A unifying concept of the TMJ pain-dysfunction syndrome

Martin D. Lerman, DDS, Chicago

Pain, muscle tenderness, clicking, or alteration of mandibular movement are cardinal symptoms of the temporomandibular joint (TMJ) pain-dysfunction syndrome (PDS). Controversy still exists over the origin of the syndrome and what is the best treatment for it. One group holds that occlusion is the major etiologic factor; another that psychological and muscle factors are of the greatest etiologic importance. A number of investigators now hold that occlusal and psychologic factors operate together to produce the PDS. A theory is offered here that muscle hyperactivity associated with PDS is not a generalized hyperactivity but one in which activity of certain motor units is increased disproportionately. Treatment should combine psychologic and physiologic modalities. Pain and tension should be controlled, and spasm eliminated. However, if symptoms continue, occlusal treatment should be instituted. A full-coverage splint may serve either as the sole occlusal treatment or as a preliminary to equilibration or restorative procedures.

The cardinal symptoms of the TMJ pain-dysfunction syndrome (PDS) are pain, muscle tenderness, clicking in the joint, and limitation or alteration of mandibular movement.[1,2] It is generally agreed that these are nonorganic symptoms, based usually not in the joint itself, but in nearby muscles, primarily the masseter, temporal, and the medial and lateral pterygoid muscles.[3-7]

Although most investigators hold the syndrome to have multiple causes,[7-9] considerable controversy still exists regarding both its origin and best treatment. One group, whose roots go back to Prentiss,[10] Monson,[11] Goodfriend,[12] and Costen,[13] believes the occlusion to be the major etiologic factor. Successful occlusal treatment of the PDS has been reported with regularity. Two approaches are used, either singly or in combination: alteration of the occlusion with use of one of many types of removable occlusal splints,[14-17] and alteration of the teeth themselves, by equilibration,[18-20] rehabilitation,[21-23] or orthodontic movement.[24]

A second and more recent school of thought, the psychophysiologic, holds psychological and muscle factors to be of greatest etiologic importance. This group includes Schwartz and Moulton,[3,8,25-29] and Laskin and Lupton.[7,30-33]

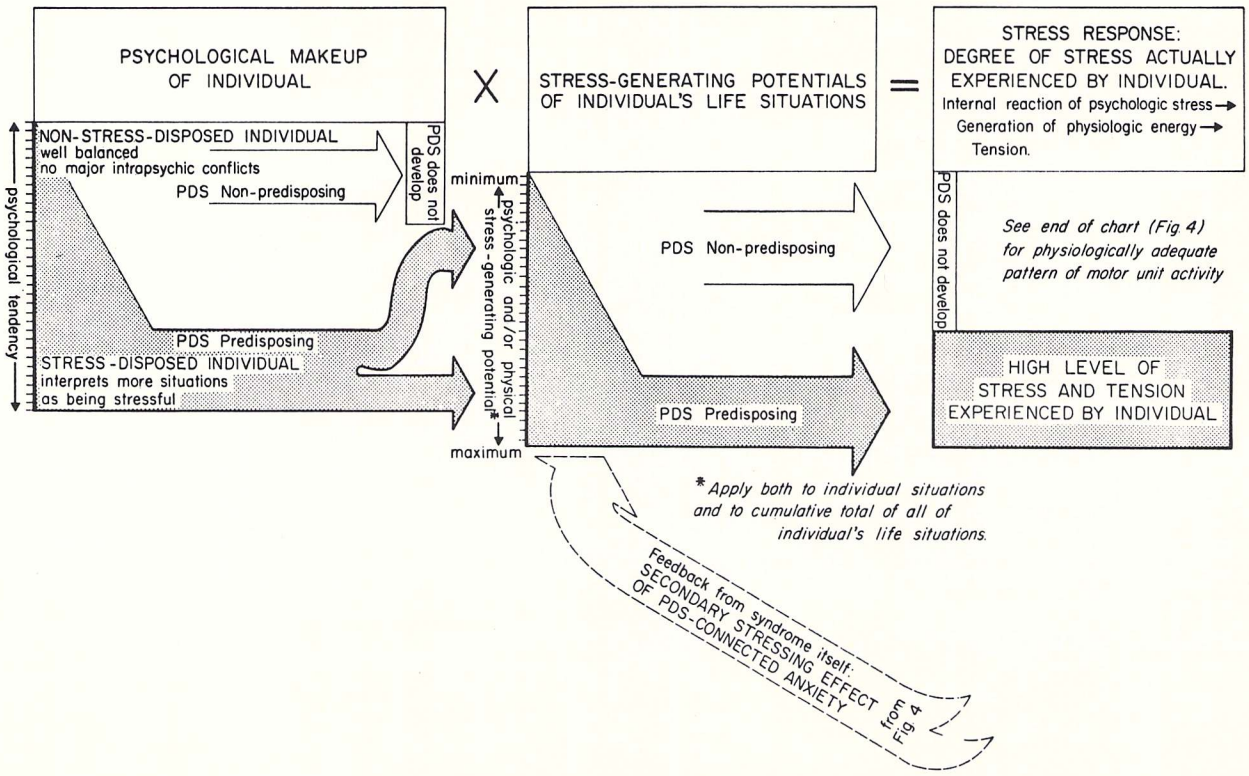

Fig 1 ■ Interaction of psychological and situational factors in PDS.

Although not excluded completely, occlusal factors are held to be of lesser, or even of only occasional importance.[7,34] These authors recommend treating the PDS with tranquilizing and muscle-relaxing drugs,[35,36] physiotherapy of the muscles themselves,[27,35] and psychotherapy.[29,31] The occlusion is altered in only a small percentage of cases.

These two schools of thought hold diametrically opposed positions: the occlusal group contends that occlusal disharmonies exist before the development of the PDS, and are of prime etiologic significance, whereas the psychophysiologic school holds that they are not vital to development of the PDS, and in fact can be a consequence of it.[7]

An increasing number of investigators now hold that occlusal and psychologic factors operate together to produce the PDS.[37-38] Ramfjord and Ash[37] state this position succinctly: "the cause . . . is a combination of psychic tension and occlusal disharmony resulting in muscular hyperactivity."

A theory is offered here which extends this position farther: it holds that the muscle hyperactivity associated with the PDS is not simply a generalized hyperactivity, but rather one in which the activity of certain motor units is increased disproportionately. These are the motor units which must carry out the compensatory adjustments in mandibular placement necessitated by the specific occlusal disharmony. PDS symptoms arise when the hyperactivity of these accommodatively affected motor units has brought them to the point at which they can no longer respond physiologically. The PDS's characteristic symptoms arise when these motor units become uncoordinated or go into spasm.

Etiological chain

The flow chart (Fig 1-4), sets forth an overview of PDS etiology. It illustrates how each of the psychologic, occlusal and physiologic factors involved in PDS etiology can be predisposing to the development of the syndrome, and the many ways in which these same factors, though present, may not predispose to its development. The text is intended to supplement the flow chart and may be read in conjunction with it.

■ *Interaction of psychological and situational factors (Fig 1):* Psychological factors play a vital role in the PDS. Emotionally based stress con-

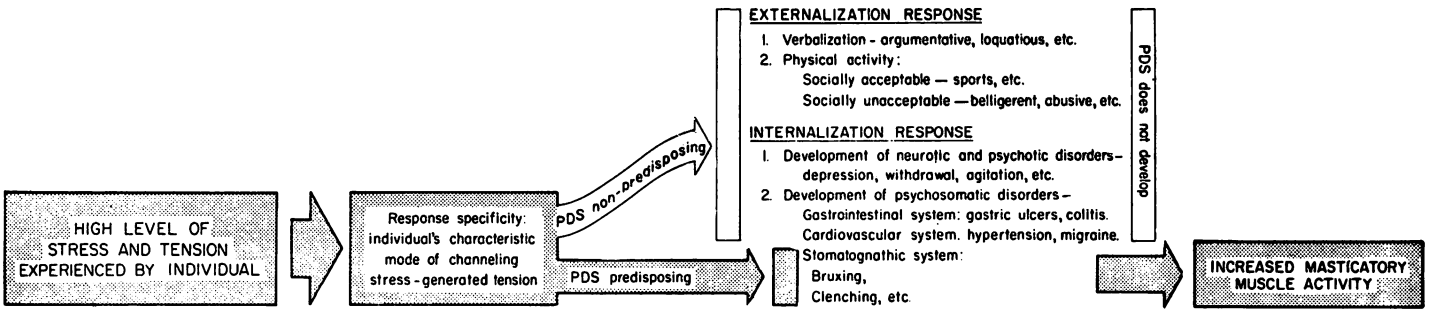

Fig 2 ■ Response specificity: psychophysiologic channeling of tension in PDS.

tributes to the development of the syndrome initially by increasing the overall activity of the masticatory muscles, mainly through bruxing and clenching.[39] Once the syndrome has developed, the pain and other symptoms may then cause the patient to become anxious and tense about its future course. This, in feedback fashion, increases further both the masticatory muscle activity and the severity with which the patient reacts to symptoms,[18,28,40] so that the syndrome tends to perpetuate itself.

Every element of life (whether momentary or of long duration) has some greater or lesser stress-generating potential. How much stress the individual actually experiences, however, is dependent not only on the objective reality of a situation (for example, financial or marital difficulties, environmental circumstances, and so on) but, in addition, on the individual's interpretation of this reality.[41] Together these determine the amount of stress the individual experiences psychologically, and to which he then reacts physiologically.[42] These physiologic reactions generate and release energy internally, and this creates internal tension.[43-45]

Whether the person discharges this tension or "bottles it up" internally is of great significance. Most persons discharge the tensions that build up under stress during the process of coping with the stress-producing situation. Others, however, are unable or unwilling to vent them, and habitually turn them inward. Ultimately, these persisting and accumulating physiologic energies can have physical consequences: a psychosomatic (or psychophysiologic) disorder such as gastric ulcers, colitis, hypertension, or neurodermatitis may develop.[46] The TMJ PDS is conceived of herein as one special form of this wider group of psychosomatic disorders.

Studies of psychological characteristics indicate that a high degree of commonality exists among all psychosomatic patients.[47] They share certain pivotal characteristics: they are stress-disposed; they retain, rather than vent, the tension created by the response to stress; and—of special interest to us—they turn this unvented tension inward into somatic channels.

■ *Response specificity (Fig 2):* Each individual channels his tension into a relatively specific organic modality; this process is known as "response specificity."[48-51] The channel into which tensions are directed determines, in large measure, which psychophysiologic disorder may develop. Hence, if the gastrointestinal tract is involved, gastric ulcers or colitis may develop. Similarly, if the stomatognathic system is involved, and the other requisite elements of the chain of etiology are present (see flow chart), the PDS may develop. If one or more links is missing, however, or if the tension is channeled elsewhere, it is unlikely that the PDS will develop.[52]

It is contended, then, that patients with PDS are highly similar in their personality makeup to patients with other psychophysiologic disorders.[28,31,32,53,54] They differ in their tendency to channel undischarged psychic energy and tension through the stomatognathic system.

■ *Effect of stomatognathic functional imbalance on masticatory muscle activity (Fig 3):* Since the individual components of the stomatognathic

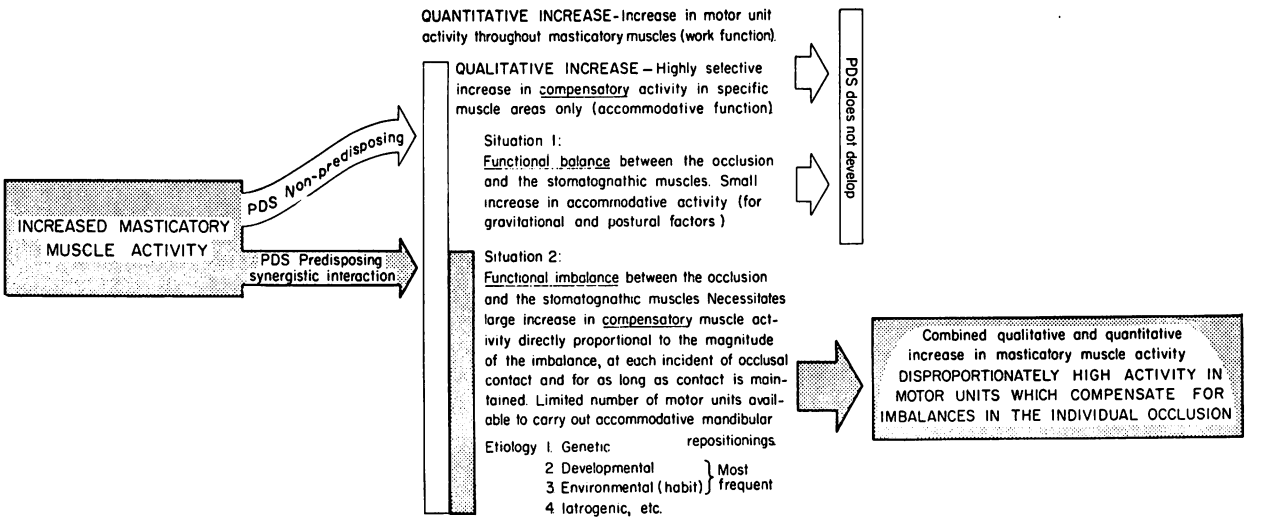

Fig 3 ■ Effect of stomatognathic functional imbalance on masticatory muscle activity.

system interact closely in function, displacement of any stomatognathic component, or discrepancy in its configuration, must be compensated for. This theory holds that the muscles implement these compensatory reactions. One of the most frequent sources of stomatognathic imbalance is theorized to be malpositioning of the mandible by an imperfect occlusion,[55] in response to which a compensatory (that is, accommodative) reaction is triggered, primarily in the masticatory muscles, and to a lesser extent in the other stomatognathic muscles. From the electromyographic studies of Perry,[18] Ramfjord,[56] and others, it can be seen that in these accommodative reactions the quantity, distribution, or sequence of motor unit activity may be altered at any point in the bilateral muscle complex. The pattern of motor units that become involved in each accommodative reaction is determined by the individual occlusal disharmony.[57] One element, however, is theorized to be common to all of these patterns and central to the development of the PDS: activity of the accommodatively affected motor units is increased disproportionately. Psychologically induced (and stress-connected) bruxing and clenching increase the impact of this unequal distribution of motor unit activity enormously. It is held, then, that frequent bruxing or clenching and an occlusal disharmony are both necessary to produce this extremely disproportionate activity. One without the other does not, and the syndrome does not develop. Increased accommodated muscle activity is therefore held to predispose to the PDS, whereas muscular hyperactivity per se (an overall increase in unaccommodated muscle activity) does not.

Frequently affected in PDS patients are the lateral pterygoid, anterior and posterior fibers of the temporal, and the deep portion of the masseter muscles. These are the muscle areas capable of making horizontal adjustments in the position of the mandible, and there are far fewer motor units in these than in the muscle areas with the capability of elevating the mandible. Therefore, where horizontal adjustments in mandibular placement must be made continuously because of occlusion-muscle incompatibility, each of these fewer appropriate motor units must be called into activity more frequently. This disproportionately high activity may prove excessive, and the motor unit's ability to function properly may ultimately be disturbed. In clinical terms, this may be said to represent exhaustion of these accommodatively involved motor units.

■ *Adequate and inadequate motor unit response and the development of PDS symptoms (Fig 4):* Whether a muscle or its component motor units responds adequately or inadequately in function depends on several factors: the magnitude of the demand for activity, the quantity of appropriately vectored motor units available to share this demand, and the capacity of each of these motor units and its neural elements. Together these can be stated as the functional demand/physiologic capacity (FD/PC) balance. As long as the repeated demands for a given motor unit to generate its force vector remain consistently within its capacity, the motor unit continues to perform asymptomatically. When the functional

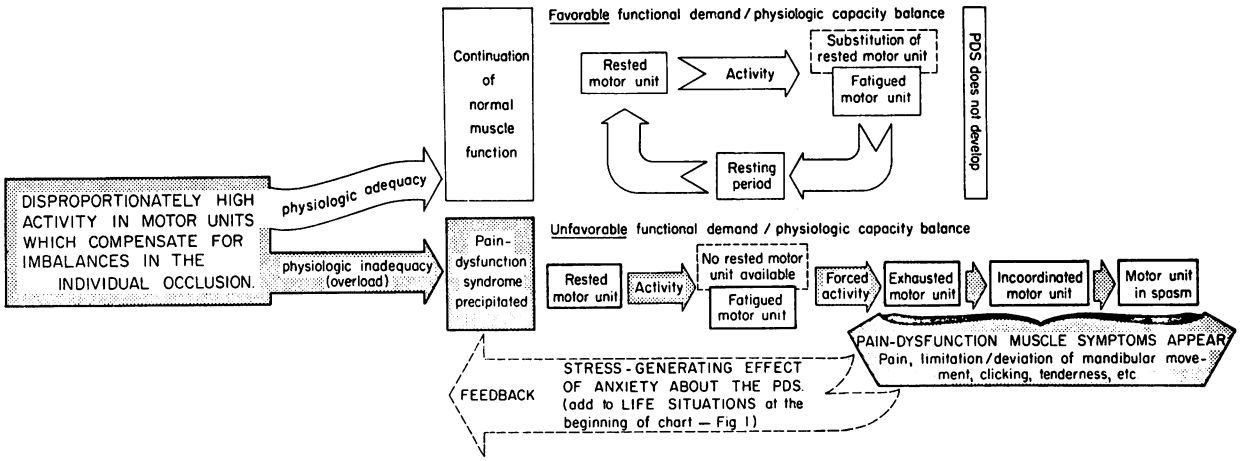

Fig 4 ■ Adequate and inadequate motor unit response and development of PDS symptoms.

demand exceeds the motor unit's capacity it becomes progressively more incoordinated with the other motor units in the same and other muscles, and it may finally go into spasm.

The FD/PC balance can shift either favorably or unfavorably, and this is commented on in the discussion section.

Treatment of the PDS

In most instances functional demand and physiologic capacity may be restored to favorable balance, and in its broadest sense, this is the underlying goal of most PDS treatment modalities. There are two main avenues to accomplishing this: reducing stomatognathic activity overall (by psychologic modalities; tranquilizers, muscle relaxants, and so on); or reducing it selectively, that is, reducing the accommodative demand to the affected motor units, which is what successful occlusal therapy accomplishes. On this basis, successful occlusal therapy can be far more effective in eliminating symptoms (especially severe ones) than can methods designed to reduce overall stomatognathic activity,[58] which can be only partially successful.

Treatment should aim at breaking the syndrome's chain of origin, by eliminating one or more of its individual etiologic links, if this is possible. If it is not, the intensity of those elements amenable to treatment should be reduced as much as is practical.

Treatment should begin simply and conservatively, and should escalate only as necessary. The first stage of treatment should deal with the control of pain with analgesics, resolution of muscle spasm, reduction of tension—for example, diazepam (Valium) 5 mg, three times a day—and importantly, the relief of PDS-connected anxiety.

This relief can be accomplished by giving the patient a reassuring explanation of what the PDS is and of what may be expected: that the syndrome is not life-threatening (not cancer); that the possibility of surgery is remote; that symptoms are due to a disturbance in masticatory muscle function which is usually reversible with treatment; that stress and tension play a large part in the development of the syndrome; that habitual oral activity (clenching, bruxing, and so on) is usually involved, and that it is to the patient's advantage if this can be controlled voluntarily.

Relieving PDS-connected anxiety in this manner is important, takes very little time, and is well within the capacity of every dentist. Moreover, according to Millon,[59] it probably achieves much of what more extensive forms of psychotherapy can accomplish in treating the PDS. The work of Lupton,[30] another psychologist, also supports this view. He found that a reassuring explanation of the PDS (which therefore dealt only with PDS-connected anxiety) was as effective in controlling symptoms as a course of traditional psychological counseling. This can probably be explained on the basis of

two links in PDS etiology (the personality factors which predispose to internal stress and tension,[47] and the habituated channeling of tension through the stomatognathic system[58]) being deep-seated and very resistant to change, even with long-term psychotherapy. PDS-connected anxiety, by contrast, is more superficial and more easily dealt with.[59]

If muscle spasm is present, its self-perpetuating cycle should be interrupted by the use of muscle relaxants, physical therapy (heat, ice packs, or ethyl chloride spray), and muscle exercises.[26-27]

In mild and responsive cases the preceding measures by themselves may control symptoms adequately, so that no further treatment may be necessary. In many patients, however, pain, spasm and other symptoms may persist. In these, a second stage, that of occlusal therapy, should be instituted.

Definitive evaluation or treatment of the occlusion should not be attempted while spasm exists, however. Spasm may distort the occlusal and skull-mandible relationships which are observable clinically, and evaluations based on distorted relationships will themselves be distorted. It is unwise, therefore, to employ irreversible occlusal measures in the hope of thereby relieving persisting spasm. Only easily reversible treatments, such as removable occlusal splints, should be employed. The splint's occlusal configuration may be readjusted as necessary until spasm is resolved.

It cannot be overemphasized that since symptoms are muscle-based, occlusal therapy of the PDS should be muscle-oriented. Since, by definition, the neuromuscular position (also called "muscular") is that position of the mandible which is most harmonious with the muscles,[55] occlusal corrections should attempt to make the intercuspal and neuromuscular positions of the mandible coincide.[60] If this can be accomplished successfully, motor unit activity would be redistributed in a pattern better tolerated by the muscles, and muscle accommodation would be reduced to minimum.

Unfortunately, however, present clinical techniques, which establish either terminal hinge or long centric placement, do not consistently accomplish this. With current techniques, an entire physiologic dimension is largely missing from analysis of the occlusion; namely, the occlusion's compatibility with the muscles.

This limitation may now have been overcome. It may now be possible to establish the neuromuscular position clinically, with use of hydrostatic appliance and techniques described elsewhere.[61-63] Exceptionally successful results have been obtained with treatment-resistant PDS patients.[64] It is thought that permitting the mandible to migrate into its neuromuscular position is responsible.

When conventional occlusal techniques are used, however, great caution and conservatism should be exercised. As in treating spasm, it is prudent to first use an easily reversible procedure before altering the teeth irreversibly or permanently changing skull-mandible relationships. The upper full coverage occlusal splint[17] is recommended as being a conservative, safe, maximally stabilizing, and relatively effective means of treating the PDS. It may serve as either the sole occlusal treatment (worn indefinitely full- or part-time, or only during episodes of symptom recurrence), or as a preliminary to further occlusal procedures such as equilibration or rehabilitation.

When skull-mandible relationships are to be changed permanently, the new relationships are best established and tested first on a temporary basis by use of a splint, since PDS patients have been found to be acutely sensitive and hyperreactive to factors of mandibular placement.[64] Final restorative procedures should be carried out only after the patient has worn the splint long enough to demonstrate acceptance of the new occlusal and skull-mandible relationships by the muscles.

Discussion

A number of practical questions arise about the PDS, regarding both this concept specifically and the syndrome generally:

What accounts for the fact that success is claimed for such widely differing treatments as psychotherapy and occlusal rehabilitation? Each treats a different link in the etiologic chain. If this chain can be broken at any point, symptoms undergo resolution.[52]

Are these etiologic links all equally responsive to treatment? No. The internal psychodynamics of the stress-disposed individual can rarely be so completely restructured as to render that person nonstress-disposed,[47] even with long-term psychotherapy. By contrast, if occlusal therapy is successful (success is measured in terms of improving the pattern of muscle activity), it can

eliminate the disproportionate distribution of activity to the symptomatic motor units, thereby effectively breaking the etiologic chain. An arbitrary choice between the two is unnecessary, however, since treatment should always begin by dealing with PDS-connected anxiety, the most readily accessible psychological element. When this is relieved by the dentist it may, by itself, relieve symptoms adequately. If further treatment is necessary, beyond relieving spasm, more effective results can be obtained from muscle-oriented occlusal therapy than from more extensive psychotherapy.

Why do many with gross malocclusions never develop the PDS, whereas others with normal-appearing occlusions do? One of the most significant, albeit unseen, criteria of the ideal occlusion is its compatibility with the muscles.[65,66] Two factors which strongly influence adequacy of the muscle response are totally unrelated to the ideals of tooth intercuspation: these are the extent of muscle accommodation necessary with each centric contact, and the frequency and duration of these contacts.

An occlusion with seemingly ideal intercuspation may invisibly require a great deal of muscle accommodation and in time may lead to the development of PDS symptoms. Another occlusion, despite being malaligned cuspally, may require only slight accommodation, well within the muscles' physiologic capacity. Frequency of occlusal contact, too, may vary widely from individual to individual. In some, these contacts are limited to those associated with mastication and round-the-clock swallowing. In patients who have harmful oral habits, occlusal contacts are greatly increased by frequent and sustained bruxing and clenching. An accommodative demand, however high, can be tolerated by the muscles if called into play infrequently enough. Conversely, a smaller accommodative demand, if triggered frequently enough, can exceed physiologic capacity and lead to the PDS.

Why do symptoms often appear spontaneously without any apparent correlation to dental procedures, trauma, and so on? The balance between activity and capacity may have become unfavorable subtly and gradually, without the patient's awareness. Stomatognathic activity may have increased because of an increase in psychological stress; muscle accommodation may have increased as a result of tooth loss, attrition, migration of teeth into less favorable positions, and so on. Physiologic capacity may have declined subtly because of aging, degeneration, and so on. In regard to the latter point, it is no mere coincidence that most patients with PDS are middle aged.[2]

Why are PDS symptoms often episodic? Since activity, accommodation and capacity are each variables which can increase or decrease, their balance may be favorable at times (absence or resolution of symptoms), and unfavorable at others (symptoms appear).

Why do symptoms sometimes appear after dental work of only a seemingly minor nature? Undetected subclinical PDS symptoms may have existed before treatment was begun: muscle incoordination or tenderness (particularly in the seldom examined lateral pterygoid muscle), or an unreported TMJ click. Even if none of these was present, the patient may have been on the brink of developing clinical symptoms of the PDS because of an already high but undetectable level of accommodative muscle activity. The dental work may have introduced only a minor interference into the occlusion. However, this small increment of muscle accommodation, added to an already high level of accommodation, could have caused symptoms to increase to a clinical level.

What causes an occlusion to require sizable muscle accommodation? Or, to rephrase the question, how does incompatibility between the occlusion and the muscles develop? Incompatibilities can develop in many ways and at many points during the lifetime of a dentition:[67-69] for example, genetic mismatching of teeth and skeletal elements; faulty growth and development; habits which distort tooth position or the underlying bones; trauma; loss of teeth; or interfering dental restoration. Two points should be mentioned for good perspective; dental restorative procedures, although significant, are far from being the most frequent source of occlusion-muscle incompatibility, and only some occlusion-muscle incompatibilities ultimately result in the development of PDS (see flow chart).

Summary and conclusions

This unifying concept holds that the PDS results from the interplay of an unbroken chain of etiologic factors. Some of these, the psychologic factors, lead to an overall increase in masticatory muscle activity. Others, occlusal and anatomic in nature, increase muscle activity selectively.

The dynamics of this theory are as follows:

some individuals are psychologically predisposed to magnify the stress-generating potentials of their life situation. Consequently, these individuals actually experience excessive degrees of psychological stress. The body responds physiologically to this by generating energy and liberating it internally. Instead of venting and discharging the resulting tensions during the coping process, potential PDS patients turn them inward, and channel them through the stomatognathic system. Increased clenching and bruxing activity results. When occlusal disharmony exists, the mandible must be repositioned accommodatively by the muscles at each of its frequent centric contacts. This causes a selective, disproportionately high increase in activity of those motor units which carry out the accommodative response, that is, compensate for the disharmony in that particular occlusion. As long as these motor units maintain physiologic adequacy and can satisfy these demands, the PDS does not develop, and the individual remains asymptomatic. When they cannot, however, the affected motor units become progressively more incoordinated, finally going into spasm. PDS symptoms appear during the stage of muscle inadequacy, and at the sites of the accommodatively affected motor units.

Treatment should routinely combine both psychologic and physiologic modalities, and should follow an orderly sequence. In the first stage of treatment, pain should be controlled with analgesics; tranquilizers should be given to reduce tension; PDS-connected anxiety should be alleviated; and spasm, if present, should be eliminated.

If symptoms persist, a second stage—occlusal treatment—should be instituted. Occlusal correction should be muscle-oriented: that is, aimed at restoring the mandible to its neuromuscular position, and eliminating the interferences which cause muscle incoordination. Because present techniques do not consistently accomplish this, occlusal correction should be approached conservatively. Teeth or interarch relationships should not be altered irreversibly until the proposed changes are first tested with a reversible treatment, such as the full occlusal coverage splint. Such a splint may serve either as the sole occlusal treatment, or as a preliminary to equilibration or restorative procedures.

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Dr. Lerman is assistant professor, department of prosthodontics, University of Illinois, College of Dentistry, Chicago, 60612.

1. Ramfjord, S.P., and Ash, M.M. Occlusion. Philadelphia, W. B. Saunders Co., 1966, p 363.
2. Greene, C.S., and others. The TMJ pain-dysfunction syndrome: heterogeneity of the patient population. JADA 79:1168 Nov 1969.
3. Schwartz, L.A. Temporomandibular joint pain-dysfunction syndrome. J Chronic Dis 3:284 March 1956.
4. Bell, W.E. Clinical diagnosis of the pain-dysfunction syndrome. JADA 79:154 July 1969.
5. Copland, J. Abnormal muscle tension and the mandibular joint. Dent Rec 74:331 Dec 1954.
6. Husted, E. Surgical diseases of the temporomandibular joint. Acta Odontol Scand 14:119 Sept 1956.
7. Laskin, D.M. Etiology of the pain-dysfunction syndrome. JADA 79:147 July 1969.
8. Schwartz, L. Conclusions of the Temporomandibular Joint Clinic at Columbia. J Periodontol 29:210 July 1958.
9. Sicher, H. Oral anatomy, ed 3. St. Louis, C. V. Mosby Co., 1960, p 169.
10. Prentiss, H.J. A preliminary report upon temporo-mandibular articulation in the human type. Dent Cosmos 60:505 June 1918.
11. Monson, G.S. Occlusion as applied to crown and bridge work. J Nat Dent Assoc 7:399 May 1920.
12. Goodfriend, D.J. Symptomatology and treatment of abnormalities of the mandibular articulation. Dent Cosmos 75: 844 Sept 1933.
13. Costen, J.B. Syndrome of ear and sinus symptoms dependent on disturbed function of temporomandibular joint. Ann Otol Rhinol Laryngol 43:1 March 1934.
14. Posselt, U. Physiology of occlusion and rehabilitation, ed 2. Philadelphia, F. A. Davis Co., 1968, p 99.
15. Ramfjord, S.P., and Ash, M.M. Occlusion. Philadelphia, W. B. Saunders Co., 1966, p 223.
16. Krough-Poulsen, W.G. Management of the occlusion of the teeth (part II). In Schwartz, L.L., and Chayes, C.M., eds. Facial pain and mandibular dysfunction. Philadelphia, W. B. Saunders Co., 1968, p 271.
17. Shore, N.A. Occlusal equilibration and temporomandibular joint dysfunction. Philadelphia, J. B. Lippincott Co., 1959, p 131.
18. Perry, H.T. Muscular changes associated with temporomandibular joint dysfunction. JADA 54:644 May 1957.
19. Ramfjord, S.P. Dysfunctional temporomandibular joint and muscle pain. J Prosthet Dent 11:353 March-April 1961.
20. Hankey, G.T. Discussion: affectations of the temporomandibular joint. Proc R Soc Med 49:1130 Dec 2, 1956.
21. Mann, A.W., and Pankey, L.D. Concepts of occlusion: the PM philosophy of occlusal rehabilitation. Dent Clin North Am Nov 1963, p 621.
22. Freese, A.S., and Scheman, L.D. Management of temporomandibular joint problems. St. Louis, C. V. Mosby Co., 1962, p 155.
23. Ramfjord, S.P., and Ash, M.M. Occlusion. Philadelphia, W. B. Saunders Co., p 380.
24. Salzmann, J.A. Practice of orthodontics. Philadelphia, J. B. Lippincott Co., 1966, p 572.
25. Schwartz, L.L. Disorders of the temporomandibular joint. Philadelphia, W. B. Saunders Co., 1959, p 28.
26. Schwartz, L.L. Disorders of the temporomandibular joint. Philadelphia, W. B. Saunders Co., p 231.
27. Schwartz, L.L., and Chayes, C.M. Facial pain and mandibular dysfunction. Philadelphia, W. B. Saunders Co., 1968, p 340.
28. Moulton, R.E. Emotional factors in non-organic temporomandibular joint pain. Dent Clin North Am Nov 1966, p 609.
29. Moulton, R.E. Psychologic considerations in the treatment of occlusion. J Prosthet Dent 7:148 March 1957.
30. Lupton, D.E. A comparative analysis of the effectiveness of counseling, instruction and dental treatment, PhD dissertation. University of Chicago, 1967.

31. Lupton, D.E. A preliminary investigation of the personality of female temporomandibular joint dysfunction patients. Psychother Psychosom 14:199 No. 3, 1966.

32. Lupton, D.E. Psychological aspects of temporomandibular joint dysfunction. JADA 79:131 July 1969.

33. Shipman, W.G.; Greene, C.S.; and Laskin, D.M. Personality clusters in the myofascial pain-dysfunction syndrome. Abstracted, IADR Program and Abstracts, No. 450 March 1971.

34. Schwartz, L.L. The pain dysfunction syndrome. In Schwartz, L.L., and Chayes, C.M., eds. Facial pain and mandibular dysfunction. Philadelphia, W. B. Saunders Co., p 152.

35. Bell, W.H. Nonsurgical management of the pain-dysfunction syndrome. JADA 79:161 July 1969.

36. Kutscher, A.H.; Zegarelli, E.V.; and Lamon-Havers, R.W. Pharmacologic methods. In Schwartz, L.L., and Chayes, C.M., eds. Facial pain and mandibular dysfunction. Philadelphia, W. B. Saunders Co., 1968, p 300.

37. Ramfjord, S.P., and Ash, M.M. Occlusion, ed 2. Philadelphia, W. B. Saunders Co., 1971, p 184.

38. Graber, T.M. Overbite—the dentist's challenge. JADA 79: 1135 Nov 1969.

39. Perry, H.T., and others. Occlusion in a stress situation. JADA 60:626 May 1960.

40. Goldstein, I.B. Role of muscle tension in personality theory. Psychol Bull 61:413 June 1964.

41. Millon, T. Modern psychopathology. Philadelphia, W. B. Saunders Co., 1969, p 210.

42. Selye, H. Physiology and pathology of exposure to stress. Montreal, Medical Publications, 1950.

43. Alexander, F. Psychosomatic medicine. New York, W. W. Norton Co., 1950.

44. Alexander, F., and Flagg, G. The psychosomatic approach. In Wolman, B.B., ed. Handbook of clinical psychology. New York, McGraw-Hill, 1965, p 855.

45. Alexander, F., and French, T. Studies in psychosomatic medicine. New York, Ronald Press, 1948.

46. Millon, T. Modern psychopathology. Philadelphia, W. B. Saunders Co., p 417.

47. Millon, T. Modern psychopathology. Philadelphia, W. B. Saunders Co., p 618.

48. Lacey, J.I.; Bateman, E.E.; and Van Lehn, R. Autonomic response specificity. Psychosom Med 15:8 Jan-Feb 1953.

49. Lacey, J.I., and Lacey, B.C. Verification and extension of the principle of autonomic response stereotype. Am J Psychol 71:50 March 1958.

50. Mirsky, I.A. Physiologic, psychologic and social determinants in the etiology of duodenal ulcer. Am J Dig Dis 3:285 No. 4, 1958.

51. Malmo, R.B. Activation. In Bachrach, A.J., ed. Experimental foundations of clinical psychology. New York, Basic Books, 1962.

52. Ramfjord, S.P., and Ash, M.M. Occlusion. Philadelphia, W. B. Saunders Co., 1966, p 162.

53. McCall, C.M.; Szmyd, L.; and Ritter, R.M. Personality characteristics in patients with temporomandibular joint symptoms. JADA 62:694 June 1961.

54. Lupton, D.E. Some personality characteristics associated with temporomandibular joint dysfunction. Abstracted, IADR Program and Abstracts, No. 367, 1966.

55. Lerman, M.D. The neuromuscular position of the mandible. Unpublished.

56. Ramfjord, S.P. Bruxism, a clinical and electromyographic study. JADA 62:21 Jan 1961.

57. Perry, H.T. Implications of myographic research. Angle Orthod 25:179 Oct 1955.

58. Millon, T. Modern psychopathology. Philadelphia, W. B. Saunders Co., p 424.

59. Millon, T. Personal communication, 1971.

60. Lammie, G.A. Dental orthopaedics. Oxford, Alden Press, 1966, p 14.

61. Lerman, M.D. Inter-arch hydrostatics: new approach to treatment of the TMJ pain-dysfunction syndrome. Abstracted, IADR Program and Abstracts No. 239 March 1969.

62. Lerman, M.D. Comparative analysis of hydrostatic and occlusally derived mandibular planes. Abstracted, IADR Program and Abstracts No. 186 March 1972.

63. Lerman, M.D. The neuromuscular basis for conventional and hydrostatic splint therapy. In Laskin, D.M., ed. Disorders of the temporomandibular joint. Springfield, Ill, Charles C Thomas. In press.

64. Lerman, M.D. Hydrostatic appliance therapy of the myofascial (TMJ) pain-dysfunction syndrome. Abstracted, IADR Program and Abstracts No. 287 March 1970.

65. Sicher, H. Functional anatomy of the temporomandibular joint. In Sarnat, B., ed. Temporomandibular joint. Springfield, Ill, Charles C Thomas, 1967, p 57.

66. Horowitz, S.L., and Hixon, E.H. The nature of orthodontic diagnosis. St. Louis, C. V. Mosby Co., 1966, p 162.

67. Salzmann, J.A. Principles of orthodontics, ed 2. Philadelphia, J. B. Lippincott Co., 1950.

68. Graber, T.M. Orthodontics: principles and practice, ed 2. Philadelphia, W. B. Saunders Co., 1966.

69. Moyers, R.E. Development of occlusion. Dent Clin North Am 13:523 July 1969.