Segmental definition: Part I. A focal point for diagnosis of somatic dysfunction

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This paper gives a scientific foundation for palpatory diagnosis through the description of specific palpatory tests and the use of criteria for judging the responses to the tests. In the descriptive and clinical research studies summarized here, the somatic structures are examined as a mobile system. A series of rotary and translatable movement tests provide a pattern for examination of function of mobile bony segments. A primary segmental dysfunction is identified by asymmetric responses to opposing directions of each motion test and the presence of mirror image asymmetries in immediately adjacent segments. Thus, the location of segmental dysfunction is verified by comparison of asymmetries within a dysfunctional unit involving three spinal segments. The diagnostic site of the primary segmental asymmetry provides the physician with a specific locus for treatment of somatic dysfunction. New information from the motion tests suggests reassessment of previously held concepts of segmental dysfunction.

A clearly defined focus for the principled application of an osteopathic manipulative procedure has been historically elusive. Careful definition of the location and distinguishing characteristics of those segmental aspects of somatic dysfunction fundamental to the clinical use of osteopathic manipulative procedures is needed. In palpatory diagnosis, a precise spinal segmental level is lo-

calized where the findings of local dysfunction are more noticeable and more clinically significant. In the past, descriptions of the palpable features of segmental dysfunction have included alterations in bony position, soft tissue fluid content, muscular tone, and joint mobility¹. In view of the frequency with which these kinds of changes, individually, can be palpated throughout the musculoskeletal system, the terms have been somewhat nonspecific. The problem is to distinguish the many areas of compensatory (secondary) or minor alterations from those fewer primary areas of more major dysfunction that are focused at precise spinal levels. Terms such as "arthritis," "myositis," "fasciitis," and "neuritis" are convenient to refer to inflammation in separate tissue components of the somatic system, but they are inadequate to describe the clinical condition reflected by disturbance in each of these tissues, in concert, at a dysfunctional spinal segment.

This report has been organized from mainly descriptive clinical investigations which formed the basis for studies with instrumental measurements at Michigan State University—College of Osteopathic Medicine and at Chicago College of Osteopathic Medicine. Part I concentrates on the way the body's response is monitored during selected movement tests to elicit more precise diagnostic signs of the segmental dysfunction as shown by palpable features of altered mobility. Information about asymmetries in segmental motion help to clarify a basis for the procedural approaches to osteopathic manipulative management of somatic dysfunction; an example will be explored in Part II. Finally, in Part III, segmental definition^{2,3} of asymmetric response to motion tests will be used to facilitate the differential diagnosis of somatic dysfunction, and to distinguish somatic findings of somatic reflex origin from those of visceral reflex origin.

Fig. 1. A coordinate system to illustrate straight-line directions of movement, used in description of translatory motion tests.

Fig. 2. A coordinate system to illustrate directions of movement about axes, used in description of rotary motion tests.

Procedure

In formulating tests of the body's movement system, attention first was given to a classification of body movements whose eventual description would be in relation to the coordinates or axes about which movement of an object can be described. There are three degrees of freedom for translatory movement (Fig. 1). These include straight-line movements along the coordinates z (up/down), y (right/left), and x (anterior/posterior). In addition,

there are three degrees of freedom for rotations, as illustrated in Figure 2: axial rotation about the vertical z axis; forward flexion/extension about y ; sidebending or lateral flexion about the x axis. These six kinds of passive gross movement are the "tools" used for testing the body's response to movement.

Such a classification provides a foundation for improving clinical communication about tests for symmetry and range of body movement. Basic modes of everyday body movement can be reproduced in a series of tests for obtaining clinical information, not only about overall performance of the whole movement,⁴ but especially about the local behavior of the mobile parts during the movement.⁵ Passive movement, introduced by the examiner, is used (1) to minimize active interference by the patient, (2) standardize the procedure for the examiner, and (3) contribute to reliability for clinical use and for research.

In the description of these test procedures, certain points of reference are necessary. The position of the subject must be clearly established (seated, supine, or other). Where is motion being introduced by the examiner—for example, through contact at the head, shoulders, extremities? Where does the examiner stand in relation to the subject? Where is palpatory contact established for monitoring a segment's response to the motion being introduced?

In testing the response of each body region (rather than each segment) to gross motions, the criteria for a palpable finding of asymmetry are not measured visually at the endpoint of range, *after* the response has taken place.⁴ Instead, criteria have been established for the palpable quality of the response within the range, and for the quality of the endpoint. Resistance to gross movement, palpated earlier in one direction than in the opposite, is a tentative sign of the presence of dysfunction in the somatic region being tested.^{6,7} These regional tests screen overall response to motion for a first impression of the presence of a clinical problem in the mobile system.

When passive gross motion tests are applied to monitor behavior of a segment, the criteria for response are concerned with palpable findings at a single mobile segment. The bony part moves in a manner that is controlled by the feedback of information about the quality of that response from its own adnexal tissues and adjacent segments.⁸ On the basis of this feedback, the response is self-correcting for minor readjustments, *while the movement is occurring*, so that the resulting behavior of the mobile segment will match the demands of the whole movement.

From an appreciation of this feedback mecha-

nism, Bowles⁹ implied that the tissues about a moving bony part constantly reflect its behavior, as they comply with demands for movement and positioning of the whole system. Because the segmental tissues reflect the behavior of this mobile segment, they are monitored for their palpable expression of compliance with, or resistance to, the movement being initiated. Thumb and second or third finger of the palpating hand straddle the bony vertebral segment to contact tissues bilaterally overlying the transverse processes, palpating the vertebral segment as a single mobile unit. Contact is light, but the pressure is sufficient to distinguish the degree of resistance to pressure reflected by deep muscular tissues; the degree of resistance may vary from segment to segment. When motion is initiated, the examiner notes whether the segmental tissues comply easily or reflect an increasing degree of resistance.

These criteria have been applied to make decisions on the specific asymmetries at a diagnosed dysfunctional segment, that is, a segment showing signs of dysfunction in its altered bony position and deep tissue tone. (Contact may be equally appropriate at the spinous process, as a bony contact for monitoring the mobile unit. Criteria at the bony point of contact are the same, that is, a sense of compliance versus resistance in response to initial directions of movement; however, the palpable sense of bony response to motion might be better described as structural mobility versus rigidity.) For example, with the patient seated during axial rotation right introduced through the shoulders (Fig. 3), the palpating left hand can monitor at T6 for that segment's response during rotation right and compare it with the response during rotation left. Hand placements are often switched for testing rotation left.

Observations

The characteristics of a segment's easy response to a normal demand for movement can be illustrated in Figure 4, which diagrammatically represents only axial rotation to right and left. First of all, with the patient seated, and before motion is introduced, the palpable resistance to pressure (tonicity) of tissues at this thoracic spinal segment is a uniform basal level necessary to stabilize its resting position in a given postural pattern. The tissue tone is not palpably different from that of surrounding segments (indicated at intersect *x* on the baseline).

The nonlesioned segment responds to initial rotation of the shoulders and trunk with very little palpable change; there is little perceptible sense of increasing/decreasing resistance in the initial response. As the endpoint of range approaches, reflex

Fig. 3. The subject is seated, with arms folded: At a defined mobile segment, (indicated by marker at T6) the examiner's left hand contacts the paravertebral tissues bilaterally. Left thumb and forefinger monitor response to axial rotation of the shoulders and trunk to the right, introduced by the examiner's right hand at the subject's right elbow. (Choice of monitoring fingers may vary among examiners, and may change in different spinal regions.)

Fig. 4. Schematic representation for symmetric response to motion at a nonlesioned thoracic segment: Only axial rotation is represented. The tone scale registers changes in tension in underlying segmental soft tissues, reflected to palpating fingers as increasing (or decreasing) resistance to pressure. Diagram shows equal initial compliance to right and left, and then increasing resistance toward equidistant final anatomic endpoints.

mechanisms are aroused by tissue stretch and structural positional stress. Tissues increase their resistance in a protective phase that reaches its peak at the point of physiologic and then anatomic barriers. So, first there is a palpable sense of early

Fig. 5. Schematic representation for asymmetric behavior at a dysfunctional (lesioned) segment: Palpable increase in resistance to pressure (at rest) reflects an elevated level on the tension scale at X1. As indicated, immediately increasing resistance is met in the direction toward the barrier sensed in rotation to the right, and immediately decreasing resistance, toward the left. Fig. 6. Schematic representation for response to rotation tests at adjacent segments: Secondary lesioned behavior is indicated by an asymmetry with increasing resistance toward the left and increasing compliance toward the right, that is, in opposing directions to the example of primary dysfunction illustrated in Figure 5.

compliance, then an eventual endpoint resistance. During gross axial rotation, similar segmental responses to rotation right and left express a functional symmetry.

The example in Figure 4 serves as a graphic representation for all six kinds of movement; the proportions of the compliance and resistance phases will vary with different movements and in the different regions of the body. Both bony and soft tissues of a segment reflect these palpable cues about compliance and resistance during tests for response to motion. This example describes symmetric, or *nonlesioned*, segmental behavior.

The characteristics of *lesioned* behavior in response to movement are illustrated in Figure 5. The resting tissues at a lesioned segment express the disturbed function even before movement is initiated, as demonstrated by their increased resistance to palpatory pressure; this is illustrated on the diagram by the higher position on the tension scale at x1. During rotation right in this example, there may be no compliant phase; the tissues express an immediately increasing resistance, with the endpoint arriving soon. During rotation left, the tissues express an immediately decreasing resistance and increasing compliance; the endpoint in this opposing direction comes much later.

As an example, again, Figure 5 represents graphically the asymmetric response to motion that is palpable at a lesioned segment during all six directions of motion tested (three rotations and three translations). Our investigations indicate that segmental dysfunction is expressed by a *complete* asymmetry in the six degrees of freedom.

This dysfunction does not exist as an isolated finding among normally functioning segments. Immediately adjacent segments also show a palpable increase in tissue resistance to some directions of

initial movement.^{3,10} As indicated in Figure 6, the tissue resistance of the two adjacent segments (at rest) is not quite as high on the tension scale at x2. Also, they express asymmetry in response to motion, but, significantly, the resistance is in directions of motion opposite those of the more centrally located lesioned segment. At the adjacent segments, their increasing resistance is in response to rotation left rather than right in the central segment, and the tissues demonstrate initial increasing compliance during rotation right. There is, predictably, a complete opposing asymmetry in response to tests of all six degrees of freedom, when the segments immediately above and below a primary lesioned segment are examined.

Figure 7 illustrates the asymmetry throughout a fundamental unit of spinal segmental dysfunction. There is increasing tissue resistance to rotation right at the location of the primary motor asymmetry indicated at T6, with opposing asymmetries resisting rotation left at adjacent T5 and T7. Again, only axial rotation is indicated. When multiplied by six for the six degrees of limited freedom—three rotations and three translations—it becomes apparent that motion testing provides six variables to describe the individuality of a given unit of segmental dysfunction. Comparison testing for the mirror-image findings at adjacent segments confirms the location of the primary central segment for the medical record.

Discussion

The term "segmental definition"² refers to the final stage, or level 3, in an orderly sequence of diagnostic tests for somatic dysfunction. This sequencing of visual and palpatory tests for examining the somatic system is modeled after the classic approach for observing each of the body's systemic

Fig. 7. Schematic representation of a fundamental unit of segmental dysfunction: A primary motor asymmetry is indicated as the central segment T6, which resists rotation right, with opposing asymmetries at adjacent segments, which resist rotation left.

Fig. 8. A concept of mobile segments operating individually, and in concert, in response to demand for a gross movement within a mobile system.

functions during the regional physical examination.

In the somatic model, level 1 tests are used to screen for signs of asymmetry of structure, tissue, and motion throughout the body to determine if initial signs of dysfunction are present in the somatic system. Comparable level 1 tests in the cardiac system, for example, would include the initial screen for signs of irregularity of cardiac rate and rhythm and for alteration in the quality of the heart sounds.

Level 2 tests for the somatic system are designed to scan bony structures, soft tissues, and motion properties from point to point throughout a body region (identified in the screen), to determine a precise location of the dysfunction. Level 3 tests of *segmental definition* are used to examine at that location for the individual characteristics of bony malposition, tissue texture abnormality, and motion asymmetry that provide a diagnosis of segmental dysfunction, and justify consideration of treatment.

For perspective at this point, let us recognize that the soma participates in many of the body's *functional* systems: electric energy; fluid conduction; tissue interconnection; and, structural support functions. The diagnostic approach outlined here relates to the soma in its primary role as the body's skeletal movement system, and a test pattern for investigating aspects of that movement function is described. As introduced, the report only briefly references the kinds of diagnostic tests that screen the body for impressions of the presence of somatic dysfunction, or those that scan from point to point

for precise location of dysfunction. Mainly, it details a framework of tests for defining the motion characteristics at a dysfunctional mobile segment.

The anatomic concept of a joint—one bone in relation to the one below—has often been useful for describing palpable findings of disturbed position and mobility at a spinal joint, but it has inherent limitations for study of segmental motion. Although the idea of body movement requires speculation about relationships of one bony part to another, the joint concept does not easily convey the functional dynamics of reception of stimuli, motor controls, and the part-to-whole relations that are basic to a human movement system. The joint concept may even be somewhat counterproductive, when part-to-whole relations in body movement function are the primary consideration—as they are in the examination procedures described here.

During active clinical practice, the study of this mobile system has involved our initial recognition and then application of the concept involved in the term “system.” The term refers to any grouping of dynamically-related components (in this instance mobile segments) that operate in concert, or in related fashion, for the purpose of achieving a specified goal or set of goals (in this instance, movement). Together, mobile segments respond to a central demand for active whole-body movement. It

Fig. 9. Kimberly's¹¹ diagram reproduced here describes the components of joint motion by the physiologic barrier (resistance) and anatomic barrier reached by bone and ligaments. (From JAOA, April 1980.)

may be to reach for an object; it may be to step and walk, bend and twist, and even breathe and talk. At any particular moment, the demand is for a whole movement pattern, not, for example, in the joint model, for T4 to specifically sidebend and rotate in particular directions on T5. What emerges (Fig. 8) is a series of mobile segments moving in concert, in response to a demand for overall movement that essentially is organized in the central nervous system. A mobile segment can be described as a bone, with articular surfaces for movement, and the adnexal tissues that (1) stabilize position, (2) allow the bone to be moved from one position to another, and (3) initiate and carry out the response to movement.

In systems theory, these mobile segments may be referred to as "subsystems." The response of each mobile bony part is controlled in a major way on a segmental basis by local spinal reflexes of proprioceptive sensory origin.⁸ This sensory input provides ongoing feedback of information needed for fine tuning of the behavior of each subsystem, establishing spinal reflex motor control for each mobile segment to be in the right place at the right time *in its relation to the whole movement pattern being performed*. Significantly, spinal reflexes control (self-correct) each segment's response whether the gross movement is performed actively or within a framework of passive movement tests.

The foregoing description of palpable findings in the passively induced gross motion tests for response at a mobile segment (Fig. 4) adds a dimension of tissue tone (increment of compliance/resistance) to Kimberly's¹¹ more linear diagram of the restrictive barrier, reproduced in Figure 9.

Bowles¹² was particularly fascinated with the hyperbolic aspect of the tissue resistance (bind) palpable during motion tests for the asymmetric behavior at a dysfunctional segment (Fig. 5). He

perceived it as an essential characteristic of disturbed neuromusculoskeletal response that needed major consideration in the development of any research model to simulate motion dysfunction in the human body.¹³

This current report of findings is at some variance with previous concepts of vertebral joint lesioning as being one-sided (as reflected in references to *T4 on the left*, for example). Such concepts have generally emerged within the framework of the joint concept, which considers position and motion of one vertebra's articular facet in relation to the one below to describe palpable findings of restricted joint motion. However, during passive gross motion tests, the asymmetry of segmental dysfunction is palpable *bilaterally*. With the subject seated (Fig. 3), and registering resistance of the mobile unit to rotation of the shoulders and trunk to the right, resistance cues are palpable at tissues overlying both the right and left transverse processes. For this reason, sidedness of resistance is described only in relation to the direction of the motion being tested—for example, resisting rotation right—rather than in relation to left-sided locking or restriction of the joint's facet motion, which implies a conceptual *cause* of the increased resistance being palpated.

Further, the completeness of the pattern of asymmetric response to all six motion tests is at odds with one general classification of lesioned joints: type 1 describes contralateral restriction of lateral flexion and axial rotation, without restriction of either forward flexion or extension; and type 2 is ipsilateral restriction of lateral flexion and axial rotation, when accompanied by restriction of either forward flexion or extension.

The assumption originally made in this classification may have been related to Fryette's¹⁴ statements of physiologic motion in the spine. On face value, *normal* vertebral movement would not seem to offer a predictable basis for expectations of the findings at *dysfunctional* spinal segments. Another possible assumption is that this classification of type 1 and type 2 dysfunctions may have been related originally to group (lateral curve) and single (joint) kinds of spinal findings respectively. Certainly, the findings in a lateral spinal curve usually involve rotation of the bodies of the vertebrae into the convexity (contralateral to the lateral flexion). However, segments in a group curve usually offer a smooth sequence of behavior; they fulfill criteria for their group activity that are quite different from those for the lesioned behavior of individual segments described in this report for a fundamental three-segment unit, where adjacent segments show opposing asymmetries. The uniform segmental ac-

tivities recognized as grouped within lateral spinal convexities are more appropriately classified as primary and secondary scolioses, rather than a series of type 1 dysfunctional segments.

The major issue here is that each fundamental unit of segmental dysfunction has a definable component of forward flexion/extension asymmetry when responses to these opposing directions of a motion test are compared. This feature helps to characterize the dysfunctional unit as totally asymmetric, that is, in all six degrees of freedom.

The definition of specific segmental asymmetries elicited during the test pattern described here begins to provide a description of segmental dysfunction as a clinical focus within the more general diagnostic category of somatic dysfunction. The findings suggest, in turn, a frame of reference for an approach to treatment and the evaluation of response to treatment. These factors will be elaborated in part II of this paper.

I am indebted to Albert F. Kelso, Ph.D., for his comments and assistance.

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CONTRAINDICATIONS: FELDENE (piroxicam) should not be used in patients who have previously exhibited hypersensitivity to it, or in individuals with the syndrome comprised of bronchospasm, nasal polyps and angioedema precipitated by aspirin or other nonsteroidal anti-inflammatory drugs.

WARNINGS: Peptic ulceration, perforation and GI bleeding—sometimes severe, and, in some instances fatal—have been reported with patients receiving FELDENE. If FELDENE must be given to patients with a history of upper gastrointestinal tract disease, the patient should be under close supervision (see ADVERSE REACTIONS).

PRECAUTIONS: Renal Effects. As with other nonsteroidal anti-inflammatory drugs, long-term administration of piroxicam to animals has resulted in renal papillary necrosis and other abnormal renal pathology. In humans, there have been reports of acute interstitial nephritis with hematuria, proteinuria, and occasionally, nephrotic syndrome.

A second form of renal toxicity has been seen in patients with prerenal conditions leading to a reduction in renal blood flow or blood volume, where the renal prostaglandins have a supportive role in the maintenance of renal perfusion. In these patients administration of an NSAID may cause a dose-dependent reduction in prostaglandin formation and may precipitate overt renal decompensation. Patients at greatest risk of this reaction are those with impaired renal function, heart failure, liver dysfunction, those taking diuretics, and the elderly.

Because of extensive renal excretion of piroxicam and its biotransformation products (less than 5% of the daily dose excreted unchanged), lower doses of piroxicam should be anticipated in patients with impaired renal function, and they should be carefully monitored.

Although other nonsteroidal anti-inflammatory drugs do not have the same direct effect on platelets that aspirin does, all drugs inhibiting prostaglandin biosynthesis do interfere with platelet function to some degree.

Because of reports of adverse eye findings with nonsteroidal anti-inflammatory agents, it is recommended that patients who develop visual complaints during treatment with FELDENE have ophthalmic evaluation.

As with other nonsteroidal anti-inflammatory drugs, borderline elevations of one or more liver tests may occur in up to 15% of patients. A patient with symptoms and/or signs suggesting liver dysfunction, or in whom abnormal liver tests have occurred, should be evaluated for evidence of the development of more severe hepatic reaction while on therapy with FELDENE.

Severe hepatic reactions, including jaundice and cases of fatal hepatitis have been reported with FELDENE. Although such reactions are rare, if abnormal liver tests persist or worsen, if clinical signs and symptoms consistent with liver disease develop, or if systemic manifestations occur (e.g., eosinophilia, rash, etc.), FELDENE should be discontinued. (See also ADVERSE REACTIONS.)

Although at the recommended dose of 20 mg/day of FELDENE increased fecal blood loss due to gastrointestinal irritation did not occur, in about 4% of the patients treated with FELDENE alone or concomitantly with aspirin, reductions in hemoglobin and hematocrit values were observed.

Peripheral edema has been observed in approximately 2% of the patients treated with FELDENE. Therefore, FELDENE should be used with caution in patients with heart failure, hypertension or other conditions predisposing to fluid retention.

A combination of dermatological and/or allergic signs and symptoms suggestive of serum sickness have occasionally occurred in conjunction with the use of FELDENE. These include arthralgias, pruritus, fever, fatigue, and rash including vesiculo bullous reactions and exfoliative dermatitis.

DRUG INTERACTIONS: Interactions with coumarin-type anticoagulants have been reported with FELDENE since marketing. Therefore, physicians should closely monitor patients for a change in dosage requirements when administering FELDENE to patients on coumarin-type anticoagulants and other highly protein-bound drugs.

Plasma levels of piroxicam are depressed to approximately 80% of their normal values when FELDENE is administered in conjunction with aspirin (3900 mg/day), but concomitant administration of antacids has no effect on piroxicam plasma levels.

Nonsteroidal anti-inflammatory agents, including FELDENE, have been reported to increase steady state plasma lithium levels. It is recommended that plasma lithium levels be monitored when initiating, adjusting and discontinuing FELDENE.

Carcinogenesis, Chronic Animal Toxicity and Impairment of Fertility: Subacute and chronic toxicity studies have been carried out in rats, mice, dogs, and monkeys.

The pathology most often seen was that characteristically associated with the animal toxicology of anti-inflammatory agents: renal papillary necrosis (see PRECAUTIONS) and gastrointestinal lesions.

In classical studies in laboratory animals, piroxicam did not show any teratogenic potential. Reproductive studies revealed no impairment of fertility in animals.

Pregnancy and Nursing Mothers: Like other drugs which inhibit the synthesis and release of prostaglandins, piroxicam administration continued late into pregnancy increased the incidence of dystocia and delayed parturition in animals. Gastrointestinal tract toxicity was increased in pregnant females in the last trimester of pregnancy compared to non-pregnant females or females in earlier trimesters of pregnancy.

FELDENE is not recommended for use in nursing mothers or in pregnant women because of the animal findings and since safety for such use has not been established in humans.

Use in Children: Dosage recommendations and indications for use in children have not been established.

ADVERSE REACTIONS: Gastrointestinal symptoms are the most prominent side effects, occurring in approximately 20% of the patients, which in most instances did not interfere with the course of therapy. Of the patients experiencing gastrointestinal side effects, approximately 5% discontinued therapy with an overall incidence of peptic ulceration of about 1%.

Adverse reactions are listed below by body system for all patients in clinical trials with FELDENE (piroxicam) at doses of 20 mg/day.

Incidence Greater Than 1%: The following adverse reactions occurred more frequently than 1 in 100.

Gastrointestinal: stomatitis, anorexia, epigastric distress, nausea, constipation, abdominal discomfort, flatulence, diarrhea, abdominal pain, indigestion.

Hematological: decreases in hemoglobin* and hematocrit* (see PRECAUTIONS), anemia, leucopenia, eosinophilia.

Dermatologic: pruritus, rash.

Central Nervous System: dizziness, somnolence, vertigo.

Urogenital: BUN and creatinine elevations (see PRECAUTIONS).

Body as a Whole: headache, malaise.

Special Senses: tinnitus.

Cardiovascular/Respiratory: edema (see PRECAUTIONS).

* Reactions occurring in 3% to 9% of patients treated with FELDENE.

Reactions occurring in 1% to 3% of patients are unmarked.

Incidence Less Than 1% (Causal Relationship Probable): The following adverse reactions occurred less frequently than 1 in 100. The probability exists that there is a causal relationship between FELDENE and these reactions.

Gastrointestinal: liver function abnormalities, jaundice, hepatitis (see PRECAUTIONS), vomiting, hematemesis, melena, gastrointestinal bleeding, perforation and ulceration (see WARNINGS), dry mouth.

Hematological: thrombocytopenia, petechial rash, ecchymosis, bone marrow depression including aplastic anemia, epistaxis.

Dermatologic: sweating, erythema, bruising, desquamation, exfoliative dermatitis, erythema multiforme, toxic epidermal necrolysis, Stevens-Johnson syndrome, vesiculo bullous reactions, photoallergic skin reactions.

Central Nervous System: depression, insomnia, nervousness.

Urogenital: hematuria, proteinuria, interstitial nephritis, renal failure, hyperkalemia, glomerulitis, papillary necrosis, nephrotic syndrome (see PRECAUTIONS).

Body as a Whole: pain (colic), fever, flu-like syndrome (see PRECAUTIONS).

Special Senses: swollen eyes, blurred vision, eye irritations.

Cardiovascular/Respiratory: hypertension, worsening of congestive heart failure (see PRECAUTIONS), exacerbation of angina.

Metabolic: hypoglycemia, hyperglycemia, weight increase, weight decrease.

Hypersensitivity: anaphylaxis, bronchospasm, urticaria/angioedema, vasculitis, "serum sickness" (see PRECAUTIONS).

Incidence Less Than 1% (Causal Relationship Unknown): Other adverse reactions were reported with a frequency of less than 1 in 100, but a causal relationship between FELDENE and the reaction could not be determined.

Gastrointestinal: pancreatitis.

Dermatologic: onycholysis, loss of hair.

Central Nervous System: akathisia, hallucinations, mood alterations, dream abnormalities, mental confusion, paresthesias.

Urogenital System: dysuria.

Body as a Whole: weakness.

Cardiovascular/Respiratory: palpitations, dyspnea.

Hypersensitivity: positive ANA.

Special Senses: hearing impairment.

OVERDOSAGE: In the event treatment for overdosage is required, the long plasma half-life of piroxicam should be considered. The absence of experience with acute overdosage precludes characterization of sequelae and recommendations of specific antidotal efficacy at this time. It is reasonable to assume that the standard measures of gastric evacuation and general supportive therapy would apply. In addition to supportive measures, the use of activated charcoal may effectively reduce the absorption and reabsorption of piroxicam. Experiments in dogs have demonstrated that the use of multiple-dose treatments with activated charcoal could reduce the half-life of piroxicam elimination from 27 hours (without charcoal) to 11 hours and reduce the systemic bioavailability of piroxicam by as much as 37% when activated charcoal is given as late as 6 hours after administration of piroxicam.

ADMINISTRATION AND DOSAGE: Rheumatoid Arthritis, Osteoarthritis: It is recommended that FELDENE therapy be initiated and maintained at a single daily dose of 20 mg. If desired, the daily dose may be divided.

Dosage recommendations and indications for use in children have not been established.

More detailed professional information available on request.

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