Factor (2015): 6.391

Mathematical Model of Minimal Repair for Excess Diuresis and Natriuresis during Acute Sleep Deprivation in Healthy Adults

Geetha .T[1], Sangeetha .B[2]

[1]Asst.Professor of Mathematics, K.N.Govt. Arts College for Women Thanjavur. Tamilnadu. South India.

[2]Lecturer of Mathematics, BDU (W) College. Orathanadu. Thanjavur. Tamilnadu. South India

Abstract: ***The transition from wakefulness to sleep is associated with a pronounced decline in diuresis, a necessary physiological process that allows uninterrupted sleep. The aim of this study was to assess the effect of acute sleep deprivation (SD) on urine output and renal water, sodium, and solute handling in healthy young volunteers. The effect was more pronounced in men who shared significantly higher diuresis levels during SD compared with women. Renal water handling and arginine vasopressin levels remained unaltered during SD, but the circadian rhythm of the hormones of the renin-angiotensin-aldosterone system was significantly affected. This paper is applied to Optimal time T*, Minimal Repair δ and Random Lead Time g to minimize the renin level.***

Keywords: SD, Renin, minimal repair, Repair cost limit function

NOTATIONS

Xj = time between the successive Reninlevel [j = 1,2,…….n], r.v.
yj = amount of damage to the Rennin level due to depression[j = 1,2,…….n], r.v.
f(y) – pdf of time to damage of Renin level
g(x) – pdf of lead time
δ(y) - Repair cost limit function of Rennin level
T* - optimal time
C1 – cost in terms of Renin level

1. Assumption

This model has random leadtime & minimal repair.
Two types of failure occur
1. Type-I : with probability q(y) and is corrected with minimal repair
2. Type-II : with probability p(y) = 1-q(y) and followed by unit replacement.

2. Application

Sleep is the essential behavioural state that covers approximately one-third of our lives. Urine production is reduced during sleep, a necessary physiological process that allows sleep to be continued uninterrupted. Failure to do so leads to excess nocturnal diuresis, a common finding in clinical settings such as enuresis in children and nocturia in the elderly. The sleep-arousal cycle affects a number of physiological processes considered important for renal function and the production of urine. Blood pressure is reduced during sleep in normotensive humans . Lack of this nocturnal dipping in blood pressure (6, 7) is a common finding in hypertension.

Acute sleep deprivation (SD) leads to increased sympathetic activity and higher blood pressure , heart rate , and catecholamine excretion. Circadian variations are to be found in arginine vasopressin (AVP) secretion in children , and the hormones of the renin-angiotensin-aldosterone system (RAAS), all-important modulators of urine production(3, 4, 5). Lower nocturnal AVP levels have been demonstrated in enuretics with excess nocturnal urine production although the exact reasons for this blunted AVP rhythm remain unclear. Recent research indicates excess sodium and osmotic excretion in selected populations of enuretics with nocturnal polyuria, as well as adults with nocturia. Furthermore, studies indicate sleep architecture disturbances in children with enuresis although the relationship with renal function was not addressed. It has been hypothesized that abnormalities in nocturnal urine production may be the result of sleep architecture disturbances, but we are still short of experimental data that would allow definite conclusions.

The aim of this study was to investigate the impact of acute SD on the nocturnal urine production in young adults and to evaluate renal water and solute handling in a sleep-deprived state. A secondary aim was to evaluate the possible role of gender on the renal response to acute SD(8, 9).

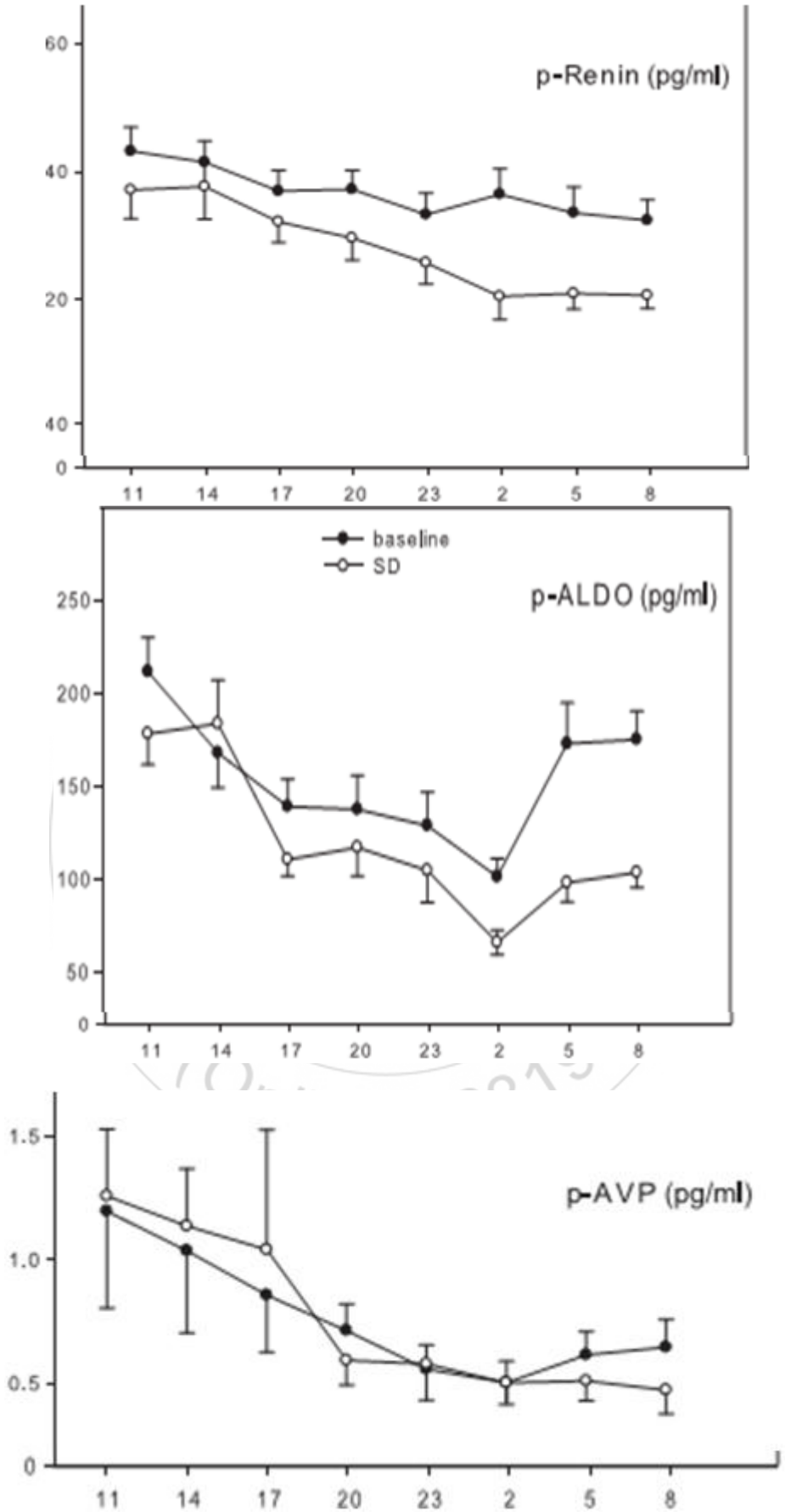


Figure 1: Diurnal variations in plasma (p) levels of aldosterone (ALDO), renin, arginine vasopressin (AVP) during baseline and SD.

3. Materials and Methods

Study subjects. The study protocol was approved by the local regional committee on biomedical research ethics, and informed consent was obtained from all participants. The protocol conformed to the recommendations for good clinical practice. Twenty healthy adults (10 women) aged 18 –35 yr (mean age 251.5 yr) were included in the study. Healthy controls were recruited through the personnel and their acquaintances of the Department of Pediatrics and Department of Urology, Aarhus University Hospital, Skejby. Inclusion criteria for the participants were as follows: no history of urinary incontinence, no clinical or laboratory signs suggestive of an underlying disease, a normal uroflowmetry with residual urine assessment, an unremarkable clinical examination, and normal urinalysis.

4. Mathematical Model

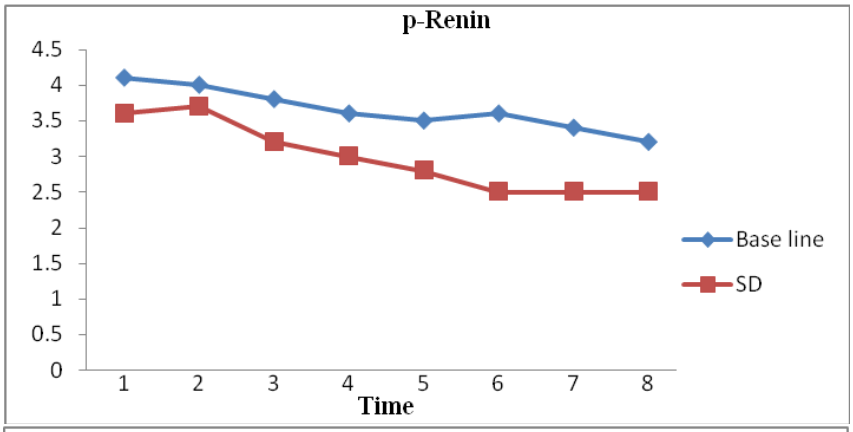
Consider the system with a weibull distribution. The pdf of the weibull distribution with parameters β and θ is given by $f(y) = \beta/\theta\ (y/\theta)\ \beta\text{-}1\exp(-y/\theta)\ \beta$, $y > 0$, $\beta, \theta > 0$ where $\beta = 1.326$; $\theta = 5.326$

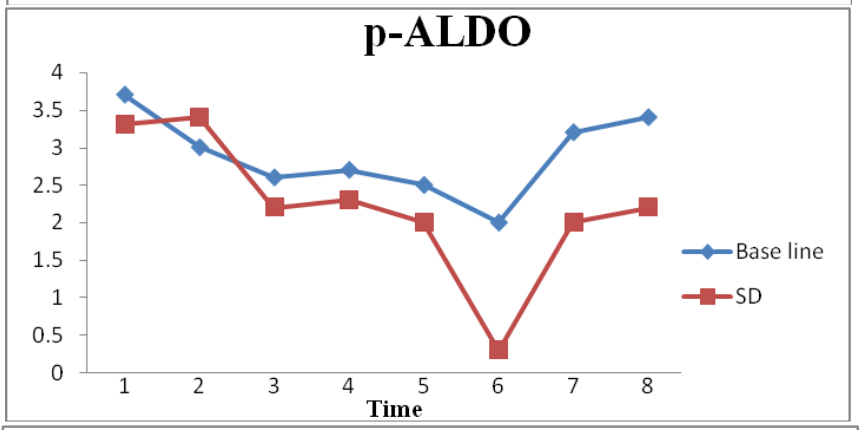
The pdf of the random lead time of an order is,
$g(x) = 1\mu\exp(-x/\mu)$, $x > 0$, $\mu > 0$. Where $\mu = 3.5217$

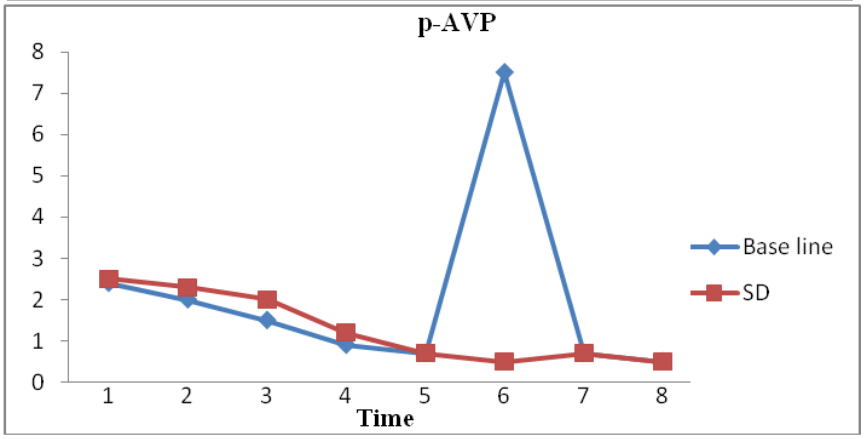
Suppose the random repair cost is ω, If $\omega \leq \delta(y)$. $c\infty$ ($c\infty \equiv$ the constant cost) then there is a minimal repair.

If δ(y).can be explained as a fraction of the constant cost , $c\infty$, at age y and $0 \leq \delta(y) \leq 1$.

Let $\delta(y) \equiv \delta(\exp(-\lambda,y))$ with $0 \leq \delta(y) \leq 1$ & $\lambda \geq 0$.







Now, The optimal time T* which minimizes C1(T) is, (1, 2)
$C1(T^*) = \lambda\ C1\ (1\text{-}g(x))\ e\text{-}\lambda(1\text{-}g(x))T^*$
When C1= 5.1

λ=0.214
g(x)=0.1241
T*= 2.4 then C1 (T*) = 0.0187

5. Conclusion

Acute SD leads to excess urine production, an effect evident in both sexes although more pronounced in men. The amount of urine produced during these sleepless nights by far exceeds bladder reservoir ability, thus leading to nocturia. Suppression of the RAAS may be the result of a direct effect of SD on the sensitivity of the RAAS but could also be mediated through sympathetic-parasympathetic system disparities. Variations exist in renin levels during the different sleep stages, with suppressed levels during non-REM sleep , a sleep stage characterized by increased sympathetic activity . It can be thus hypothesized that SD, being a stressor factor, may increase sympathetic activity, thus resetting the RAAS regulation, with natriuresis as the net effect. Also found Optimal time T*, Minimal Repair and Random Lead Time to minimize the rennin level.

References

[1] Berg.M and Cleroux.R, "A marginal cost analysis for an age replacement policy with minimal repair", vol.20, 258-263, 1982.
[2] Block H.W, Borges.W.S and Savits.T.H, „"Age dependent minimal repair", J.Applied probability, Vol.22, pp370-385, Jun 1985.
[3] Charloux A, Gronfier C, Chapotot F, Ehrhart J, Piquard F, Brandenberger G.Sleep deprivation blunts the night time increase in aldosterone release in humans.J Sleep Res10: 27–33, 2001.
[4] Charloux A, Gronfier C, Lonsdorfer-Wolf E, Piquard F, Brandenberger G.Aldosterone release during the sleep-wake cycle in humans.Am J Physiol Endocrinol Metab276: E43–E49, 1999.
[5] Charloux A, Piquard F, Ehrhart J, Mettauer B, Geny B, Simon C,Brandenberger G.Time-courses in renin and blood pressure during sleep in humans. J Sleep Res11: 73–79, 2002.
[6] Clerico A, Del Ry S, Maffei S, Prontera C, Emdin M, Giannessi D.The circulating levels of cardiac natriuretic hormones in healthy adults: effects of age and sex.Clin Chem Lab Med40: 371–377, 2002.
[7] Coca A.Circadian rhythm and blood pressure control: physiological and pathophysiological factors.J Hypertens Suppl12: S13–S21, 1994.
[8] Dhondt K, Raes A, Hoebeke P, Van Laecke E, Van Herzeele C, Vande Walle J.Abnormal sleep architecture and refractory nocturnal enuresis.J Urol182: 1961–1965, 2009.
[9] Donckier J, Anderson JV, Yeo T, Bloom SR.Diurnal rhythm in the plasma concentration of atrial natriuretic peptide.N Engl J Med315: 710 –711, 1986.