应激与颞下颌关节紊乱病研究新进展

孟 媛 石一涵 黄 飞 刘 丽 王红雷

(解放军第 451 医院口腔科 陕西 西安 710054)

摘要:颞下颌关节紊乱病是慢性面部疼痛最常见的诱因,常常与躯体和心理主诉症状密切联系,包括疲劳,睡眠失调,焦虑和抑郁等。即使未发现任何能够合理解释疼痛原因的时候,健康专业人士也常常忽略疼痛感受的主观性。从严格的生物医学角度来讲,对疼痛的这种理解是不科学的。本文的主要目的是通过查阅近年来大量的研究文献资料,发现应激引起疼痛感觉的生物学途径以及导致颞下颌关节紊乱的原因。研究发现下丘脑 - 垂体 - 肾上腺轴、5- 羟色胺和阿片样物质通路都与颌面部疼痛的发病密切相关,同时也提出了未来可能使用的治疗方法。同时,也希望本文能把与疼痛学科差别较大的口腔医学融入到需要多学科合作的颞下颌关节紊乱的诊断和治疗中,从科学角度提高对该病的临床诊疗效率。

关键词:应激;疼痛;颞下颌关节紊乱;伤害感受;颞下颌关节

中图分类号:R78,R782.13 **文献标识码**:A **文章编号**:1673-6273(2011)12-2391-04

Current Status and Perspectives of Researches between Stress and the Temporomandibular Disorders

MENG Yuan, SHI Yi-han, HUANG Fei, LIU Li, WANG Hong-lei

(Department of Orthodontics, PLA 451 Hospital, Shanxi, XiAn 710054)

ABSTRACT: Temporomandibular disorders (TMD) comprise the most common cause of chronic facial pain conditions, and they are often associated with somatic and psychological complaints including fatigue, sleep disturbances, anxiety, and depression. For many health professionals, the subjectivity of pain experience is frequently neglected even when the clinic does not find any plausible biologic explanation for the pain. This strictly biomedical vision of pain cannot be justified scientifically. The purpose of this study is to demonstrate, by original articles from the literature and recent studies conducted in our own laboratory, the biological processes by which psychological stress can be translated into the sensation of pain and contribute to the development of TMD. The role of the hypothalamic-pituitary-adrenal axis, the serotoninergic and opioid systems in the pathogenesis of facial pain is exposed, including possible future therapeutic approaches. It is hoped that knowledge from apparently disparate fields of dentistry, integrated into a multidisciplinary clinical approach to TMD, will improve diagnosis and treatment for this condition through a clinical practice supported by scientific knowledge.

Key words: Pain; Temporomandibular disorders; Nociception; Temporomandibular joint

Chinese library classificationr: R78, R782.13 Document code: A

Article ID:1673-6273(2011)12-2391-04

颞下颌关节紊乱是一种以颞下颌关节疼痛和/或咀嚼肌疼痛为特征的骨骼肌疼痛疾病[1],同时它也包含下颌运动弹响以及下颌运动受限[2]。颞下颌关节疼痛是颞下颌关节紊乱患者被迫来就诊的最常见主诉症状。颞下颌关节紊乱的各种体征和症状的发病率从 6%到 93%不等,但只有 3.6%到 7%的患者需要进行治疗[3-7],导致流行率变化幅度这么大的原因可能是由于使用不同的诊断标准方法论导致的。尽管目前我们还不能完全掌握颞下颌关节紊乱的准确诱因,但国际公认它是多因素诱因导致的,其中包括心理,行为和环境因素。在口腔研究中,咬合因素和功能紊乱是流行病学研究中被公认的两大诱因[8,9]。同样,咬合紊乱、下颌位置和生物力学因素也同样被列为可能诱因。例如,很多研究并未发现咬合与颞下颌关节紊乱之间的联系[10-12]。即使出现了相关性,一些研究也仅仅发现其与关节紊乱发病联系不是很紧密[13,14]。在过去 20 年中,关于颞下颌关节紊乱的病因学研究也发现,后退接触位和牙尖交错位之间的侧向合力以及单侧反合导致的侧向合力与其他局部诱因一样与颞下颌关节紊乱的发生发展都密切相关[13]。一些实验研究发现,通过实验诱导口腔功能紊乱可以导致颌面部疼痛,这与临床颞下颌关节紊乱患者症状相似[15,16]。尽管异常的磨牙癖也包括咀嚼肌活性增加[15]常常也会引起疼痛[17,18],但目前认为磨牙症与颞下颌关节紊乱的疼痛不相关[19]。此外,研究也发现被诊断为磨牙症的患者也常常不表现出咀嚼肌疼痛[20,21]。因此,目前还没有任何研究结果能够证明功能紊乱与颞下颌关节紊乱直接相关。

另一方面,Laskin [22]等首次提出情绪因素可能是比躯体因素更重要的颞下颌关节紊乱病的诱因。最近 10 年中有大量的文献证实了心理应激在颞下颌关节紊乱疾病发病中的作用[23-25]。颞下颌关节紊乱病的患者常常主诉他们的疼痛感觉与应激强度正相关[26]。De Leeuw 等[27]研究发现肌肉功能紊乱以及伴随的疼痛常常是肌肉功能亢进的结果,应激导致的肌肉功能紊乱常常会继发性的引起颞下颌关节改变 升颌肌群肌肉强直导致颞下颌关节关节内压力增加,引起正常生物力学发生改变,最终引起关节囊和盘附着损伤。但是,有些研究也出现了相互抵触的结果,如有研究[28-30]指出,颞下颌关节紊乱的患者的肌电图基础阈值高于正常人群,但其他研究[31]却发现了相反的结果,提示两者之间无明显差异,其可能是研究使用的方法不同造成的。

本文的目的是演示应激影响疼痛感受和颞下颌紊乱疾病发展的生物学进程,文中包括应激系统的生理学和病理生理学特征,并提出了未来可能采用的治疗方法。

作者简介:孟媛(1980-),女,住院医师,医学学士,

电话:18992891016,13379066868,E-MAIL:443885860@qq.com

(收稿日期:2011-02-23 接受日期:2011-03-18)

1 应激系统 -- 生理学机制

生命，作为一个高级动态平衡个体，每时每刻都不断受到外界的刺激，如应激，这些刺激都不断的机体内部的调节最终达到体内平衡[32,33]。神经通路主要维持躯体植物神经功能，如吃喝和繁殖；外周调节功能主要作用于能量的再分配，如氧气和能量直接转送至中枢神经系统和应激部位[34]。

不是所有应激因素都是对躯体有害的。Selye[35]通过比较 " 正性应激 " 和 " 副性应激 " 后提出，适度、短暂和可控的应激状态可以被躯体作为一种愉快或者兴奋的感受，可以积极促进情感和智力的发育，他同时也指出严重的、不可控的心理和躯体应激状态即 " 副性应激 " 可以间接导致疾病的发生和发展。

应激系统的中枢部分位于下丘脑和脑干部分，包括促肾上腺皮质激素和去甲肾上腺素的释放以及自主神经系统[36]，其外周部分主要由下丘脑 - 垂体 - 肾上腺素轴，传出交感 / 肾上腺髓质系统和副交感系统组成[32]，中枢释放的促肾上腺皮质激素和去甲肾上腺素系统，以及外周释放的大量的糖皮质激素和儿茶酚胺几乎可以影响到躯体的每一个细胞[37]。此外，应激系统也与其他主要的中枢神经系统相互作用，如多巴胺能系统，杏仁核，海马和促黑激素神经系统的弓状核[37]。脑中各种神经递质系统之间的相互作用是应激后机体各系统应答的基础，其中包括行为、内分泌、内脏、自主和免疫应答。这些神经递质包括促肾上腺皮质激素、精氨酸后叶加压素、类鸦片类物质、P 物质、多巴胺、5- 羟色胺和去甲肾上腺素。值得注意的是，绝大多数与应激相关的神经递质和疼痛调节递质都是相同的，因此应激与疼痛感受的相关性是明显的[38,39]。

2 丘脑 - 垂体 - 肾上腺素轴 病理学机制

研究已经证实，许多应激相关的精神病学疾病如抑郁[40]和创伤后应激性失调等，都与丘脑 - 垂体 - 肾上腺素轴的调节异常有关，这些疾病都与患有颞下颌关节紊乱的患者密切相关[41]。应激系统失调常常表现为技能亢进或者技能减退。例如，丘脑 - 垂体 - 肾上腺素轴功能亢进常常发生在忧郁症[42]、厌食症[43]、强迫症[44]、恐慌症[33]和慢性酒精中毒症[45]。另一方面，应激系统功能减退可以减低促肾上腺素激素的分泌，常常发生于纤维肌痛[46]、周期性抑郁症[47]、非典型抑郁[48]、不同程度的肥胖症[42]和慢性疲劳综合征[49]。在颞下颌关节紊乱患者中，应激系统常常表现为功能亢进。Geissler[50]等使用生化方法证实患有颞下颌关节紊乱的患者尿中的皮质醇含量要高于正常个体，因此提示他们处于更大的应激状态。但最近的一些研究[51]也发现，面部疼痛患者白天表现出很高的皮质醇含量，其含量却高于患有常见的纤维肌痛或者处于抑郁状态的疼痛患者，他们分析其可能原因是面部区域疼痛对丘脑 - 垂体 - 肾上腺素轴的刺激要大于躯体其他部位的疼痛[52]。

鉴于疼痛本身作为一种丘脑 - 垂体 - 肾上腺素轴被激活的标志，那么疼痛本身也可能是一种应激源，它常常与长期增加的促肾上腺皮质激素水平或其他丘脑 - 垂体 - 肾上腺素轴的其他介质水平相关联，最终表现为颞下颌关节紊乱患者对慢性应激长期表现出高水平的皮质醇含量，中枢应激系统的过度活化常常表现为疼痛敏感[53]。

由于方法论、心理学和伦理学方面的一些难题，使关于人类应激和疼痛调节之间机制问题的研究越来越困难。另一方面，随着动物疼痛模型发展的不断成熟，使研究疼痛感受机制中神经调节部分成为可能。Gameriro 等[54]使用动物模型评估应激对福尔马林诱导的颞下颌关节疼痛的影响，他们发现经过 2 个月的束缚应激后，大鼠对疼痛敏感度增强，提示慢性应激诱导了疼痛敏感[55]。目前关于慢性应激诱导疼痛敏感的机制尚不清楚，丘脑 - 垂体 - 肾上腺素轴只是应激系统生物学机制中的一部分。

3 5- 羟色胺系统

在疼痛感受中上调伤害性通路的神经元常常受到下行 5- 羟色胺和去甲肾上腺素纤维的抑制[56,57]。在不同的应激条件下，中枢 5- 羟色胺系统活性的改变在一定程度上可以解释应激产生的疼痛双向作用，即疼痛敏感和痛觉缺失。例如，在急性的不同种副性心理应激或者躯体应激后，可以发现在大脑的许多区域都可以发现 5- 羟色胺分泌增加，特别是脑脊区[58]。相反，持续应激可以减低大脑中应激诱导的 5- 羟色胺分泌量，这些区域包括杏仁核和侧隔膜[59]。研究也发现，在脊髓通路中疼痛传递的抑制幅度与机体的应激后行为状态有关 (如抑郁，焦虑和恐惧) [58]。Gameriro 等提出应激后的焦虑状态可以引起中枢 5- 羟色胺分泌的减，他们通过每天制动 1 个小时 (急性应激) 或者 2 个月 (慢性应激) 诱导应激状态 (通过放免法分析皮质醇和去甲肾上腺素确定) [59]，然后进行颞下颌关节伤害感受评估，发现应激可以导致疼痛敏感，检测前半个小时注射氟西汀可以产生和吗啡同等的止痛效果[60]，这与焦虑诱导疼痛敏感的临床研究结果相一致[61,62]。

Schreiber 等[63]发现氟西汀可以减轻后背疼痛感觉，这与阿米替林的效果相似，因此他们提议氟西汀可以作为那些不能耐受三环类抗抑郁药副作用的替代药品。同样也有临床研究证实氟西汀作为一种 5- 羟色胺再摄取抑制剂，可以有效治疗有磨牙症症状的颞下颌关节紊乱患者[64]。

4 阿片样物质的调节作用

在过去的十年里关于神经性疼痛机理的研究取得了很大的进步，目前已经明确了解疼痛不只是被神经系统被动接收，同样也通过复杂的调节机制中第一级感觉突触进行过滤和调控[65,62]。复杂的疼痛调节系统可以阐明不同疼痛临床表现的原因，这个复杂疼痛调节系统中的最重要的组成就是固有阿片样物质系统，这个系统可以在应激状态下激活以及降低疼痛感受[66]。Maixner 等[67]研究发现患有左侧臂缺血性疼痛的患者能够因为急性牙科疼痛刺激导致左侧壁疼痛感觉减轻。关于这方面研究面临的最大难题就是内源性抑制系统是否在慢性颌面部疼痛中发挥作用，有研究通过对颞下颌关节紊乱患者的调查发现，中枢神经系统中的抑制系统功能减弱[68,69]，其中也有研究发现，颞下颌关节紊乱患者心理应激和生理应激相关的生化指标与正常人群相比表达增高[50,51]。

Gameriro 等[55]研究发现在慢性应激条件下，与空白对照组相比，重复应激大鼠对吗啡的敏感性逐渐减弱，进而证实应激状态下内源性抑制系统功能减弱。这种对吗啡作用的耐受效应与先前研究中提出的关于慢性应激可以改变阿片样物质系统活性的假设相一致[70]。

5 应激是颞下颌关节紊乱发病的重要诱因

目前虽然已经确定了心理因素在颞下颌关节紊乱中发病的重要性，但是尚缺少病因学直接证据。尽管有很多研究表明

患有颞下颌关节紊乱的患者会表现出比正常个体更加焦虑或者抑郁的状态，但是尚无直接的人体实验研究证实二者之间的明确关系。有研究[71-74,61]可以证实焦虑、肌肉紧张和颞下颌关节紊乱症状的相关性。在一份关于成人颞下颌关节紊乱患者的调查中发现[75]，焦虑患者占 16.58%，抑郁患者占 26.71%。另一份研究[59]发现，39.8%的颞下颌关节紊乱患者有中到重度的抑郁表现，47.6%的患者在心理躯体化检测中发现其有着中到重度的非特异性躯体症状。

在颞下颌关节紊乱与年龄、性别和应激关系的研究中，Kuttilla 等[76]发现，女性表现出更多的颞下颌关节紊乱的症状，提示她们可能出于更高的应激状态，同时也提出女性发病高的原因可能是由于其在相同应激条件下女性比男性的激素分泌更多造成的。

目前用于研究生理性和心理性疼痛之间关系的研究主要包括心理学、认知学、行为学和生物学这四个方面，其中心理学方面是指无法调节和表达的心理矛盾（如恐惧和内疚），认知学方面是指无助和无法控制，行为学方面是指为了逃避压力大的工作或者欠债导致的严重的行为减少，生物学方面是指关于疼痛和应激调节中共同的其关键作用的神经递质[55,77,35,78]。这里我们强调生物学诱因的主要目的是为了更全面的为颞下颌关节紊乱紊乱的病因学机制提供一个完整性的生物 - 心理学概念。

用于应激和颞下颌关节疼痛的二者之间联系的科学研究方法应该包括动物实验和临床研究 2 个部分。但目前为止，只有少数研究从动物实验研究方面进行探讨二者之间的联系。在临床领域中，心理神经免疫学研究已经证实了心理应激与颞下颌关节紊乱发病之间的相关性[79-82]。尽管目前还没有完全明确心理应激在颞下颌关节紊乱发病中的明确机制，但是其二者之间的相关性已经得到广泛公认。关于颞下颌关节紊乱的诊断、评估和治疗必须包括生理因素（如颞下颌关节，咬合和肌肉）和心理因素（如个性、情感状态等）。

6 总结

关于颞下颌关节紊乱和心理应激之间的关系不仅仅是 Laskin 等[68,82]提出的那样，应激引起慢性反复的肌肉活性增强进而损伤关节，出现紊乱症状，而是应激状态可以直接影响到疼痛传递和感受通路，造成机体异常的反射进而形成一个持续的 " 恶性 " 循环，出现各种疼痛症状。同时，伤害性感受调节作用不仅仅出现在刺激强度较大的情况下，其实轻度的应激状态下其同样发挥着重要的作用。关于应激状态导致机体功能紊乱调节异常的理论研究也会为颞下颌关节紊乱发病机理研究提供一个新的方向，为其诊断和治疗方法提供理论依据。

参 考 文 献(References)

[1] LeResche L, Mancl L, Sherman JJ, et al. Changes in temporomandibular pain and other symptoms across the menstrual cycle [J]. Pain, 2003, 106:253-261

[2] Miller VJ, Karic VV, Myers SL, , et al. Following treatment of myogenous TMD patients with the temporomandibular opening index: an initial report[J]. J Oral Rehabil, 2003, 30: 668-670

[3] Carlsson GE. Epidemiology and treatment need for temporomandibular disorders[J]. J Orofac Pain, 1999, 13:232-237

[4] Dworkin SF, Huggins KH, LeResche L, et al. Epidemiology of signs and symptoms in temporomandibular disorders: clinical signs in cases and controls[J]. J Am Dent Assoc, 1990, 120:273-281

[5] LeResche L, Mancl L, Sherman JJ, et al. Changes in temporomandibular pain and other symptoms across the menstrual cycle [J]. Pain, 2003, 106:253-261

[6] Macfarlane TV, Blinkhorn AS, Davies RM, et al. Oro-facial pain in the community: prevalence and associated impact [J]. Community Dent Oral Epidemiol, 2002, 30:52-60

[7] Okeson JP. In: Okeson JP (ed) Orofacial pain, guidelines for assessment, diagnosis, and management. Quintessence Publishing, Carol Stream, Illinois, 1996

[8] Magnusson T, Egermark I, Carlsson GE. A longitudinal epidemiologic study of signs and symptoms of temporomandibular disorders from 15 to 35 years of age[J]. J Orofac Pain, 2000, 14:310-319

[9] Thilander B, Rubio G, Pena L, et al. Prevalence of temporomandibular dysfunction and its association with malocclusion in children and adolescents: an epidemiologic studyrelated to specified stages of dental development[J]. Angle Orthod, 2002, 72:146-154

[10] Clark GT, Adler RC. A critical evaluation of occlusal therapy: occlusal adjustment procedures [J]. J Am Dent Assoc, 1985, 110: 743-750

[11] Koh H, Robinson PG. Occlusal adjustment for treating and preventing temporomandibular joint disorders [J]. J Oral Rehabil, 2004, 31: 287-292

[12] Seligman DA, Pullinger AG. The role of functional occlusal relationships in temporomandibular disorders: a review [J]. J Craniomandib Disord, 1991, 5:265-279

[13] Magnusson T, Egermarki I, Carlsson GE. A prospective investigation over two decades on signs and symptoms of temporomandibular disorders and associated variables [J]. A final summary. Acta Odontol Scand, 2005, 63:99-109

[14] Mohlin BO, Derweduwen K, Pilley R, et al. Malocclusion and temporomandibular disorder: a comparison of adolescents with moderate to severe dysfunction with those without signs and symptoms of temporomandibular disorder and their further development to 30 years of age[J]. Angle Orthod, 2004, 74:319-327

[15] Glaros AG, Burton E. Parafunctional clenching, pain, and effort in temporomandibular disorders[J]. J Behav Med, 2004, 27:91-100

[16] Christensen L. Some effects of experimental hyperactivity of the mandibular locomotor system in man [J]. J Oral Rehabil, 1975, 2: 169-178

[17] Moss RA, Ruff MH, Sturgis ET. Oral behavioural patterns in facial pain, headache and non-headache populations [J]. Behav Res Ther, 1984, 22:683-687

[18] Ahlberg K, Ahlberg J, Kononen M, et al. Perceived orofacial pain and its associations with reported bruxism and insomnia symptoms in media personnel with or without irregular shift work [J]. Acta Odontol Scand, 2005, 63:213-217

[19] Pergamalian A, Rudy TE, Zaki HS, et al. The association between wear facets, bruxism, and severity of facial pain in patients with temporomandibular disorder[J]. J Prosthet Dent, 2003, 90:194-200

[20] Fujii T, Torisu T, Nakamura S (2005) A change of occlusal conditions after splint therapy for bruxers with and without pain in the masticatory muscles[J]. Cranio 23:113-118

[21] Lavigne GJ, Kato T, Kolta A, et al. Neurobiological mechanisms involved in sleep bruxism[J]. Crit Rev Oral Biol Med, 2003, 14:30-46

[22] Laskin DM. Etiology of the pain-dysfunction syndrome. J Am Dent Assoc, 1969, 79:147-153

[23] Grzesiak RC. Psychologic consideration in temporomandibular dysfunction. A biopsychosocial view of symptom formation[J]. Dent Clin North Am, 1991, 35:209-226

[24] Vanderas AP. Relationship between craniomandibular dysfunction and malocclusion in white children with and without unpleasant life events[J]. J Oral Rehabil, 1994, 21:177-183

[25] Wexler GB, Steed PA. Psychological factors and temporomandibular outcomes[J]. Cranio, 1998, 16:72-77

[26] Suvinen TI, Hanes KR, Gerschman JA, et al. Psychophysical subtypes of temporomandibular disorders [J]. J Orofac Pain, 1997, 11: 200-205

[27] De Leeuw JR, Steenks MH, Ros WJ, et al. Multidimensional evaluation of craniomandibular dysfunction. I: symptoms and correlates[J]. J Oral Rehabil, 1994, 1:501-514

[28] Kapel L, Glaros AG, McGlynn FD. Psychophysiological responses to stress in patients with myofascial pain-dysfunction syndrome[J]. J Behav Med, 1989, 12:397-406

[29] Mercuri LG, Olson RE, Laskin DM. The specificity of response to experimental stress in patients with myofascial pain dysfunction syndrome[J]. J Dent Res, 1979, 58:1866-1871

[30] Rugh JD, Montgomery GT. Physiological reactions of patients with TM disorders vs symptom-free controls on a physical stress task [J]. J Craniomandib Disord,1987, 1:243-250

[31] Moss RA, Adams HE. Physiological reactions to stress in subjects with and without myofascial pain dysfunction symptoms [J]. J Oral Rehabil, 1984, 11:219-232

[32] Chrousos GP. Stressors, stress, and neuroendocrine integration of the adaptive response. The 1997 Hans Selye Memorial Lecture[J]. Ann N Y Acad Sci, 1998, 851:311-335

[33] Gold PW, Pigott TA, Kling MK, et al. Basic and clinical studies with corticotropin releasing hormone: implications for a possible role in panic disorder[J]. Psychiatr Clin North Am, 1988, 11:327

[34] Habib KE, Gold PW, Chrousos GP. Neuroendocrinology of stress[J]. Endocrinol Metab Clin North Am, 2001, 30:695-728

[35] Selye H. Stress in health and disease. Butterworths, Boston, 1976.

[36] Chrousos GP, Gold PW. The concepts of stress and stress system disorders. Overview of physical and behavioural homeostasis[J]. JAMA, 1992, 267:1244-1252

[37] Chrousos GP, Loriaux DL, Gold PW. Advances in experimental medicine and biology, vol 245. Mechanisms of physical and emotional stress. Plenum, New York, 1988

[38] Melzack R. From the gate to the neuromatrix[J]. Pain Suppl, 1999, 6: S121-S126

[39] Millan MJ. Descending control of pain[J]. Prog Neurobiol, 2002, 66: 355-474

[40] Ferrier IN. Disturbed hypothalamic-pituitary-adrenal axis regulation in depression: causes and consequences[J]. In: Montgomery SA, Corn TH (eds) Psychopharmacology of depression. Oxford University Press, NewYork, 1994: 47-56

[41] Korszun A, Hinderstein B, Wong M. Comorbidity of depression with chronic facial pain and temporomandibular disorders [J]. Oral Surg Oral Med Oral Pathol Oral Radiol Endod, 1996, 82:496-500

[42] Bernini GP, Argenio GF, Vivaldi MS, et al. Effects of fenfluramine and ritanserin on prolactin response to insulin-induced hypoglycemia in obese patients: evidence for failure of the serotoninergic system[J]. Horm Res, 1989, 3:133-137

[43] Kaye WH, Gwirtsman HE, George DT, et al. Elevated cerebrospinal fluid levels of immunoreactive corticotropinreleasing hormone in anorexia nervosa: relation to state of nutrition, adrenal function, and intensity of depression[J]. J Clin Endocrinol Metab, 1987, 64:203-208

[44] Insel TR, Kalin NH, Guttmacher LB, et al. The dexamethasone suppression test in patients with primary obsessive-compulsive disorder [J]. Psychiatry Res, 1982, 6: 153-160

[45] Wand GS, Dobs AS. Alterations in the hypothalamic-pituitary-adrenal axis in actively drinking alcoholics [J]. J Clin Endocrinol Metab, 1991, 72:1290-1295

[46] Griep EN, Boersma JW, de Kloet ER. Altered reactivity of the hypothalamic- pituitary- adrenal axis in the primary fibromyalgia syndrome[J]. J Rheumatol, 1993, 20:469-474

[47] Vanderpool J, Rosenthal N, Chrousos GP, et al. Evidence for hypothalamic CRH deficiency in patients with seasonal affective disorder [J]. J Clin Endocrinol Metab, 1991, 72:1382-1387

[48] Gold PW, Chrousos GP. The endocrinology of melancholic and atypical depression: relation to neurocircuitry and somatic consequences [J]. Proc Assoc Am Physicians, 1999, 111:22-34

[49] Demitrack MA, Dale JK, Straus SE, et al. Evidence for impaired activation of the hypothalamic-pituitary-adrenal axis in patients with chronic fatigue syndrome [J]. J Clin Endocrinol Metab, 1991, 73: 1224-1234

[50] Geissler PR. An investigation of the stress factor in the mandibular dysfunction syndrome[J]. J Dent, 1985, 13:283-287

[51] Korszun A, Young EA, Singer K, et al. Basal circadian cortisol secretion in women with temporomandibular disorders [J]. J Dent Res, 2002, 81:279-283

[52] Klerman EB, Goldenberg DL, Brown EN, et al. Circadian rhythms of women with fibromyalgia [J]. J Clin Endocrinol Metab, 2001, 86: 1034-1039

[53] Lariviere WR, Melzack R. The role of corticotropinreleasing factor in pain and analgesia[J]. Pain, 2000, 84:1-12

[54] Roveroni RC, Parada CA, Cecilia M, et al. Development of a behavioural model of TMJ pain in rats: the TMJ formalin test [J]. Pain, 2001, 94:185-191

[55] Gameiro GH, Andrade Ada S, de Castro M, et al. The effects of restraint stress on nociceptive responses induced by formalin injected in rat's TMJ[J]. Pharmacol Biochem Behav, 2005, 82:338-344

[56] Wei F, Dubner R, Ren K. Nucleus reticularis gigantocellularis and nucleus raphe magnus in the brain stem exert opposite effects on behavioural hyperalgesia and spinal Fos protein expression after peripheral inflammation[J]. Pain, 1999, 81:215-219

[57] Wei F, Dubner R, Ren K. Laminar-selective noradrenergic and serotoninergic modulation includes spinoparabrachial cells after inflammation[J]. Neuroreport, 1999, 10:1757-1761

[58] Adell A, Casanovas JM, Artigas F. Comparative study in the rat of the actions of different types of stress on the release of 5- HT in raphe nuclei and forebrain areas[J]. Neuropharmacology, 1997, 36:735-741

[59] Kirby LG, Allen AR, Lucki I. Regional differences in the effects of forced swimming on extracellular levels of 5-hydroxytryptamine and 5-hydroxyindole acetic acid[J]. Brain Res, 1995, 682:189-196

[60] Gameiro GH, Gameiro PH, Andrade AS, et al. Nociception- and anxietylike behavior in rats submitted to different periods of restraint stress[J]. Physiol Behav, 2006, 87:643-649

[61] Palermo TM, Drotar D. Prediction of children's postoperative pain: the role of pre-surgical expectations and anticipatory emotions [J]. J Pediatr Psycho, 1996, 21:683-698

[62] Passchier J, Verheij R, Tulen JH, et al. Positive associations between anticipatory anxiety and needle pain for subjective but not for physiological measures of anxiety[J]. Psychol Rep, 1992, 70:1059-1062

[63] Schreiber S, Vinokur S, Shavelzon V, et al. A randomized trial of fluoxetine versus amitriptyline in musculo-skeletal pain[J]. Isr J Psychiatry Relat Sci, 2001, 38:88-94

(下转第 2341 页)

2010,21(4):1213-1217

[3] Shaw GY, Khan J.Precise repair of orbital maxillary zygomatic fractures[J]. Arch Otolaryngol Head Neck Surg, 1994,120(6):613-619

[4] Yu H, Shen G, Wang X,et al. Navigation-guided reduction and orbital floor reconstruction in the treatment of zygomatic-orbital-maxillary complex fractures [J].J Oral Maxillofac Surg, 2010,68(1):28-34

[5] Turco C, Nisio A, Brunetti F,et al. Fracture of the orbitomaxillo-zygomatic complex. Follow- up study [J].Minerva Stomatol, 1989,38(7):811-813

[6] 张益,孙勇刚.颌骨坚固内固定[M].北京:北京大学医学出版社,2003:247-253
ZHANG Yi,SUN Yong-gang.The firm internal fixation of jaw [M]. Beijing: Peking University Medical Press,2003:247-253

[7] Nardi P, Acocella A, Acocella G.Sequelae of zygomaticorbitomaxillary fractures. Report of 70 cases and review of literature [J]. Minerva Stomatol,2003, 52(6):261-266

[8] 徐金科,刘彦普,薄斌,等.548 例颌面创伤患者的回顾性分析[J].中国口腔颌面外科杂志,2007,2(5):391-94
XU Jin-ke,LIU Yan-pu,BO Bin,et al.The retrospective analysis on 548 patients of maxillofacial trauma [J]. Chinese Oral and Maxillofacial Surgery Journal, 2007,2(5):391-94

[9] Dziadek H, Cieślik T.Treatment of zygomatico-orbital and zygomatico-maxillo- orbital fractures by open reduction and rigid internal fixation[J].Wiad Lek, 2005, 58(5-6):270-274

[10] Dziadek H, Cieślik T. Causes and effects of zygomatico-orbital and zygomatico- maxillo- orbital fractures managed by open reduction and rigid internal fixation [J]. Ann Univ Mariae Curie Sklodowska Med, 2004,59(2):44-51

[11] Giudice M, Colella G, Marra A.The complications and outcomes of fractures of the orbital- maxillary-zygomatic complex [J].Minerva Stomatol,1994,43(1-2):37- 41

[12] 王东,彭诚,崔江涛,等. 颧骨复合体骨折临床治疗探讨[J]. 现代口腔医学杂志,2006,20(3):261-262
WANG Dong,PENG Cheng,CUI Jiang-tao,et al. The research on the clinical treatment of zygomatic complex fracture [J]. Modern Oral Medicine Journal,2006,20(3):261-262

[13] Zhang QB,Dong YJ,Li ZB,et a1. Coronal incision for treating zygomatic complex fractures [J]. J Craniomaxillofac Surg,2006,34(3):182-185

[14] 夏德林,归来,张智勇,等.头皮冠状切口并发症分析及防治 [J].中华整形外科杂志,2005,21(4):255-257
XIA Hou-lin,GUI Lai,ZHANG Zhi-yong,et al. The prevention and analysis of the complica- tions of scalp coronal incision [J]. Chinese Plastic Surgery Journal,2005,21(4):255-257

[15] 张清彬,东耀峻,李祖兵,等.头皮冠状切口整复颧骨复合体骨折的临床分析[J].中华创伤杂志,2005,21(2):136-137
ZHANG Qin-bing,DONG Yao-jun,LI Zu-bing,et al.The clinical analysis on the restoration of zygomatic complex fractures by coronal scalp incision[J]. Chinese Trauma Journal,2005,21(2):136-137

(上接第 2394 页)

[64] Gerber PE, Lynd LD. Selective serotonin-reuptake inhibitor-induced movement disorders[J]. Ann Pharmacother, 1998, 32:692-698

[65] Contoreggi C, Rice KC, Chrousos G. Nonpeptide corticotropin-releasing hormone receptor type 1 antagonists and their applications in psychosomatic disorders. Neuroendocrinology, 2004, 80:111-123

[66] Bodnar RJ, Klein GE. Endogenous opiates and behavior: 2003 [J]. Peptides, 2004, 25: 2205- 2256

[67] Maixner W, Gracely RH, Zuniga JR, et al. Cardiovascular and sensory responses to forearm ischemia and dynamic hand exercise [J]. Am J Physiol, 1990, 259: R1156-R1163

[68] Laskin DM. Etiology of the pain-dysfunction syndrome. J Am Dent Assoc, 1969, 79:147-153

[69] Rugh JD, Solberg WK. Psychological implications in temporomandibular pain and dysfunction[J]. Oral Sci Rev, 1976, 7:3-30

[70] Drolet G, Dumont EC, Gosselin I, et al. Role of endogenous opioid system in the regulation of the stress response [J]. Prog Neuropsychopharmacol Biol Psychiatry, 2001, 25:729-741

[71] Feinmann C. Psychogenic facial pain: presentation and treatment[J]. J Psychosom Res,1983, 27:403-410

[72] Feinmann C. The mouth, the face and the mind[J]. Oxford University Press, Oxford, 1999.

[73] Fricton JR. Masticatory myofascial pain: an explanatory model integrating clinical, epidemiological and basic science research [J]. Bull Group Int Rech Sci Stomatol Odontol, 1999, 41:14-25

[74] McQuade R, Young AH. Future therapeutic targets in mood disorders: the glucocorticoid receptor [J]. Br J Psychiatry, 2000, 177: 390-395

[75] Bonjardim LR, Gaviao MB, Pereira LJ, et al. Anxiety and depression in adolescents and their relationship with signs and symptoms of temporomandibular disorders[J]. Int J Prosthodont, 2005, 18:347-352

[76] Kuttila M, Niemi PM, Kuttila S, et al. TMD treatment need in relation to age, gender, stress, and diagnostic subgroup [J]. J Orofac Pain, 1998, 12:67-74

[77] Held K, Kunzel H, Ising M, et al. Treatment with the CRH1-receptorantagonist R121919 improves sleep-EEG in patients with depression[J]. J Psychiatr Res, 2004, 38:129-136

[78] Kunzel HE, Ising M, Zobel AW, et al. Treatment with a CRH-1-receptor antagonist (R121919) does not affect weight or plasma leptin concentration in patients with major depression [J]. J Psychiatr Res, 2005, 39:173-177

[79] Auvenshine RC. Psychoneuroimmunology and its relationship to the differential diagnosis of temporomandibular disorders [J]. Dent Clin North Am, 1997, 41:279-296

[80] Maier SF, Watkins LR, Fleshner M. Psychoneuroimmunology. The interface between behavior, brain, and immunity [J]. Am Psychol, 1994, 49:1004-1017

[81] Marbach JJ, Schleifer SJ, Keller SE. Facial pain, distress, and immune function[J]. Brain Behav Immun, 1990, 4:243-254

[82] Suvinen TI, Hanes KR, Gerschman JA, et al. Psychophysical subtypes of temporomandibular disorders [J]. J Orofac Pain, 1997, 11: 200-205