Jaws: diversities of gnathological history and temporomandibular joint enterprise

Dewey A Nelson, William M Landau

Epitome: etymology, epistemology, aetiology, and epidemiology
Since 1887, temporomandibular dysfunction (TMD) has been a clouded subject with a large penumbra and a complex and ever changing nomenclature (table 1). It is described as a primary disease entity involving the temporomandibular joint (TMJ) with the key symptom of pain ranging from aching and burning to sharp and jabbing. Various concepts of the syndrome(s) have interested, confused, angered, and often frightened potential patients. In addition, prolonged disabilities and expensive radical treatments with serious complications are notorious. The common wisdom is that modern concepts of TMD began with three publications by Costen, an otolaryngologist. However, long before the term TMD originated, pre-Costen authors had already published many of the speculations regarding the disturbed meniscal disc and the associated signs and symptoms that later became known as Costen’s syndrome (tables 2, 3). At first, his “new disease,” allegedly associated with “bony erosions” of the temporomandibular joint (TMJ) and the tympanic plate of the temporal bone, was heralded and enthusiastically accepted by both dentistry and otolaryngology. But by the next decade, this tidy synthesis was undone by well planned studies and the contretemps of definition. Solberg and incidence have exposed the underlying conundrums of definition. Solberg et al maintained that 76% of young adults in this country (about 110 million) have one or more signs of TMD diagnosed during research dental examinations. But 74% of this cohort are totally unaware of their gnathologic dangers because they are asymptomatic. Epidemiologists estimated that at present, 5%-6.7% of the adult population needs treatment. The average age of this endangered population is 34 years, over 85%-90% of whom are women. The disease virtually disappears after age 60, an interesting phenomenon of obvious research potential. McNeill speculated, “...that older subjects are less bothered by their symptoms.” Both clinical and socioeconomic considerations direct neurological interest to the TMD problem that is estimated to consume about 32 billion dollars annually in the United States.

Gnathologic prehistory: archeozoic disc erosion BC (before Costen)
Between 1887 and 1929, surgical meniscectomies began to be performed to relieve TMD pain and jaw locking. The authors of several postmortem studies ascribed TMJ pain to perforations of the articular disc that were traumatised by backward pressure from the mandibular condyle. Prentiss, an anatomist, provided a pre-Model A Ford simile that

Table 1: Evolution of TMD nomenclature

<table>
<thead>
<tr>
<th>Year</th>
<th>Name(s) of syndrome and reference</th>
<th>Abbreviation(s)</th>
</tr>
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<tbody>
<tr>
<td>1934–1937</td>
<td>COSTEN’S (COSTEN) SYNDROME</td>
<td>None</td>
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<tr>
<td>1948</td>
<td>TEMPOROMANDIBULAR JOINT OVERCLOSURE</td>
<td>TMJ</td>
</tr>
<tr>
<td>1955</td>
<td>TM J PAIN-DYSFUNCTION SYNDROME OF SCHWARTZ</td>
<td>PDS, TMPDS</td>
</tr>
<tr>
<td>1959</td>
<td>TEMPOROMANDIBULAR JOINT DYSFUNCTION</td>
<td>TMD</td>
</tr>
<tr>
<td>1963</td>
<td>TEMPOROMANDIBULAR DISORDER</td>
<td>TMS</td>
</tr>
<tr>
<td>1969</td>
<td>MYOFASCIAL PAIN DYSFUNCTION SYNDROME</td>
<td>MPD, MPDS</td>
</tr>
<tr>
<td>1973</td>
<td>MANDIBULAR PAIN DYSFUNCTION SYNDROME</td>
<td>MDP</td>
</tr>
<tr>
<td>1973</td>
<td>TEMPOROMANDIBULAR JOINT PAIN</td>
<td>TMJ - PDS</td>
</tr>
<tr>
<td>1989</td>
<td>MANDIBULAR WHIPLASH</td>
<td>None</td>
</tr>
<tr>
<td>1990</td>
<td>CRANIOMANDIBULAR DISORDER</td>
<td>CMD</td>
</tr>
<tr>
<td>1992</td>
<td>TM J PAIN AND DYSFUNCTION SYNDROME</td>
<td>TMPDS</td>
</tr>
<tr>
<td>1993</td>
<td>TM J WHIPLASH</td>
<td>None</td>
</tr>
</tbody>
</table>
Table 2* Evolution of TMD: clinical symptoms 1887–1956

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annandale T 1887</td>
<td>TMJ Pain during chewing, joint fixation pain during chewing, “clicking” type jaw sounds</td>
</tr>
<tr>
<td>Lanz W 1909</td>
<td>Jaw pain during chewing, “clicking” of the jaw</td>
</tr>
<tr>
<td>Summa R 1918</td>
<td>TMJ Pain, mastication weakness</td>
</tr>
<tr>
<td>Pringle J 1918</td>
<td>TMJ Pain, locking of the joint</td>
</tr>
<tr>
<td>Wright WH 1920</td>
<td>TMJ Pain, “traumatic deafness,” weakness of deglutition, deafness, tinnitus, mental torpor</td>
</tr>
<tr>
<td>Monson GS 1921</td>
<td>Weakness of deglutition, deafness, tinnitus, mental torpor</td>
</tr>
<tr>
<td>Decker JC 1925</td>
<td>TMJ “Grinding and cracking,” low pitched tinnitus, deafness</td>
</tr>
<tr>
<td>Wakeley CPG 1929</td>
<td>TMJ Pain, deafness, vertigo, speech defects, personality changes</td>
</tr>
<tr>
<td>Goodfriend DJ 1933</td>
<td>Deafness, vertigo, speech defects, personality changes</td>
</tr>
<tr>
<td>Riesner SE 1936</td>
<td>Facial sagging and deformities from condylar slippage, “personality departures,” deafness, tinnitus, vertigo</td>
</tr>
<tr>
<td>Seaver EP 1937</td>
<td>“Costen’s syndrome,” (also) TMJ subluxation, “popping jaws”</td>
</tr>
<tr>
<td>Summa R 1934,1937,1939</td>
<td>Deafness, stuffy ears at mealtime, snapping noises on chewing, auricular and periauricular pain, low pitched tinnitus, vertigo, “sinus symptoms,” vertex and occipital headache, end of day headache, burning skin at side of nose, pain and paraesthesia “in distribution of chorda tympani,” pain anterior to 2/3 tongue, trismus, limited TMJ opening</td>
</tr>
<tr>
<td>Yule OJ 1937</td>
<td>Facial measurements, palpate condylar movements</td>
</tr>
<tr>
<td>Schuyler CH 1939</td>
<td>Trigeminal neuralgia</td>
</tr>
<tr>
<td>Dingman RO 1940</td>
<td>TMJ Pain, inefficient chewing</td>
</tr>
<tr>
<td>Block LS 1947</td>
<td>“Costen (sic) syndrome”</td>
</tr>
<tr>
<td>Richer H 1948</td>
<td>TMJ Crepitus, locking, pain on opening</td>
</tr>
<tr>
<td>Kiehn GL 1952</td>
<td>Pain in muscles of mastication, tension, anxiety, neuroses</td>
</tr>
<tr>
<td>Schwartz LL 1955,1956</td>
<td>Deafness, stuffy ears at mealtime, snapping noises on chewing, auricular and periauricular pain, low pitched tinnitus, vertigo, “sinus symptoms,” vertex and occipital headache, end of day headache, burning skin at side of nose, pain and paraesthesia “in distribution of chorda tympani,” pain anterior to 2/3 tongue, trismus, limited TMJ opening</td>
</tr>
</tbody>
</table>

*Table 2 is intended only to demonstrate general evolutionary trends of TMD symptoms and may not cite all authors who originally described them. Some of these authors disagreed with the specificity of the signs listed.

compared symptoms from a diseased TMJ “...to the result of a runaway when the bunny is destroyed and the individual, entangled in the lines, is bumped over the ground.” But Connors32 repeated Prentiss’ work and concluded just the opposite; the articular discs and soft tissues were neither thinned nor perforated, but rather hypertrophied; and what thinning existed was a congenital variation. Other early workers hypothesised that the tympanic plate of the glenoid fossa was eroded by the mandibular condyle that was forced backward secondary to a “closed bite,” the consequence of missing posterior teeth.24,25 Monson25 in 1921 wrote that the condyle “...encroaches upon the external auditory meatus, and often causes a resultant defect in hearing in a degree proportionate to the amount of encroachment.” Using dental models and cadaver dissections, but with no anatomical or clinical correlates, Goodfriend23 provided a more elaborate story of pathogenesis: “The joint may ‘rust in’ overnight ...psychological findings demonstrate that there is an association of malocclusions with defective speech. The deepening of the fossa and deformation of the condyle are anatomical alterations which involve the ear and eustachian tube...”

The Costen era: a Camelot age of authoritarian security (tables 2 and 3)

In his most often cited paper Costen2 described...
11 patients with symptoms of deafness, tinnitus, and vertigo. Later he added 52 additional cases of deafness or vertigo, and 94 of “neuralgic pain and headache.” These patients also had facial paraesthesiae, glossodynia, and trismus. However, the drawings used to justify his theories of bony erosion and irritation of adjacent nerves were anatomically incorrect (see below). Absent the burden of providing clinical-pathological correlation, he concluded, “The anatomic explanation...is fairly simple....deep erosion leaves only a thin plate between the condyles and dura—practically nil...all soft tissues next to the eustachian tube were seen to wrinkle and close the tube firmly, permitting close contact of the condyle with the dura...” His therapeutic recommendations included insufflation of the eustachian tube, and “opening the bite” by the use of built up dentures, inlays, bridges, biteguards, tongue depressors, and gauze pads clenched between the teeth.

This halcyon epoch was documented by enthusiastic endorsement of Costen supporters for over a decade. In 1931, Yule concluded that TMD could produce personality disturbances and “reflex neurosis.” Edmund noted, “The favorable results obtained from increasing the vertical dimension of the jaws...are now a matter of record. Thus, we are also able to improve the wry physiognomy of a patient whose chin almost touches the tip of his nose.” In 1937, Schultz disapproved of the then traditional method consisting of retracting a wandering condyle with a steel headband or wiring the jaws shut for months. His novel invention was to sclerose the TMJ tissues with injections of sodium psylliate, an over the counter bulk laxative. Early on, Chor, a neurologist, cast the first critical gauntlet to challenge Costen. He cautioned that, “...the alleged [TMJ] syndrome be considered carefully in the light of well established syndromes, or syndrome complexes, which they may resemble...the literature is replete with bald statements and hypotheses...such theories are of little value unless substantiated by experimental facts...as a result of a more critical attitude, a great deal of misguided therapy may be prevented.” This admonition was followed by a report by Schuyler who cautioned concerning complications from “opening the bite” by grinding occlusal surfaces; and constructing onlays, inlays, crowns, and bridges. “These dental therapies had deleterious effects from alveolar bone and tooth root absorption accompanied by severe pain, anxiety, bruxism, and weight loss.

Landmark anatomical investigations in the 1940s finally put Costen’s theory to rest. Dingman stated that his colleagues had examined 16,000 skulls at the Smithsonian Institute and found no thinning of the glenoid fossa, including skulls with missing posterior teeth (the latter should have “closed the bite” and therefore stressed the TMJ). Two postmortem studies discovered no perforations of articular discs, no thinning of the tympanic plate of the temporal bone, no bony erosions of the external auditory meatus, and no folding or occlusions of eustachian tubes. Based on audiometric and anatomical studies, Shapiro and Truex pointed out that, “If one realizes that most patients with overbite and loss of high tones fall into a group in which such ear symptoms are expected, the age factor no longer remains merely an interesting and coincidental observation.” Looking back on the Costen era from a 1989 perspective, Bell wrote, “By viewing the half century since Costen...the dental profession has come a long way—from a state of nearly total unawareness of the significance of temporomandibular disorders to nearly obsessive concern.”

**An alternative malady: occlusive disharmony**

During the Costen era, many deviant dentists advocated attainment of “occlusive harmony” by bite adjustments usually accomplished by generous grinding of dental cusps. This seemingly benign practice has remained in the dental armamentarium even though it was proved ineffective in several recent reviews. Seligman and Pullinger in 1991 concluded, “Controlled studies fail to demonstrate any association between occlusal interferences and TMD signs or symptoms.”

**Post-Costen era: muscular mechanisms and collateral confusion**

The temporomandibular pain and dysfunction syndrome (PDS or TMPDS, table 1) was the first direct investigative challenge to Costen’s joint erosion theory by an organised clinical research programme. The central conceptual conflicts of this new myogenic pain hypothesis were whether the pain was “organic” or “psychogenic.”

**PSYCHOPHIC Jaw Muscle TMD**

Schwartz, a dentist, headed a multidisciplinary TMD clinic where over 500 patients were treated. His hypothesis was that TMD symptoms originated in mandibular muscles that went through three pathological phases: (1) Early incoordination of muscles producing joint clicking and recurrent subluxation. (2) A middle phase of limitation of mandibular movements by muscle spasm. (3) A final phase of muscle shortening and fibrosis, often irreversible. Psychogenic causes were the most common. Schwartz wrote, “Psychiatric examinations...disclosed all the group...examined to be highly tense individuals with noticeable oral habits.” Early EMG studies supported the concepts of Schwartz; however, later well conceived studies proved surface EMG to be useless.

Over the next 35 years, the Schwartz advocates studied other large TMD cohorts and drew these conclusions: (1) Over 85% of subjects were women, 80% of whom have histories of stress, depression, daytime tooth clenching, and nocturnal bruxism. (2) The largest number of patients had other psychogenic disorders, along with atypical pain syndromes and low pain thresholds. (3) Antidepressant medications were far superior.
to placebo or bite guard prostheses. (4) Prognosis was more favourable in those with recent stress and no operations. (5) Psychological counselling gave excellent results. (6) Those examined a year after diagnosis showed 90% improvement with loss of abnormal jaw sounds in over 80%. (7) Patients with TMD and normal TMJs have higher psychometric scores denoting pain, chronic disablement, and depression.51-55

In a rigorously structured interview study of 50 chronic patients with TMD in which DSM-III-R psychiatric diagnostic criteria were used, Kinney et al discovered the prevalences of psychogenic disease to be two to 10 times higher than those in the general population. The lifelong rate for at least one axis I clinical diagnosis was 86%, and 46% met the criteria for two or more. In addition to somatiform disorders (50%) and somatoform pain (40%), more serious axis I conditions identified were: affective disorder (78%) including major depression (74%), anxiety disorders (24%), and substance misuse (30%). Compared with the lifetime rates just summarised, 46% had current axis I disorders and the majority major depression. Forty per cent had axis II personality disorders: paranoid (18%), obsessive-compulsive (10%), borderline (10%), and histrionic (8%). These are more than twice the prevalences in the general population.

"ORGANIC" JAW MUSCLE TMD

Opponents of the psychogenic theory claimed that "organic" TMJ derangements were the primary sources of pain, muscular spasm, and shortening. In psychometric testing, Patients with TMD had less than expected anxiety, neuroses, and depression; and no correlation was found with parental bonding indices.7 The PDS or TMFDS muscle spasm aetiology of Schwartz was challenged by authors of two chapters in a recent review of TMD research.50 Lund stated that the "vicious cycle" hypothesis of muscle pain and spasm had no basis in fact, "...neither the originator (Travell) nor her collaborators offered direct proof...The idea that TMD is a single progressive disorder...is the end product of this line of speculation." Rudy and Hussein agreed, stating that psychological myogenic factors alone do not explain TMD.

Return of the meandering meniscus

From relatively tentative 19th century speculations, the precept of articular disc disease regained great popularity during the 1950s. Dingman and Moormann recommended complete resection of the offending articular meniscus. Extending this era of ablative surgery, Henny and Baldridge introduced the more definitive procedure of amputating condylar heads while leaving the discs intact. They advocated this radical technique because after meniscectomy, "...many patients develop recurrence of symptoms at a later date." Dentists then recommended holding the displaced disc in place by bite guards and bite planes, procedures still in general use. Joint sounds, the most common sign of TMD, are said by many TMD experts to result from disc displacement. Rinchuse et al reported that these sounds ("clicks and pops") existed in 14%-65% of the general population, depending on whether or not a stethoscope was used for diagnosis. They concluded that "...there is no conclusive scientific evidence...that a patient with TMJ clicking who has no other symptoms would be better off treated...the pathogenesis and ontogeny of TMJ sounds are not known."

Many reports question the utility of TMJ imaging studies because 30% of normal people have disc displacements and joint arthrosis (degenerative processes affecting the TMJ) is usually benign.50-51 Postmortem examinations of a total of 140 persons (dental histories unknown) showed that 40%-80% had joint pathology or disc displacements.52 The relevance of bony joint arthrosis was also disputed by evidence that patients with TMJ rheumatoid arthritic pathology actually had fewer symptoms than normal subjects.53 Alling stated that symptoms associated with degenerative joint changes in young women tended to clear spontaneously in 2 years.

Surgery to "recapture the articular disc" has been proved unsuccessful because the disc often returns to its original position after surgery; and as summarised above, discs "displaced" anterior to the condyle are usually normal.54 Disc replacements using plastic implants were followed by 6%-75% complication rates that included ankylosis, bone necrosis, foreign body reactions, condylar osteophytosis, and osteoarthritis.55 In the foreword of a book reviewing TMD research, Loe described the chronic pain and anguish of patients whose implants failed. One said, "My life hasn't changed, its gone. I feel like a big blob of pain, with big burning hot screws constantly twising into my skull bone in front of my ears." In the same volume, Laskin and Dolwick concluded, "Although surgery is frequently used...a review of the literature provided little objective evidence regarding the efficacy of most of these procedures."

The test's the thing

Various early diagnostic aids included an "ear test" to see if the mouth would drop open when a ticking watch was distanced from the ear, insertion of fingers into ear canals to detect "clicks" during jaw movements, eustachian tube insufflation to evaluate vertigo, comparative models of dental occlusion (gnathic orthomorphos), and trials of cork wedges to predict success of "opening the bite.22 23 25 27 More advanced armamentaria included radiographs of the TMJ using routine, transcranial, oblique views, and laminographic studies, wax casts and dentures to check results, as well as trial appliances and splints. A reawakening of the articular disc theory in the 1950s spawned a cornucopia of controversial state of the art diagnostic tests. This led to the appointment of two blue ribbon study groups by the Canadian Dental Association and by the American Dental Association. Readily proved unreliable were: surface and needle EMG of mastication muscles, CT and MRI of the TMJ, dynamic arthography, mandibular kinesiography, silent
period durations, thermography, sonography, electrovibratography, and electrostimulation (TENS) devices.

**Mandibular whiplash: litigious profit in the neck kink**

The term “whiplash” was first used in 1928 and became a popular medical term after Gay and Abbott’s publication concerning rear end motor vehicle accidents in which the authors mistakenly concluded that the subject’s head is initially driven forward.72 “Cervical strain” as a cause of TMD was described by Roydhouse84 in a short letter to the editor. However, the terms “mandibular whiplash” and “TMJ whiplash” came later (table 1). Lader’s 1983 article65 was “...written for the purpose of providing insurance companies with an explanation concerning the mechanism by which trauma to the cervical area may result in the development of a temporomandibular joint dysfunction problem.” There rapidly followed a large amount of literature in assent to the Roydhouse precepts with only a rare dissent.80–89 During this time frame, several reports claimed that MRI of the TMJ was diagnostic of mandibular or TMJ whiplash; however, such uncontrolled retrospective study series must be judged to have doubtful reliability.80–97

Brooke and Stenn68 reported that patients with post-traumatic TMD have a poor prognosis for recovery compared with non-traumatic TMD. The authors stated that: “Reasons for this difference...may be a consequence of litigation and, in addition, may be due to the personality of the patient.” Some authors reported that some patients claimed the onset of symptoms days or weeks after the professed whiplash incident with diagnoses and treatment beginning even later.12–56 “No pathophysiologcal explanation has been provided for this magically retarded evolution of the TMJ malady from a region supplied by a luxuriant network of A-δ and polymodal C fibres.70–71 Obviously, direct trauma to the jaw region by steering wheel, fist, or bat will produce acute pain and there is no doubt that severe trauma and other pathological processes that disrupt the anatomy of the TMJ can sometimes produce symptoms. Olin,73 an oral-maxillofacial surgeon recently affirms, “The onset of signs and symptoms should appear soon after the trauma and should be relevant to the trauma.”

Thorough acceleration-deceleration studies on human volunteers concluded that the force of a low velocity extension-flexion injury is less than the forces exerted by normal mastication.73 Similar extensive experiments on human subjects sponsored by the Society of Automotive Engineers concluded, “...no jaw motion relative to the cranium was seen for any human subject during rear-end impacts.”74–76 In 1993, The American Academy of Orofacial Pain published their official opinion of mandibular whiplash, “Thus, the condition of mandibular strain at the time of a motor vehicular accident, without a direct blow to the mandible, resulting in hyperextension of the mandibular capsule, ligaments, and masticatory muscles is questionable.”89 Sceptical neurologists must suspect that “TMJ whiplash” is often a clinical manifestation of malingering.

**Extraordinary TMD symptoms and claims**

Clinics and treatment centres for TMD advertise extensively in brochures, flyers, electronic media, symposia, and course summaries as well as in non-peer reviewed periodicals. These inform that serious diverse symptoms from many organ systems can devolve from TMD. Among those listed are: sullen and violent behaviour in preschool children, poor personal image from rounded shoulders, major depression requiring cingulotomy, temporary amnesia, intractable migraine, sagittal suture tenderness, carotid artery ischaemia, bulging erythematous eyes and photosensitivity, abnormal lingual movements and dysphagia, painful chewing and swallowing, laryngitis and chronic cough, scoliosis and tilted pelvis with “short leg,” menstrual cramps with bloating and bleeding, etc. There are also remarkable therapeutic claims including cure of infertility by proper jaw alignment, significant improvement in long distance running and football place kicking, heightened IQ scores all the way from retardation to being a gifted student, amelioration of hypertension, etc.

**Comments**

A consensus of disbelief is evolving. Turk et al40 declared, “Although TMDs have been reported for over six decades, there is a lack of consensus regarding what constitutes a clinically significant syndrome...Cases and controls are distinguished most readily by reports of pain, pain in response to palpation of muscles, restricted vertical range of motion of the mandible, and clicking jaw sounds...In short, many of the most common presenting symptoms and signs associated with TMD are quite common in asymptomatic individuals....” The failure of TMD to quality as a verifiable science was voiced by Weinberg and Lager49 in their study of 138 patients with TMD, “The scientific method cannot be applied to TMD patients because of the impossibility of isolating variables and due to the multicausality of the syndrome.” In a treatise describing drug therapy for TMD, Denucci et al47 stated, “It is now recognised...that many putative dental and surgical therapies for chronic orofacial pain have not withstood the scientific scrutiny of well controlled clinical trials...it would appear that patients with TMD have pain not unlike that observed in other chronic pain conditions.”

Persistently sceptical neurologists have argued that the TMD is a vaguely defined and overly diagnosed “pseudosyndrome” similar to thoracic outlet syndrome, generalised back pain, and coccydynia.48 49 54 A critical evolution toward conservatism in the dental literature is summarised in conclusions of the 1996 NIH Technology Assessment Conference:100 “There are significant problems with the present diagnostic classification...Consensus has not been developed...including which TMD problems should be treated....The preponderance of the
data does not support...any method of initial management.... Moreover, the superiority of such methods to placebo controls and no treatment controls remains undetermined.... Although clinical observation cannot dictate direction, these insights must be followed by rigorous scientific evaluation...”

We have been unable to discover any scientifically valid justification for the concept that symptoms in the region of the jaw joints comprise any more coherent meaningful or useful pathophysiological syndromes(s) or diagnostic entity(ies) than do nomenclature bellyache or backache. Most distressing for patients, there are no reliable controlled outcome studies of the undiminished varieties of either traditional or radical therapeutic programmes. The TMD family of labels is reminiscent of other antiquated medical terms such as “chilblain” and “miasma” that now have no utility in rational medical practice. We hope that programmed apoplectic will continue to discipline and constrict this confusing anatomically focused diagnostic nomenclature. We think that traditional therapeutic programmes will be served best by clinicians who are especially competent in the diagnosis and treatment of somatisation disorders, depression, anxiety, and substance misuse, as well as the pathophysiology of chronic pain syndromes.

We thank our colleagues who gave us much professional help and encouragement, even though they may not have always agreed with our conclusions: Robert R Am, John E Dodes, Herbert L Goldberg, and Myer S Leonard. Special thanks to our colleagues who gave us much professional help and encouragement, even though they may not have always agreed with our conclusions: Robert R N Arm, John E Dodes, Herbert L Goldberg, and Myer S Leonard. Special thanks to our colleagues who gave us much professional help and encouragement, even though they may not have always agreed with our conclusions: Robert R Arm, John E Dodes, Herbert L Goldberg, and Myer S Leonard. Special thanks to

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