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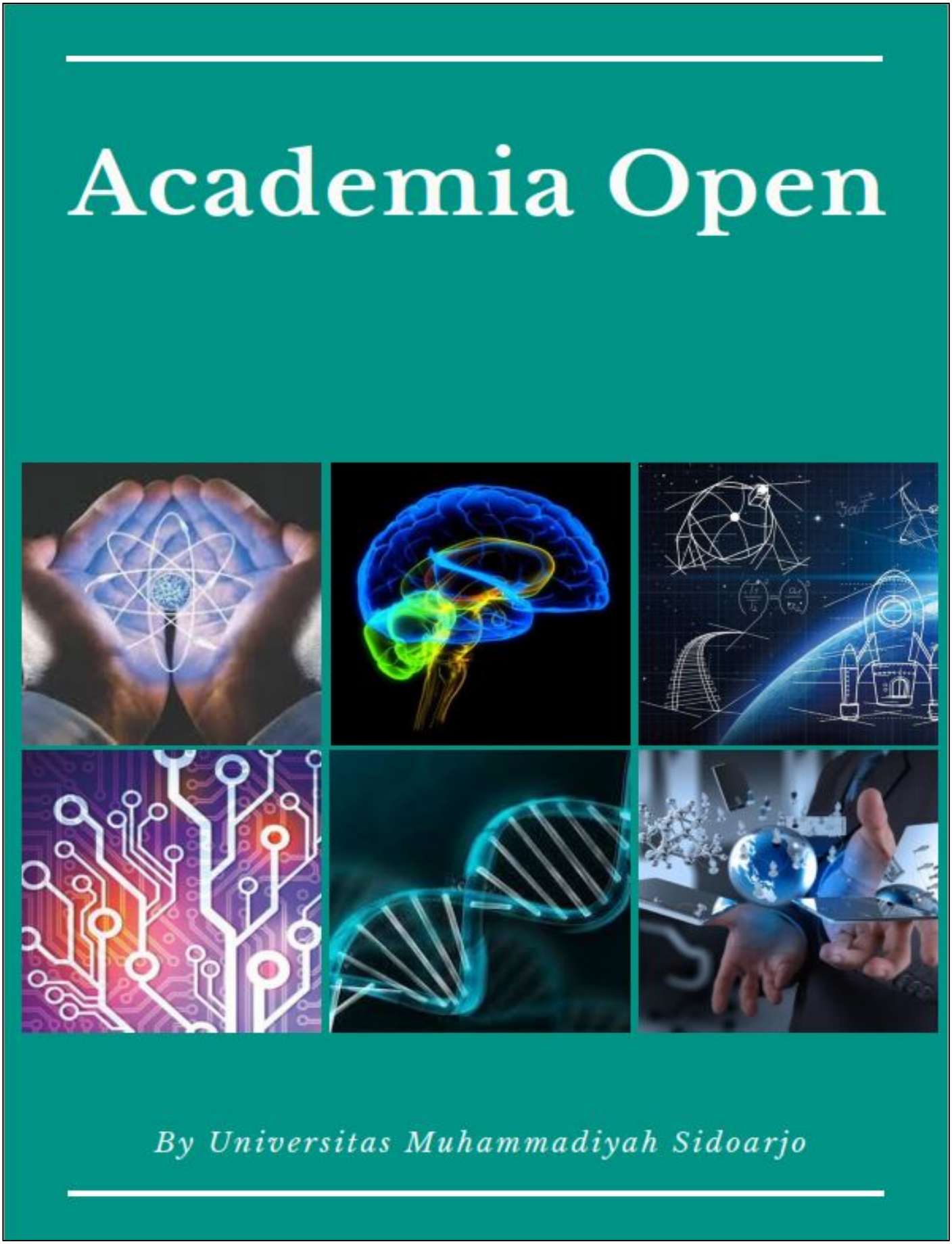

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Physiology of Nerve Supply for Renal System

Fisiologi Suplai Saraf untuk Sistem Ginjal

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Abstract

General Background: The renal nervous system regulates kidney function through sympathetic and sensory neurons, impacting blood pressure and fluid balance. **Specific Background:** Renal nerve hyperactivity is linked to hypertension and metabolic disorders, prompting interest in renal denervation as a treatment. **Knowledge Gap:** The precise mechanisms by which renal nerves influence disease remain unclear, and clinical outcomes of renal denervation are inconsistent. **Aims:** This review explores renal nerve structure, neurotransmission, and functional roles in health and disease. **Results:** Sympathetic nerves release norepinephrine and co-transmitters, affecting vascular resistance and sodium handling, while sensory nerves modulate sympathetic output. **Novelty:** It integrates recent findings on renal neurophysiology, emphasizing afferent-sympathetic interactions. **Implications:** Advancing knowledge of renal nerves could refine treatments for hypertension and kidney disorders.

Highlights:

- Renal nerve signaling controls blood pressure and kidney function through neurotransmitters like norepinephrine and ATP.
- Renal denervation therapy shows potential for treating hypertension but has inconsistent clinical outcomes.
- Afferent-sympathetic interaction plays a crucial role in kidney function and systemic homeostasis

Keywords: Renal Nerves, Sympathetic Regulation, Hypertension, Neurotransmitters, Renal Denervation

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Introduction

Surgical renal denervation (RDN) has been shown to reduce the development of experimental hypertension, which lends credence to the theory high blood pressure is managed by renal nerves [1, 2]. This has been applied to catheter-based renal nerve ablation for the treatment of hypertension in people.CBRNA destroys the kidneys' sensory and sympathetic innervation [3, 4]. Furthermore, unexpected results from clinical studies with CBRNA have reignited interest in learning more about how renal neurons function in disease conditions. Some CBRNA-treated patients showed decreased sympathetic activity to skeletal muscle, improved glucose metabolism, and a lower incidence of cardiac arrhythmias. These results imply that the mechanism by which CBRNA's therapeutic effects are transmitted may be renal sensory (afferent) neurons, which interact with Several organs' sympathetic activity and can be altered by the central nervous system [5, 6].

Method

A comprehensive review and analysis of existing literature were conducted, focusing on sympathetic and sensory renal innervation, neurotransmitter signaling pathways, and the impact of renal nerve modulation on renal and systemic functions.

Result and Discussion

A. Sympathetic Innervation of Renal Vascular Structures

Sympathetic renal nerve fibers in the kidney emit norepinephrine (NE). The effects of NE on adrenoceptors have the greatest impact on renal function [5, 6], Autoradiography [7] electron microscopy [8] immunohistochemistry [9]. It has NE, the enzyme that limits the rate of biosynthesis. As always Neuroeffector connections in the kidney and the peripheral nervous system are made up of sympathetic Varicosities of fibers close to their effector [10, 11]. These Multiple structures may be functionally innervated thanks to en passant connections while a fiber travels past them [12] Furthermore, transmission across these varicosities has been either through capillary transport or diffusion in the renal interstitium [7].

Sympathetic renal nerves travel to the afferent and efferent arterioles in the renal cortex via the renal artery and its resulting vascular branches. The highest density of TH+ sympathetic fiber innervation is found in the afferent arterioles [7]. The afferent and efferent arterioles' sympathetic activity has particular functional significance [13, 14]. TH+ adrenergic sympathetic neurons also innervate juxtaglomerular granular cells that secrete renin in the afferent arteriole. Lastly, capillaries have a lower density than arterioles and an unknown function, although appearing to contain sympathetic innervation. The sympathetic innervation of the vasculature connecting the superficial and deep nephrons does not seem to differ [7].

1. Sympathetic Renal Tubular Structure Innervation

Although not as thickly as the vasculature, sympathetic fibers innervate a number of tubular structures [15]. According to tritiated NE uptake and autoradiography, the thick ascending limb possessed. In comparison to other structures, the collecting ducts have minimal innervation [7].

2. Renal Pelvic Sympathetic Innervation

According to NPY immunolabeling, the pelvic wall's sympathetic innervation is less numerous than the vasculature [16]. In the pelvis, sensory and sympathetic fibers sometimes move in bundles with sensory fibers, although they also disseminate independently [17, 18]. Changes in sensory fiber activity and/or the contraction of the pelvic smooth muscle are most likely linked to the sympathetic fibers' actions in the renal pelvis [19].

B. Neural Pathway Anatomy from the Brain to the Kidney

The sympathetic innervation of the kidney is made up of a two-neuron route, just like that of any other visceral organ. The cell body of the first neuron (T1–L2) is located in the spinal cord's intermediolateral (IML) cell column. After projecting to a prevertebral (such as the celiac, superior mesenteric, or aortico-renal) or paravertebral (such as the sympathetic chain) ganglion, this preganglionic neuron joins the second (postganglionic) neuron. To reach its objectives in the kidney, the postganglionic neuron follows the renal vasculature. The rostral ventrolateral medulla (RVLM) of the brainstem and the paraentricular nucleus (PVN) of the hypothalamus provide descending excitatory input to the IML, which is the primary source of sympathetic nerve activity to the kidney [1].

1. Adrenergic Transmission

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Sympathetic input to the juxtaglomerular cells triggers the generation and secretion of renin via the β1 adrenoceptor, which raises the plasma levels of angiotensin II and aldosterone M in the brainstem and the paraentricular nucleus (PVN) of the hypothalamus. NE acts on α1 receptors in the afferent and efferent arterioles to constrict vascular smooth muscle, decreasing GFR and increasing renal vascular resistance due to the afferent arteriole's significantly increased innervation density [20].

Through α1 adrenoceptor-mediated activation of the Na+/H+ antiporter, sympathetic input to the proximal tubule increases the reabsorption of water, chloride, and bicarbonate, promotes the reabsorption of sodium, and increases the acidity of urine [21]. By activating the Na+/H+ antiporter through the α1 adrenoceptor, sympathetic input to the proximal tubule promotes sodium reabsorption and makes it easier for urine to become more acidic and for water, chloride, and bicarbonate to be reabsorbed [22].

2. S ignaling through Purines

Certain renal sympathetic neurons also emit ATP, NPY, and VIP in addition to NE. Alongside NE, ATP effects at juxtaglomerular cells may contribute to an increase in renin release [23]. After ATP is converted to adenosine, afferent arteriole vasoconstriction is caused by ATP-induced P2X receptor activation [24]. Adenosine A2 receptors are present in efferent arterioles, but not A1 receptors [25], suggesting that their primary response to adenosine is vasodilation. Therefore, the regulation of GFR by P2X1-mediated vasoconstriction in response to ATP release is the main purinergic effect on arterioles. Lastly, in response to ATP release, the P2X and P2Y receptors in the collecting ducts mediate an increase in the transmembrane transfer of water and salt [26].

3. Peptidergic Signaling

The majority of sympathetic renal nerves contain NPY. Vasoconstriction through Y1 receptors is NPY's most significant impact on renal function [6]. There have also been reports of VIP and TH immunohistochemical staining colocalizing in rat kidney nerve fibers. VIP-positive are distributed equally in the kidneys of humans and monkeys, and their densities are generally lower than those of NPY-positive fibers. Other species have not consistently been observed to have renal fibers that are VIP-positive [27]. VIP release can enhance renal resistance by vascular constriction and an increase in fractional sodium excretion. However, it has also been shown that male human VIP infusion results in vasodilation and a drop in blood pressure without having any appreciable effect on GFR. Additionally, it has been shown that juxtaglomerular cells produce more renin after VIP superfusion of isolated [28].

C. Differential Sympathetic Regulation of Renal Effector Sites

In unanesthetized animals, two labs have investigated the connection between renal vascular resistance and ERNA: Barrett and associates [29], who continuously measured, arterial pressure, renal vascular resistance, and ERNA in conscious rabbits discovered that while there was no significant relationship between basal levels of ERNA and renal vascular resistance at rest, short episodic bursts of ERNA did cause renal vasoconstriction. According to the hierarchy put forth by DiBona and Kopp, renal vascular resistance is only increased by extremely high ERNA levels [30].

D. Sympathetic Renal Nerves

Explains how the renal vasculature is innervated by the sympathetic nerves. The sympathetic renal nerve fibers in the kidney emit norepinephrine the effects of NE on adrenoceptors have the greatest influence on renal function [7-9, 31].

Throughout Sympathetic fiber varicosities close to their effector make up the kidney's neuroeffector junctions in the peripheral neural system [10, 11]. Instead of conventional bouton connections at the termination. As a fiber passes by these enpassant junctions, numerous structures can be functionally innervated [32, 33] either through capillary transport or diffusion in the renal interstitium [34].

The renal artery and its offshoot vascular branches carry sympathetic renal nerves to the afferent and efferent arterioles in the renal cortex. In the afferent arteriole, TH+ adrenergic sympathetic fibers also innervate juxtaglomerular renin-secreting granular cells [13, 14, 35].

Lastly, capillaries have a lower density than arterioles despite appearing to have sympathetic innervation [36] and it is unknown what the functional relevance is. The vasculature connecting the superficial and deep nephrons seems to have the same sympathetic innervation [37].

1. Sympathetic Renal Tubular Structure Innervation

Although not as thickly as the vasculature, sympathetic fibers innervate some tubular structures [15, 38].

2. Renal Pelvic Sympathetic Innervation

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According to NPY immunolabeling, the pelvic wall's sympathetic innervation is less numerous than the vasculature [16, 39, 40]. In the pelvis, sympathetic and sensory fibers distribute independently, albeit frequently traveling in bundles with sensory fibers [17, 18, 41]. At the renal pelvis, sympathetic fiber functions are most likely associated with modulating sensory fiber activity or contracting the pelvic smooth muscle [19, 20, 42].

3. Reactions of the Body to Variations in Sympathetic Renal Nerve Activity

Adrenergic transmission NE mediates the main renal reactions to increased sympathetic activity by attaching itself to α- and β-adrenergic receptors. In the afferent and efferent arterioles, NE constricts the vascular smooth muscle by acting on α1 receptors. Renal vascular resistance rises and GFR falls as a result of the afferent arteriole's significantly higher innervation density. Angiotensin II and aldosterone plasma levels are increased by sympathetic input to the juxtaglomerular cells via the β1 adrenoceptor, which stimulates renin production and release [21, 43, 44].

The proximal tubule receives sympathetic input that activates the Na+/H+ antiporter through α1 adrenoceptors, increasing water and bicarbonate reabsorption, stimulating sodium reabsorption, improving urine acidity and aldosterone levels [21, 45-47]. Similarly, enhanced sodium reabsorption occurs when NE binds to α1B receptors on the distal convoluted tubule epithelium [22, 23, 48].

Purine-based signaling. Certain renal sympathetic neurons also generate ATP, NPY, and VIP in addition to NE. In addition to NE, juxtaglomerular cells' ATP activity might also raise renin release [24, 49, 50]. Afferent arteriole vasoconstriction is caused by ATP-induced P2X receptor activation, and after ATP is converted to adenosine, further vasoconstriction is mediated by adenosine P1 receptors [25, 51, 52]. The afferent arteriole's adenosine A1 or A2 receptors' activation causes vasoconstriction or vasodilation, respectively [53, 54]. Adenosine A2 receptors are present in efferent arterioles, but not A1 receptors [55], indicating that vasodilation is their main reaction to adenosine. Consequently, regulating GFR by P2X1-mediated vasoconstriction in response to ATP release is the main purinergic effect on arterioles [56, 57]. Finally, increased salt and water transmembrane transfer is mediated in response to ATP release [26, 58, 59].

4. Sensory Renal Nerves

Finally, increased salt and water transmembrane transfer is mediated in response to ATP release [60-63]. Their use is confirmed by the fact that they do not exist in sympathetic neurons of the kidneys. However, it should be noted that these findings also found that at least 15% of kidney-innervating sensory neurons did not express this peptide. Furthermore, studies have demonstrated the presence of myelinated renal afferent fibers [64-67].

Conclusion

The intricate sympathetic innervation of renal structures plays a critical role in modulating renal function through complex neuroeffector interactions involving neurotransmitters such as norepinephrine, ATP, and neuropeptides. The modulation of vascular resistance, renin secretion, sodium reabsorption, and urinary composition underscores the significance of renal sympathetic nerves in maintaining homeostasis and contributing to pathological states such as hypertension. The therapeutic effects observed with catheter-based renal nerve ablation (CBRNA), including reduced sympathetic activity, improved glucose metabolism, and decreased cardiac arrhythmias, highlight the potential clinical relevance of targeting renal innervation. However, the mechanisms by which renal sensory (afferent) neurons interact with central and peripheral sympathetic activity remain incompletely understood. Future research should focus on delineating the precise pathways involved in renal sympathetic signaling, exploring the differential impact of afferent and efferent innervation, and assessing long-term outcomes of renal denervation therapies in diverse patient populations. Advancing our understanding of renal neurobiology could lead to novel therapeutic interventions for hypertension, metabolic disorders, and renal dysfunction, ultimately improving clinical management strategies and patient outcomes.

References

1. J. W. Osborn and J. D. Foss, "Renal nerves and long-term control of arterial pressure," Comprehensive physiology, vol. 7, pp. 263-320, 2011.
2. H. H. Abdul-Ra’aoof, S. B. Dawood, F. A. Jassim, S. K. Jassim, S. S. Issa, A. M. Tiryag, et al., "Moderate proficiency in suture techniques among nurses: A cross-sectional study," Academia Open, vol. 9, 2024.
3. M. P. Schlaich, P. A. Sobotka, H. Krum, R. Whitbourn, A. Walton, and M. D. Esler, "Renal denervation as a therapeutic approach for hypertension: novel implications for an old concept," Hypertension, vol. 54, pp. 1195-1201, 2009.
4. M. F. Hasan, W. F. Hussein, A. M. Tiryag, I. J. Ali, and Z. M. Shaker, "Nurses’ Knowledge Toward Lower Back Pain: A Cross-Sectional Study," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 10363-10.21070/acopen. 9.2024. 10363, 2024.
5. G. E. Knight, R. Oliver-Redgate, and G. Burnstock, "Unusual absence of endothelium-dependent or-

independent vasodilatation to purines or pyrimidines in the rat renal artery," Kidney international, vol. 64, pp. 1389-1397, 2003.
6. A. Bischoff and M. Michel, "Renal effects of neuropeptide Y," Pflügers Archiv, vol. 435, pp. 443-453, 1998.
7. L. Barajas, K. Powers, and P. Wang, "Innervation of the renal cortical tubules: a quantitative study," American Journal of Physiology-Renal Physiology, vol. 247, pp. F50-F60, 1984.
8. S. Luff, S. Hengstberger, E. McLachlan, and W. Anderson, "Two types of sympathetic axon innervating the juxtaglomerular arterioles of the rabbit and rat kidney differ structurally from those supplying other arteries," Journal of Neurocytology, vol. 20, pp. 781-795, 1991.
9. S. E. Luff, S. G. Hengstberger, E. M. McLachlan, and W. Anderson, "Distribution of sympathetic neuroeffector junctions in the juxtaglomerular region of the rabbit kidney," Journal of the autonomic nervous system, vol. 40, pp. 239-253, 1992.
10. G. Burnstock, "Autonomic neuromuscular junctions: current developments and future directions," Journal of anatomy, vol. 146, p. 1, 1986.
11. G. Burnstock, "The autonomic neuroeffector junction," in Primer on the autonomic nervous system, ed: Elsevier, 2004, pp. 29-33.
12. G. F. Dibona and U. C. Kopp, "Neural control of renal function," Physiological reviews, vol. 77, pp. 75-197, 1997.
13. C. Chan, R. Unwin, M. Bardini, I. Oglesby, A. Ford, A. Townsend-Nicholson, et al., "Localization of P2X1 purinoceptors by autoradiography and immunohistochemistry in rat kidneys," American Journal of Physiology-Renal Physiology, vol. 274, pp. F799-F804, 1998.
14. G. Burnstock and A. Loesch, "Sympathetic innervation of the kidney in health and disease: Emphasis on the role of purinergic cotransmission," Autonomic Neuroscience, vol. 204, pp. 4-16, 2017.
15. L. Barajas, L. Liu, and K. Powers, "Anatomy of the renal innervation: intrarenal aspects and ganglia of origin," Canadian journal of physiology and pharmacology, vol. 70, pp. 735-749, 1992.
16. L. Liu and L. Barajas, "The rat renal nerves during development," Anatomy and embryology, vol. 188, pp. 345-361, 1993.
17. U. C. Kopp, M. Z. Cicha, L. A. Smith, J. Mulder, and T. Hokfelt, "Renal sympathetic nerve activity modulates afferent renal nerve activity by PGE2-dependent activation of α1-and α2-adrenoceptors on renal sensory nerve fibers," American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, vol. 293, pp. R1561-R1572, 2007.
18. U. C. Kopp, M. Z. Cicha, L. A. Smith, S. Ruohonen, M. Scheinin, N. Fritz, et al., "Dietary sodium modulates the interaction between efferent and afferent renal nerve activity by altering activation of α2-adrenoceptors on renal sensory nerves," American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, vol. 300, pp. R298-R310, 2011.
19. M. Merrick and T. Griffin, "Evidence for a reflex provoking contraction of the renal pelvis (with some comments on its clinical implications)," European journal of nuclear medicine, vol. 21, pp. 521-524, 1994.
20. A. Nakamura and E. J. Johns, "Effect of renal nerves on expression of renin and angiotensinogen genes in rat kidneys," American Journal of Physiology-Endocrinology and Metabolism, vol. 266, pp. E230-E241, 1994.
21. Y. Chan, "Adrenergic control of bicarbonate absorption in the proximal convoluted tubule of the rat kidney," Pflügers Archiv, vol. 388, pp. 159-164, 1980.
22. G. F. DiBona and L. L. Sawin, "Effect of renal nerve stimulation on NaCl and H2O transport in Henle's loop of the rat," American Journal of Physiology-Renal Physiology, vol. 243, pp. F576-F580, 1982.
23. K. Gaál, I. Forgács, and Z. Bácsalmásy, "Effect of adenosine compounds (ATP, cAMP) on renin release in vitro," Acta Physiologica Academiae Scientiarum Hungaricae, vol. 47, pp. 49-54, 1976.
24. E. W. Inscho, P. K. Carmines, and L. G. Navar, "Juxtamedullary afferent arteriolar responses to P1 and P2 purinergic stimulation," Hypertension, vol. 17, pp. 1033-1037, 1991.
25. P. Hansen, U. G. Friis, T. R. Uhrenholt, J. Briggs, and J. Schnermann, "Intracellular signalling pathways in the vasoconstrictor response of mouse afferent arterioles to adenosine," Acta physiologica, vol. 191, pp. 89-97, 2007.
26. E. M. Schwiebert and B. K. Kishore, "Extracellular nucleotide signaling along the renal epithelium," American Journal of Physiology-Renal Physiology, vol. 280, pp. F945-F963, 2001.
27. D. Knight, R. Fabre, and J. Beal, "Identification of noradrenergic nerve terminals immunoreactive for neuropeptide Y and vasoactive intestinal peptide in the rat kidney," American journal of anatomy, vol. 184, pp. 190-204, 1989.
28. J. Calam, R. Dimaline, W. Peart, J. Singh, and R. Unwin, "Effects of vasoactive intestinal polypeptide on renal function in man," The Journal of Physiology, vol. 345, pp. 469-475, 1983.
29. C. J. Barrett, M. A. Navakatikyan, and S. C. Malpas, "Long-term control of renal blood flow: what is the role of the renal nerves?," American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, vol. 280, pp. R1534-R1545, 2001.
30. M. Yoshimoto, T. Sakagami, S. Nagura, and K. Miki, "Relationship between renal sympathetic nerve activity and renal blood flow during natural behavior in rats," American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, vol. 286, pp. R881-R887, 2004.
31. F. A. Jassim, A. M. Tiryag, and S. S. Issa, "Effect of Bad Habits on the Growth of School Students: A Cross-Sectional Study," Indonesian Journal on Health Science and Medicine, vol. 1, pp. 10-21070, 2024.
32. U. C. Kopp, "Neural control of renal function," 2018.
33. S. K. Jassim, Z. Abbass, and A. M. Tiryag, "A Study of Diabetes Correlated Emotional Distress among Patients with Type 2 Diabetes Mellitus: A cross Sectional Study," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 10292-10.21070/acopen. 9.2024. 10292, 2024.

ISSN 2714-7444 (online), https://acopen.umsida.ac.id, published by Universitas Muhammadiyah Sidoarjo

34. A. Tiryag, M. Atiyah, and A. Khudhair, "Nurses' Knowledge and Attitudes toward Thyroidectomy: A Cross-Sectional Study," Health Education and Health Promotion, vol. 10, pp. 459-465, 2022.
35. Z. Abbass, S. K. Jassim, A.-F. Sadeq, S. Hafedh, A. M. Tiryag, and H. H. A. AL-Hadrawi, "Determination of Self-Efficacy Level: The Capacity of Patients with Hypertension to Manage their Chronic Disease," Indonesian Journal on Health Science and Medicine, vol. 1, pp. 10.21070/ijhsm. v1i2. 15-10.21070/ijhsm. v1i2. 15, 2024.
36. A. M. Tiryag and H. H. Atiyah, "Nurses’ Knowledge toward Bariatric Surgery at Surgical Wards at Teaching Hospitals in Al-Basra City," Indian Journal of Forensic Medicine & Toxicology, vol. 15, pp. 5152-5159, 2021.
37. A. M. Tiryag, "Nurses’ Knowledge and Attitudes Toward Pacemaker: A Cross-Sectional Study," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 8845-10.21070/acopen. 9.2024. 8845, 2024.
38. M. A. Mohammad, F. A. Jassim, and A. M. Tiryag, "Single-use flexible ureteroscope for the treatment of renal stone," Revista Latinoamericana de Hipertension, vol. 18, 2023.
39. M. Mohammad, F. Jassim, and A. Tiryag, "Retrograde Intrarenal Lithotripsy Using Disposable Flexible Ureteroscope," Georgian Medical News, vol. 348, pp. 44-46, 2024.
40. H. H. Abdul-Ra’aoof, A. M. Tiryag, and M. A. Atiyah, "Knowledge, attitudes, and practice of nursing students about insulin therapy: A cross-sectional study," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 8795-10.21070/acopen. 9.2024. 8795, 2024.
41. A. A. A. Al-Iedan, M. A. Akber, S. B. Dawood, A. I. H. Alobaidi, S. S. Issa, H. H. A. Raaoof, et al., "Bridging the Gap: Enhancing Open Fracture Care in Emergency Nursing," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 8847-10.21070/acopen. 9.2024. 8847, 2024.
42. M. A. Mohammad, A. Y. Al-Timary, and A. M. Tiryag, "Safety of Tubeless Double Access Percutaneous Nephrolithotomy Compared to Single Access Approach," Bahrain Medical Bulletin, vol. 45, 2023.
43. H. Kobayashi and Y. Takei, The renin-angiotensin system: comparative aspects vol. 35: Springer Science & Business Media, 2012.
44. J. L. Osborn, R. J. Roman, and J. D. Ewens, "Renal nerves and the development of Dahl salt-sensitive hypertension," Hypertension, vol. 11, pp. 523-528, 1988.
45. Z. S. Dawood, K. M. Jassim, A. M. Tiryag, and A. S. Khudhair, "Nurses' Knowledge and Attitudes Toward Deep Vein Thrombosis: A Cross-Sectional Study," Bahrain Medical Bulletin, vol. 45, 2023.
46. M. G. Cogan, "Neurogenic regulation of proximal bicarbonate and chloride reabsorption," American Journal of Physiology-Renal Physiology, vol. 250, pp. F22-F26, 1986.
47. G. F. Dibona, "Neural control of the kidney: functionally specific renal sympathetic nerve fibers," American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, vol. 279, pp. R1517-R1524, 2000.
48. M. Jabbar, M. Mohammad, and A. Tiryag, "CHANGES IN MALE REPRODUCTIVE HORMONES IN PATIENTS WITH COVID-19," Georgian Medical News, pp. 42-46, 2023.
49. M. A. Akber, A. M. Tiryag, and A. Alobaidi, "Nurses’ Knowledge Concerning Developmental Dysplasia of the Hip: A Cross-Sectional Study," American Journal of Pediatric Medicine and Health Sciences, vol. 2, pp. 155-160, 2024.
50. M. A. Mohammad, H. H. Abdul-Ra’aoof, K. A. Razzaq Manahi, and A. M. Tiryag, "Parents’ Knowledge and Attitudes toward Testicular Torsion," Bahrain Medical Bulletin, vol. 46, 2024.
51. H. Weihprecht, J. N. Lorenz, J. P. Briggs, and J. Schnermann, "Vasomotor effects of purinergic agonists in isolated rabbit afferent arterioles," American Journal of Physiology-Renal Physiology, vol. 263, pp. F1026-F1033, 1992.
52. W. D. A. Ali, S. S. Hamid, M. Sabah, Z. M. H. Al-Hijaj, S. Baker, and M. A. Atiyah, "Critical Knowledge Gaps in Iraqi Nurses' Understanding of Antihypertensive Drug Risks," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 9284-10.21070/acopen. 9.2024. 9284, 2024.
53. E. Inscho, A. Cook, J. Imig, C. Vial, and R. Evans, "Renal autoregulation in P2X1 knockout mice," Acta physiologica scandinavica, vol. 181, pp. 445-453, 2004.
54. A. M. Tiryag, S. B. Dawood, and S. K. Jassim, "Nurses’ knowledge and attitudes about enteral feeding complications by nasogastric tube in intensive care units," Rawal Medical Journal, vol. 48, pp. 689-689, 2023.
55. A. M. Tiryag and H. H. Atiyah, "Nurses' knowledge toward obesity in al-Basra city," Annals of the Romanian Society for Cell Biology, pp. 4667-4673, 2021.
56. M. A. Bailey, R. J. Unwin, and D. G. Shirley, "P2X receptors and kidney function," Wiley Interdisciplinary Reviews: Membrane Transport and Signaling, vol. 1, pp. 503-511, 2012.
57. M. A. Akber, A. M. Tiryag, and A. I. H. Alobaidi, "Nurses’ Knowledge Regarding Cast Complications of Limb Fractures: A Cross-Sectional Study," Central Asian Journal of Medical and Natural Science, vol. 5, pp. 195-200, 2024.
58. M. Burg, D. S. Zahm, and M. M. Knuepfer, "Immunocytochemical co-localization of substance P and calcitonin gene-related peptide in afferent renal nerve soma of the rat," Neuroscience letters, vol. 173, pp. 87-93, 1994.
59. H. M. Sabty, S. B. Dawood, and A. M. Tiryag, "Nurses’ Knowledge and Practices on Influenza Vaccination for Pregnant Women," Jurnal Kebidanan Midwiferia, vol. 10, pp. 50-59, 2024.
60. D. C. Kuo, J. J. Oravitz, R. Eskay, and W. C. De Groat, "Substance P in renal afferent perikarya identified by retrograde transport of fluorescent dye," Brain research, vol. 323, pp. 168-171, 1984.
61. I. Zainel, H. Abdul-Ra'aoof, and A. Tiryag, "Mothers' Knowledge and Attitudes towards her Children with Neonatal Jaundice: A Cross-Sectional Study," Health Education and Health Promotion, vol. 10, pp. 565-570, 2022.

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62. M. M. Kneupfer and L. P. Schramm, "The conduction velocities and spinal projections of single renal afferent fibers in the rat," Brain research, vol. 435, pp. 167-173, 1987.
63. E. H. Rahi, Z. M. H. Al-Hejaj, and A. M. Tiryag, "Nurses' knowledge of nonalcoholic fatty liver disease: A cross-sectional study," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 10306-10.21070/acopen. 9.2024. 10306, 2024.
64. O. Simon and L. Schramm, "Spinal superfusion of dopamine excites renal sympathetic nerve activity," Neuropharmacology, vol. 22, pp. 287-293, 1983.
65. A. Stella and A. Zanchetti, "Functional role of renal afferents," Physiological reviews, vol. 71, pp. 659-682, 1991.
66. A. M. Tiryag, "Revitalizing Hearts: The Transformative Impact of Pacemaker Therapy on Cardiac Conduction Disorders," Academia Open, vol. 9, pp. 10.21070/acopen. 9.2024. 8845-10.21070/acopen. 9.2024. 8845, 2024.
67. H. H. Abdul-Ra’aoof, M. A. Akber, F. A. Jassim, A. M. Tiryag, S. S. Issa, M. A. Atiyah, et al., "The Psychological Impact of Violence on Emergency Department and Intensive Care Unit Nurses: A Cross-Sectional Study," Research Journal of Trauma and Disability Studies, vol. 3, pp. 228-233, 2024.

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