JAOA
ORIGINAL CONTRIBUTION

Clinical biomechanic correlates for cervical function: Part III. Intermittent secondary movements

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Parts I and II of this study compared kinematic and myoelectric data for two groups of asymptomatic subjects classified as having symmetric or asymmetric motor response to a palpatory test for cervical sidebending. Kinematic data revealed that asymmetric subjects had limited mobility for primary and secondary motions. Myoelectric activity was slow to be initiated in the asymmetric subjects, and reduced in time and strength of contraction. Part III addresses additional specific kinematic data concerning three-dimensional orientations of the head; however, these data were accumulated throughout the paths of movement, not just at their end points as in our previous work. Although asymmetric subjects had demonstrated significantly reduced range of motion, paths of the more minor secondary axes did not differ significantly between groups. As with previous data (Part I and II), active and passive movements were undifferentiated; this degree of likeness in even minor aspects of the motor performance continues to indicate the remarkable similarities that can exist between primary movements directed and guided by trained physicians and those actively controlled by patients themselves.

Part III completes a study of cervical motor behavior in which a passive gross motion test distinguished an asymmetric group with subclinical motor behavior that has measurable kinematic and myoelectric correlates.

(Key words: biomechanics, human motion behavior, osteopathic medicine, somatic dysfunction, spine)

Clinical problems in which musculoskeletal complaints are associated with asymmetric motor behavior are of interest to healthcare professionals. Osteopathic physicians in particular recognize the diagnostic category somatic dysfunction to describe an important triad of diagnostic signs that accompany musculoskeletal problems: asymmetric behaviors of structure/position, of soft tissue, and of motion. Our studies[1,2] have resulted in instrumental data gathered during repeated motions to better understand asymmetric motor function of the cervical spine.

Our first report[1] presented results of head orientation measurements specifically at the end points of the ranges of motion. The data revealed significant decreases in total primary motion ranges and reduced secondary movements for the group of subjects identified as being asymmetric. A subsequent report[2] docu-

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mented myoelectric data accumulated concurrently with the kinematic data. Those results indicated that muscles of the asymmetric subjects were slower to initiate activity and reduced in time and strength of contraction. The kinematic and myoelectric results described symmetric and asymmetric motor behavior; however, neither report addressed specific kinematic information gained *during* the test procedures.

For the present report, we have further examined head motions of our subject groups. Data concerning three-dimensional orientations of the head were accumulated throughout the ranges of motion, not just at the end points of movement as in our previous work.

Procedural methods

Selection of subjects

The data reported here were gathered concurrently with previously reported kinematic and myoelectric data.[1,2] The sample population consisted of 17 male volunteers from the Michigan State University community. Final selection of the subjects occurred by complete agreement of three independent, trained examiners on the presence of palpable symmetric or right or left asymmetric responses to a passive motion test for cervical sidebending. The symmetric (control) group consisted of six subjects in whom the response to the motion test was diagnosed as symmetric. The asymmetric (experimental) group comprised 11 subjects: 6 with right cervical asymmetry (limited more in response to cervical sidebending right than left), and 5 who showed left cervical asymmetry (limited more during sidebending left than right).

Subjects were not selected on the basis of symptoms, nor were they experiencing any pain on cervical motion. By eliminating the pain variable, we chose to focus attention on more accurately testing motor response without the distortion of subjective hesitation and guarding that accompanies acute musculoskeletal disorders. Our goal was to establish a standard for motor symmetry in the neck region and gather measurable physiologic correlates for palpable motor asymmetry, which is often an early, subclinical sign of somatic dysfunction in a body region.

Testing procedures

Details of the testing procedures have been described in a previous paper.[1] These data were gathered with a mouth-held target system[3] and recorded by a videotaping system. Briefly, six elementary motions (primary rotations) were performed as single and separate entities, with the head returning to a neutral (starting) position after each motion. The definition of a primary rotation was as a motion occurring about one of the following axes: sidebending about the posterior-to-anterior axis (X); forward flexion or backward extension about the right-to-left lateral axis (Y); and axial rotation about the inferior-to-superior axis (Z). The subjects were instructed to perform the motions in the following sequence: forward flexion; backward flexion (hyperextension); sidebending right (flexion right); sidebending left (flexion left); rotation right; and rotation left. The subjects carried out active primary motions guided by their own sense of comfortable end of range. Passive primary motions were introduced and guided by the physician and terminated when a palpable sense of end point of range was achieved.

The three-dimensional orientations of the head for each test motion were accumulated over a period of time starting at a neutral position (the beginning of motion) and stopping at the end of range (referred to as the first half of movement). Once the maximum extent of range was reached, the momentary pause or slight movements or both that occurred during this pause were recorded, but they were deleted from the time-and-distance variables. Finally, a second series of measures was made from the end point of range (or maximum extent of the motion) during the return to neutral or the starting position (the second half of movement).

In the process of the complete excursion, the head moved with a primary rotation about the requested axis with small secondary rotations, typically of 5 degrees occurring about the other two axes. Secondary rotations were interpreted as if they occurred separately. From these kinematic data, we identified the movement variables as they occurred throughout each motion: first, primary rotations that represented the range for each of the intended motions; and second, the two secondary rotations representing unintended deviations from the primary motions (Fig 1).

Data reduction

Primary and secondary motions (positions) were measured at equal time intervals throughout the range. The secondary rotation data were separated into two formats: accumulated secondary motion and averaged secondary motion.

The accumulated (Acc) secondary motion repre-

(continued on page 149)

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INDICATIONS AND USAGE: TENORETIC is indicated in the treatment of hypertension. This fixed dose combination drug is not indicated for initial therapy of hypertension. If the fixed dose combination represents the dose appropriate to the individual patient's needs, it may be more convenient than the separate components.

CONTRAINDICATIONS: TENORETIC is contraindicated in patients with: sinus bradycardia; heart block greater than first degree; cardiogenic shock; overt cardiac failure (see WARNINGS); anuria; hypersensitivity to this product or to sulfonamide-derived drugs.

WARNINGS: Cardiac Failure: Sympathetic stimulation is necessary in supporting circulatory function in congestive heart failure, and beta blockade carries the potential hazard of further depressing myocardial contractility and precipitating more severe failure. In patients who have congestive heart failure controlled by digitalis and/or diuretics, TENORETIC should be administered cautiously. Both digitalis and atenolol slow AV conduction.

IN PATIENTS WITHOUT A HISTORY OF CARDIAC FAILURE, continued depression of the myocardium with beta-blocking agents over a period of time can, in some cases, lead to cardiac failure. At the first sign or symptom of impending cardiac failure, patients receiving TENORETIC should be digitalized and/or be given additional diuretic therapy. Observe the patient closely. If cardiac failure continues despite adequate digitalization and diuretic therapy, TENORETIC therapy should be withdrawn.

Renal and Hepatic Disease and Electrolyte Disturbances: Since atenolol is excreted via the kidneys, TENORETIC should be used with caution in patients with impaired renal function.

In patients with renal disease, thiazides may precipitate azotemia. Since cumulative effects may develop in the presence of impaired renal function, if progressive renal impairment becomes evident, TENORETIC should be discontinued.

In patients with impaired hepatic function or progressive liver disease, minor alterations in fluid and electrolyte balance may precipitate hepatic coma. TENORETIC should be used with caution in these patients.

Ischemic Heart Disease: Following abrupt cessation of therapy with certain beta-blocking agents in patients with coronary artery disease, exacerbations of angina pectoris and, in some cases, myocardial infarction have been reported. Therefore, such patients should be cautioned against interruption of therapy without the physician's advice. Even in the absence of overt angina pectoris, when discontinuation of TENORETIC is planned, the patient should be carefully observed and should be advised to limit physical activity to a minimum. TENORETIC should be reinstated if withdrawal symptoms occur. Because coronary artery disease is common and may be unrecognized, it may be prudent not to discontinue TENORETIC therapy abruptly even in patients treated only for hypertension.

Bronchospastic Diseases: PATIENTS WITH BRONCHOSPASTIC DISEASE SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS. Because of its relative beta$_1$-selectivity, however, TENORETIC may be used with caution in patients with bronchospastic disease who do not respond to or cannot tolerate, other antihypertensive treatment. Since beta$_1$-selectivity is not absolute, the lowest possible dose of TENORETIC should be used and a beta$_2$-stimulating agent (bronchodilator) should be made available. If dosage must be increased, dividing the dose should be considered in order to achieve lower peak blood levels.

Anesthesia and Major Surgery: It is not advisable to withdraw beta-adrenoreceptor blocking drugs prior to surgery in the majority of patients. However, care should be taken when using anesthetic agents such as those which may depress the myocardium. Vagal dominance, if it occurs, may be corrected with atropine (1-2 mg IV).

Beta blockers are competitive inhibitors of beta-receptor agonists and their effects on the heart can be reversed by administration of such agents; eg, dobutamine or isoproterenol with caution (see section on Overdosage).

Metabolic and Endocrine Effects: TENORETIC may be used with caution in diabetic patients. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. At recommended doses atenolol does not potentiate insulin-induced hypoglycemia and, unlike nonselective beta blockers, does not delay recovery of blood glucose to normal levels.

Insulin requirements in diabetic patients may be increased, decreased or unchanged; latent diabetes mellitus may become manifest during chlorthalidone administration.

Beta-adrenergic blockade may mask certain clinical signs (eg, tachycardia) of hyperthyroidism. Abrupt withdrawal of beta blockade might precipitate a thyroid storm; therefore, patients suspected of developing thyrotoxicosis from whom TENORETIC therapy is to be withdrawn should be monitored closely.

Because calcium excretion is decreased by thiazides, TENORETIC should be discontinued before carrying out tests for parathyroid function. Pathologic changes in the parathyroid glands, with hypercalcemia and hypophosphatemia, have been observed in a few patients on prolonged thiazide therapy; however, the common complications of hyperparathyroidism such as renal lithiasis, bone resorption, and peptic ulceration have not been seen. Hyperuricemia may occur, or acute gout may be precipitated in certain patients receiving thiazide therapy.

PRECAUTIONS: Electrolyte and Fluid Balance Status: Periodic determination of serum electrolytes to detect possible electrolyte imbalance should be performed at appropriate intervals.

Patients should be observed for clinical signs of fluid or electrolyte imbalance; ie, hyponatremia, hypochloremic alkalosis, and hypokalemia. Serum and urine electrolyte determinations are particularly important when the patient is vomiting excessively or receiving parenteral fluids. Warning signs or symptoms of fluid and electrolyte imbalance include dryness of the mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances such as nausea and vomiting.

Measurement of potassium levels is appropriate especially in elderly patients, those receiving digitalis preparations for cardiac failure, patients whose dietary intake of potassium is abnormally low, or those suffering from gastrointestinal complaints.

Hypokalemia may develop especially with brisk diuresis, when severe cirrhosis is present, or during concomitant use of corticosteroids or ACTH.

Interference with adequate oral electrolyte intake will also contribute to hypokalemia. Hypokalemia can sensitize or exaggerate the response of the heart to the toxic effects of digitalis (eg, increased ventricular irritability). Hypokalemia may be avoided or treated by use of potassium supplements or foods with a high potassium content.

Any chloride deficit during thiazide therapy is generally mild and usually does not require specific treatment except under extraordinary circumstances (as in liver disease or renal disease). Dilutional hyponatremia may occur in edematous patients in hot weather; appropriate therapy is water restriction rather than administration of salt except in rare instances when the hyponatremia is life-threatening. In actual salt depletion, appropriate replacement is the therapy of choice.

Drug Interactions: TENORETIC may potentiate the action of other antihypertensive agents used concomitantly. Patients treated with TENORETIC plus a catecholamine depletor (eg, reserpine) should be closely observed for evidence of hypotension and/or marked bradycardia which may produce vertigo, syncope or postural hypotension.

Thiazides may decrease arterial responsiveness to norepinephrine. This diminution is not sufficient to preclude the therapeutic effectiveness of norepinephrine. Thiazides may increase the responsiveness to tubocurarine.

Lithium generally should not be given with diuretics because they reduce its renal clearance and add a high risk of lithium toxicity. Read circulars for lithium preparations before use of such preparations with TENORETIC.

Should it be decided to discontinue therapy in patients receiving TENORETIC and clonidine concurrently, the TENORETIC should be discontinued several days before the gradual withdrawal of clonidine.

Other Precautions: In patients receiving thiazides, sensitivity reactions may occur with or without a history of allergy or bronchial asthma. The possible exacerbation or activation of systemic lupus erythematosus has been reported. The antihypertensive effects of thiazides may be enhanced in the postsympathectomy patient.

While taking beta blockers, patients with a history of anaphylactic reaction to a variety of allergens may have a more severe reaction on repeated challenge, either accidental, diagnostic, or therapeutic. Such patients may be unresponsive to the usual doses of epinephrine used to treat the allergic reaction.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Two long-term (maximum dosing duration of 18 or 24 months) rat studies and one long-term (maximum dosing duration of 18 months) mouse study, each employing dose levels as high as 300 mg/kg/day or 150 times the maximum recommended human antihypertensive dose,* did not indicate a carcinogenic potential of atenolol. A third (24 month) rat study, employing doses of 500 and 1,500 mg/kg/day (250 and 750 times the maximum recommended human antihypertensive dose*) resulted in increased incidences of benign adrenal medullary tumors in males and females, mammary fibroadenomas in females, and anterior pituitary adenomas and thyroid parafollicular cell carcinomas in males. No evidence of a mutagenic potential of atenolol was uncovered in the dominant lethal test (mouse), in vivo cytogenetics test (Chinese hamster) or Ames test *(S typhimurium)*.

Fertility of male or female rats (evaluated at dose levels as high as 200 mg/kg/day or 100 times the maximum recommended human dose*) was unaffected by atenolol administration.

Animal Toxicology: Six month oral studies were conducted in rats and dogs using TENORETIC doses up to 12.5 mg/kg/day (atenolol/chlorthalidone 10/2.5 mg/kg/day — approximately five times the maximum recommended human antihypertensive dose*). There were no functional or morphological abnormalities resulting from dosing either compound alone or together other than minor changes in heart rate, blood pressure and urine chemistry which were attributed to the known pharmacologic properties of atenolol and/or chlorthalidone.

Chronic studies of atenolol performed in animals have revealed the occurrence of vacuolation of epithelial cells of Brunner's glands in the duodenum of both male and female dogs at all tested dose levels (starting at 15 mg/kg/day or 7.5 times the maximum recommended human antihypertensive dose*) and increased incidence of atrial degeneration of hearts of male rats at 300 but not 150 mg atenolol/kg/day (150 and 75 times the maximum recommended human antihypertensive dose*, respectively).

Use in Pregnancy: Pregnancy Category C. TENORETIC was studied for teratogenic potential in the rat and rabbit. Doses of atenolol/chlorthalidone of 8/2, 80/20, and 240/60 mg/kg/day were administered orally to pregnant rats with no teratologic effects observed. Two studies were conducted. In the first study, pregnant rabbits were dosed with 8/2, 80/20, and 160/40 mg/kg/day of atenolol/chlorthalidone. No teratologic changes were noted, embryonic resorptions were observed at all dose levels (ranging from approximately 5 times to 100 times the maximum recommended human dose*). In a second rabbit study, doses of atenolol/chlorthalidone were 4/1, 8/2, and 20/5 mg/kg/day. No teratogenic or embryotoxic effects were demonstrated. It is concluded that the no-effect level for embryonic resorptions is 20/5 mg/kg/day of atenolol/chlorthalidone (approximately ten times the maximum recommended human dose*). TENORETIC should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Atenolol—Atenolol has been shown to produce a dose-related increase in embryo/fetal resorptions in rats at doses equal to or greater than 50 mg/kg or 25 or more times the maximum recommended human antihypertensive dose.* Although similar effects were not seen in rabbits, the compound was not evaluated in rabbits at doses above 25 mg/kg or 12.5 times the maximum recommended human antihypertensive dose.* There are no adequate and well-controlled studies in pregnant women.

*Based on the maximum dose of 100 mg/day in a 50 kg patient weight.

Chlorthalidone—Thiazides cross the placental barrier and appear in cord blood. The use of chlorthalidone and related drugs in pregnant women requires that the anticipated benefits of the drug be weighed against possible hazards to the fetus. These hazards include fetal or neonatal jaundice, thrombocytopenia and possibly other adverse reactions which have occurred in the adult.

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Nursing Mothers: Atenolol is excreted in human breast milk at a ratio of 1.5 to 6.8 when compared to the concentration in plasma. Caution should be exercised when atenolol is administered to a nursing woman. Clinically significant bradycardia has been reported in breast fed infants. Premature infants, or infants with impaired renal function, may be more likely to develop adverse effects.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: TENORETIC is usually well tolerated in properly selected patients. Most adverse effects have been mild and transient. The adverse effects observed for TENORETIC are essentially the same as those seen with the individual components.

Atenolol: The frequency estimates in the following table were derived from controlled studies in which adverse reactions were either volunteered by the patient (US studies) or elicited, eg, by checklist (foreign studies). The reported frequency of elicited adverse effects was higher for both atenolol and placebo-treated patients than when these reactions were volunteered. Where frequency of adverse effects for atenolol and placebo is similar, causal relationship to atenolol is uncertain.

	Volunteered (US Studies)		Total-Volunteered and Elicited (Foreign + US Studies)	
	Atenolol (n = 164) %	Placebo (n = 206) %	Atenolol (n = 399) %	Placebo (n = 407) %
CARDIOVASCULAR				
Bradycardia	3	0	3	0
Cold Extremities	0	0.5	12	5
Postural Hypotension	2	1	4	5
Leg Pain	0	0.5	3	1
CENTRAL NERVOUS SYSTEM/ NEUROMUSCULAR				
Dizziness	4	1	13	6
Vertigo	2	0.5	2	0.2
Light-Headedness	1	0	3	0.7
Tiredness	0.6	0.5	26	13
Fatigue	3	1	6	5
Lethargy	1	0	3	0.7
Drowsiness	0.6	0	2	0.5
Depression	0.6	0.5	12	9
Dreaming	0	0	3	1
GASTROINTESTINAL				
Diarrhea	2	0	3	2
Nausea	4	1	3	1
RESPIRATORY (see Warnings)				
Wheeziness	0	0	3	3
Dyspnea	0.6	1	6	4

MISCELLANEOUS: There have been reports of skin rashes and/or dry eyes associated with the use of beta-adrenergic blocking drugs. The reported incidence is small, and, in most cases, the symptoms have cleared when treatment was withdrawn. Discontinuance of the drug should be considered if any such reaction is not otherwise explicable. Patients should be closely monitored following cessation of therapy.

During postmarketing experience, the following have been reported in temporal relationship to the use of the drug: reversible alopecia, impotence, elevated liver enzymes and/or bilirubin, and thrombocytopenia.

Chlorthalidone: Cardiovascular: orthostatic hypotension; Gastrointestinal: anorexia, gastric irritation, vomiting, cramping, constipation, jaundice (intrahepatic cholestatic jaundice), pancreatitis; CNS: vertigo, parasthesias, xanthopsia; Hematologic: leukopenia, agranulocytosis, thrombocytopenia, aplastic anemia; Hypersensitivity: purpura, photosensitivity, rash, urticaria, necrotizing angiitis (vasculitis) (cutaneous vasculitis), Lyell's syndrome (toxic epidermal necrolysis); Miscellaneous: hyperglycemia, glycosuria, hyperuricemia, muscle spasm, weakness, restlessness. Clinical trials of TENORETIC conducted in the United States (89 patients treated with TENORETIC) revealed no new or unexpected adverse effects.

POTENTIAL ADVERSE EFFECTS: In addition, a variety of adverse effects not observed in clinical trials with atenolol but reported with other beta-adrenergic blocking agents should be considered potential adverse effects of atenolol. Nervous System: Reversible mental depression progressing to catatonia; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics; Cardiovascular: Intensification of AV block (see CONTRAINDICATIONS); Gastrointestinal: Mesenteric arterial thrombosis, ischemic colitis; Hematologic: Agranulocytosis, purpura; Allergic: Erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress; Miscellaneous: Peyronie's disease.

There have been reports of a syndrome comprising psoriasiform skin rash, conjunctivitis sicca, otitis, and sclerosing serositis attributed to the beta-adrenergic receptor blocking agent, practolol. This syndrome has not been reported with TENORETIC or TENORMIN® (atenolol).

Clinical Laboratory Test Findings: Clinically important changes in standard laboratory parameters were rarely associated with the administration of TENORETIC. The changes in laboratory parameters were not progressive and usually were not associated with clinical manifestations. The most common changes were increases in uric acid and decreases in serum potassium.

OVERDOSAGE No specific information is available with regard to overdosage and TENORETIC in humans. Treatment should be symptomatic and supportive and directed to the removal of any unabsorbed drug by induced emesis, or administration of activated charcoal. Atenolol can be removed from the general circulation by hemodialysis. Further consideration should be given to dehydration, electrolyte imbalance and hypotension by established procedures.

Atenolol: Overdosage with atenolol has been reported with patients surviving acute doses as high as 5 g. One death was reported in a man who may have taken as much as 10 g acutely.

The predominant symptoms reported following atenolol overdose are lethargy, disorder of respiratory drive, wheezing, sinus pause, and bradycardia. Additionally, common effects associated with overdosage of any beta-adrenergic blocking agent are congestive heart failure, hypotension, bronchospasm, and/or hypoglycemia. Other treatment modalities should be employed at the physician's discretion and may include:

BRADYCARDIA: Atropine 1-2 mg intravenously. If there is no response to vagal blockade, give isoproterenol cautiously. In refractory cases, a transvenous cardiac pacemaker may be indicated. Glucagon in a 10 mg intravenous bolus has been reported to be useful. If required, this may be repeated or followed by an intravenous infusion of glucagon 1-10 mg/h depending on response.

HEART BLOCK (SECOND OR THIRD DEGREE): Isoproterenol or transvenous pacemaker.

CONGESTIVE HEART FAILURE: Digitalize the patient and administer a diuretic. Glucagon has been reported to be useful.

HYPOTENSION: Vasopressors such as dopamine or norepinephrine (levarterenol). Monitor blood pressure continuously.

BRONCHOSPASM: A beta$_2$-stimulant such as isoproterenol or terbutaline and/or aminophylline.

HYPOGLYCEMIA: Intravenous glucose.

ELECTROLYTE DISTURBANCE: Monitor electrolyte levels and renal function. Institute measures to maintain hydration and electrolytes.

Based on the severity of symptoms, management may require intensive support care and facilities for applying cardiac and respiratory support.

Chlorthalidone: Symptoms of chlorthalidone overdose include nausea, weakness, dizziness and disturbances of electrolyte balance.

DOSAGE AND ADMINISTRATION: DOSAGE MUST BE INDIVIDUALIZED (SEE INDICATIONS)

Chlorthalidone is usually given at a dose of 25 mg daily; the usual initial dose of atenolol is 50 mg daily. Therefore, the initial dose should be one TENORETIC 50 tablet given once a day. If an optimal response is not achieved, the dose should be increased to one TENORETIC 100 tablet given once a day.

When necessary, another antihypertensive agent may be added gradually beginning with 50 percent of the usual recommended starting dose to avoid an excessive fall in blood pressure.

Since atenolol is excreted via the kidneys, dosage should be adjusted in cases of severe impairment of renal function. No significant accumulation of atenolol occurs until creatinine clearance falls below 35 mL/min/1.73m² (normal range is 100-150 mL/min/1.73m²); therefore, the following maximum dosages are recommended for patients with renal impairment.

Creatinine Clearance (mL/min/1.73m²)	Atenolol Elimination Half-life (hrs)	Maximum Dosage
15-35	16-27	50 mg daily
<15	>27	50 mg every other day

HOW SUPPLIED

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sented the individual sums of the absolute values of the stepwise differences for each of the two separate secondary motions.* This measure ignored the direction of the deviation, that is, positive or negative, and yielded the *total* amount of deviation for each of the secondary motions. The averaged (Av) secondary motion also considered the deviation that occurred about the zero line of the primary axis; however, it accounted for positive and negative excursions and indicated on which side of the zero line the subjects demonstrated more deviation.†

From these kinematic data, eight variables were identified for the secondary deviations about each of the six test motions. The accumulated secondary deviation variables were as follows:

1. during the first half of motion for one secondary axis;
2. during the second half of one secondary axis;
3. during the first half of the other secondary axis;
4. during the second half of the other secondary axis.

The averaged secondary deviation variables were as follows:

1. during the first half of motion for one secondary axis;
2. during the second half of one secondary axis;
3. during the first half of the other secondary axis; and
4. during the second half of the other secondary axis.

One-way and two-way analysis of variance procedures (ANOVA) were used to test for statistically significant differences among subjects for the four accumulated variables and the four averaged variables. Analysis of covariance (ANCOVA) was used to test for differences in secondary or unintended movements while controlling for the overall range of primary movement. The alpha level for rejection was 0.5 for all analyses.

Results

Forward flexion

No statistically significant differences were found between symmetric and asymmetric groups for the forward flexion motion on statistical comparison of both halves of the movements in the secondary axes for both accumulated and averaged deviation data.

Statistical comparisons of right asymmetric and left asymmetric subjects relative to movements along the secondary axes did not result in any statistically significant differences. This analysis included comparisons of both accumulated and averaged data for secondary movements and for both halves of each motion.

For active versus passive contrasts, statistical analyses were performed to compare secondary head movements for the two secondary axes (accumulated and averaged data). However, none of the comparisons for differences between active and passive movements was statistically significant.

Remaining motions

All secondary movement data were analyzed for both halves of each of the remaining five movements. The first format for the analysis, accumulated deviation data, included all movements regardless of the direction (positive or negative) of the deviation (Table 1). The second format, averaged deviation data, took account of the sign of the movement by adding and subtracting positive and negative deviations (Table 2).

The statistical analyses did not yield any significant trends for either of the secondary

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*Throughout the total range of motion, time, t_i, starts at $t_i = t_1$ and ends at $t_i = t_n$, and i has values starting with $i = 1$ and ending with $i = n$. S_i is the value of a secondary position when time is t_i, which is any particular time from the beginning of the motion at $t_i = t_1$ when $S_i = S_1$ to the end of the motion at $t_i = t_n$ when $S_i = S_n$. The accumulated secondary deviation, *Acc*, was computed as the sum, Σ, of the absolute values of the changes of the secondary positions, $|S_i + 1 - S_i|$, for the intervals between each of the times $t_i + 1$ and t_i:

$$\text{Acc} = \sum_{i=1}^{n-1} |S_{i+1} - S_i|$$

†The average secondary motion, *Av*, was computed as the average of the secondary positions:

$$\text{Av} = \frac{1}{n} \sum_{i=1}^{n} S_i$$

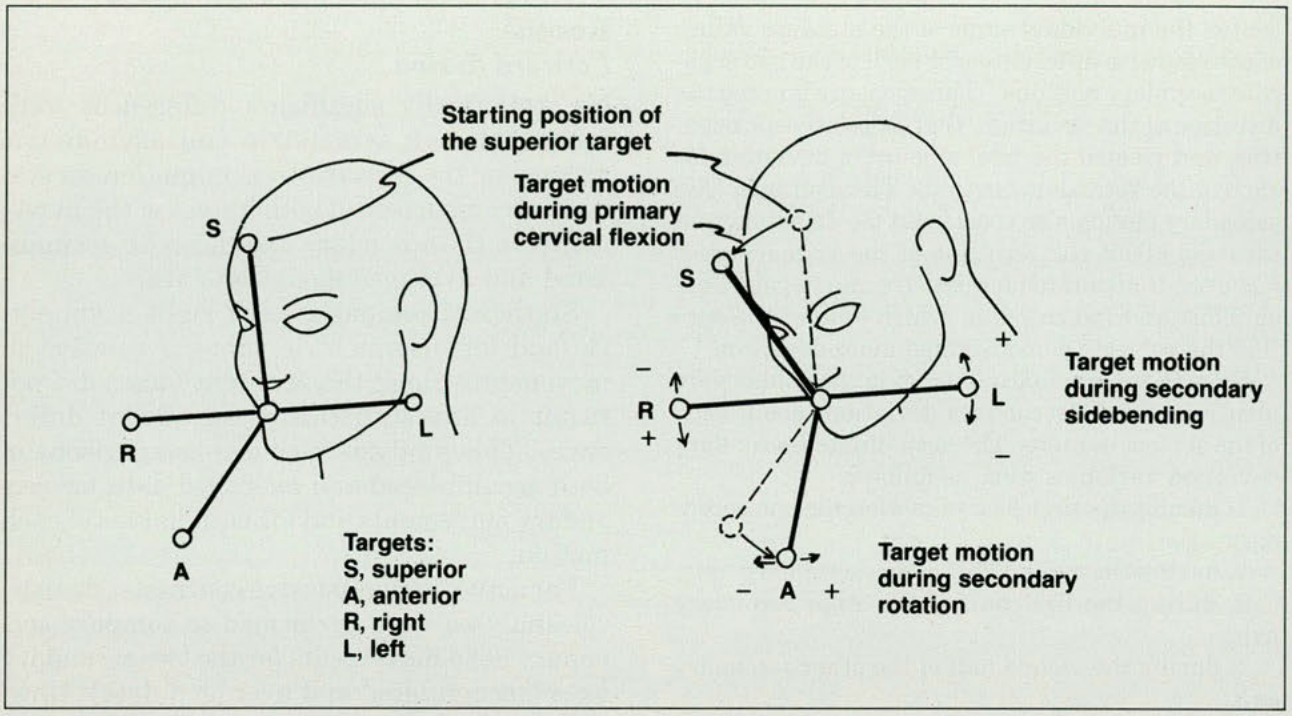

Figure 1. *Schematic of the three-dimensional orientation of head during this example of forward flexion. Major target motion occurred along the primary pathway (forward flexion) with the minor secondary deviations in sidebending and rotation.*

axes for the remaining motions: hyperextension, flexion right, flexion left, rotation right, or rotation left.

Discussion

In our previous studies,[1,2] throughout directed active movements and controlled passive movements from the starting point to an unstressed end of range, asymmetric subjects demonstrated significantly reduced total range of motion in all rotations and significantly altered myoelectric activity.

The current report is concerned with the more minor secondary deviations that occurred about the primary pathways traveled. For this subclinical phase of dysfunction, secondary motion data revealed clearly that the secondary deviations from their primary rotational path involved no distinguishing differences for the asymmetric group. These results complete a picture of valuable information on general kinematic activity of the cervical region for subjects who were asymptomatic and pain-free.

In the current study, the more minor movements about the secondary axes were recorded *while* the primary rotations were being performed. In addition, data were compiled to include both primary and secondary movements (1) from the beginning of motion to the maximum extent of range, and (2) from the maximum extent of range back to the neutral position. When both halves of the movements were compared separately, results for each still showed a trend toward asymmetric subjects' having reduced overall range of motion. This trend persisted even when the primary and secondary motion data for the total excursion were combined (Table 3).

In spite of these limited total ranges for asymmetric subjects, the accumulated and averaged data indicated that *the path followed while the rotations were being performed* did not differ significantly within this limited range. For these examples of a subclinical condition, it seems apparent that dysfunction was not currently being expressed in the more minor aspects of secondary movement. However, these motion data for secondary deviations in *symmetric* subjects in the study described one aspect of a normative behavior pattern, that is,

Table 1
Summary of Accumulated (Acc) Secondary Rotations, in Degrees (n = 17)

Motion and axis	Symmetry group	Passive		Active	
		First half	Second half	First half	Second half
Forward flexion					
Axis 1	Symmetric	0.139	0.133	0.125	0.127
	Right asymmetric	0.143	0.149	0.142	0.134
	Left asymmetric	0.159	0.154	0.128	0.158
Axis 2	Symmetric	0.121	0.105	0.113	0.103
	Right asymmetric	0.132	0.133	0.123	0.120
	Left asymmetric	0.140	0.133	0.176	0.156
Hyperextension					
Axis 1	Symmetric	0.175	0.155	0.202	0.180
	Right asymmetric	0.196	0.167	0.153	0.143
	Left asymmetric	0.170	0.203	0.185	0.160
Axis 2	Symmetric	0.170	0.135	0.193	0.162
	Right asymmetric	0.206	0.110	0.163	0.149
	Left asymmetric	0.163	0.145	0.156	0.149
Flexion right					
Axis 1	Symmetric	0.299	0.211	0.320	0.269
	Right asymmetric	0.236	0.211	0.238	0.223
	Left asymmetric	0.238	0.286	0.276	0.249
Axis 2	Symmetric	0.384	0.349	0.260	0.272
	Right asymmetric	0.289	0.288	0.314	0.297
	Left asymmetric	0.283	0.274	0.234	0.244
Flexion left					
Axis 1	Symmetric	0.277	0.231	0.289	0.282
	Right asymmetric	0.238	0.221	0.269	0.219
	Left asymmetric	0.254	0.276	0.261	0.271
Axis 2	Symmetric	0.323	0.268	0.254	0.294
	Right asymmetric	0.259	0.261	0.263	0.261
	Left asymmetric	0.298	0.308	0.269	0.306
Rotation right					
Axis 1	Symmetric	0.156	0.106	0.124	0.106
	Right asymmetric	0.142	0.140	0.132	0.128
	Left asymmetric	0.182	0.125	0.160	0.119
Axis 2	Symmetric	0.136	0.078	0.095	0.088
	Right asymmetric	0.106	0.102	0.122	0.074
	Left asymmetric	0.131	0.092	0.122	0.104
Rotation left					
Axis 1	Symmetric	0.158	0.112	0.132	0.154
	Right asymmetric	0.142	0.154	0.150	0.134
	Left asymmetric	0.119	0.134	0.150	0.132
Axis 2	Symmetric	0.133	0.137	0.080	0.094
	Right asymmetric	0.089	0.103	0.081	0.084
	Left asymmetric	0.088	0.161	0.107	0.107

Table 2
Summary of Averaged (Av) Secondary Rotations, in Degrees (n = 17)

Motion and axis	Symmetry group	Passive		Active	
		First half	Second half	First half	Second half
Forward flexion					
Axis 1	Symmetric	0.000	0.004	−0.001	0.006
	Right asymmetric	0.003	0.018	0.002	0.002
	Left asymmetric	0.002	0.008	0.003	0.002
Axis 2	Symmetric	−0.003	−0.004	−0.010	−0.012
	Right asymmetric	−0.036	−0.028	−0.007	−0.005
	Left asymmetric	−0.016	−0.016	−0.005	−0.012
Hyperextension					
Axis 1	Symmetric	0.003	−0.002	−0.035	−0.037
	Right asymmetric	0.050	0.047	−0.079	−0.059
	Left asymmetric	0.007	0.017	−0.025	−0.036
Axis 2	Symmetric	−0.017	−0.015	−0.103	−0.071
	Right asymmetric	−0.001	0.018	−0.020	−0.175
	Left asymmetric	0.002	0.006	−0.010	−0.815
Flexion right					
Axis 1	Symmetric	0.182	0.115	0.051	−0.008
	Right asymmetric	0.124	0.081	0.066	0.035
	Left asymmetric	0.238	0.191	0.050	0.033
Axis 2	Symmetric	−0.012	−0.126	−0.014	−0.028
	Right asymmetric	−0.112	−0.126	−0.044	−0.080
	Left asymmetric	−0.081	−0.090	−0.063	−0.045
Flexion left					
Axis 1	Symmetric	0.071	0.043	0.002	−0.003
	Right asymmetric	0.072	0.047	0.019	−0.008
	Left asymmetric	0.119	0.106	0.108	0.089
Axis 2	Symmetric	0.037	0.053	0.040	0.043
	Right asymmetric	−0.008	0.017	0.021	0.029
	Left asymmetric	0.023	0.048	0.015	0.026
Rotation right					
Axis 1	Symmetric	−0.027	−0.054	−0.021	−0.048
	Right asymmetric	−0.021	−0.033	−0.017	−0.038
	Left asymmetric	−0.030	−0.034	−0.015	−0.025
Axis 2	Symmetric	0.096	0.116	0.070	0.074
	Right asymmetric	0.093	0.098	0.054	0.066
	Left asymmetric	0.123	0.133	0.091	0.082
Rotation left					
Axis 1	Symmetric	−0.004	−0.032	−0.032	−0.053
	Right asymmetric	−0.030	−0.053	−0.039	−0.058
	Left asymmetric	−0.014	−0.037	−0.038	−0.052
Axis 2	Symmetric	0.142	0.089	0.081	0.078
	Right asymmetric	0.111	0.097	0.078	0.084
	Left asymmetric	0.162	0.094	0.103	0.089

a benchmark for comparing with others in the study.

Hypothetically, the motor behavior of a healthy body is optimally efficient. Standard procedures of physical examination include tests and criteria to assess this optimal motor performance; a physician can make use of the primary rotations implemented in this study to assess motor functions. The more complex torsions, for example, are difficult to standardize for study. Instructions to the subjects in our study requested only the performance of a primary direction for each movement, and the expectation was that the movement should be efficient and graceful. One's assumption might be that the symmetric group would take a more direct path for the desired movement, and that *secondary* deviations might be greater for subjects with asymmetric motor function. The secondary deviations of our asymmetric group, however, did not differ from the benchmark established for the symmetric group; therefore, our data do not support these particular assumptions about secondary movement.

Do the elements of unintended secondary movement of our subjects about the primary axis offer a factor of adaptability? — self-correction? During each primary movement, a self-correcting system of control is recognized as efficiently monitoring the intended arrival at a comfortable end of range. Even for the asymmetric subjects (who were *asymptomatic*), the secondary movements were not significantly different from those of the symmetric subjects. Can we expect that there will be significant differences if subjects have somatic dysfunction that is acute, extensive, and painful, requiring more adaptation (to pain) and more frequent or greater self-correction throughout the performance of the movement? Further study is needed to test this hypothesis and carefully define the role of secondary movement.

Along with actively resisted motion to test muscle strength, routine physical examination of spinal motor performance makes use of essentially visual skills, with attention to the following: structural alignment; regional ranges of motion *actively* introduced; signs of guarding and subjective pain on motion. Our sample consisted of asymptomatic subjects who were without pain on movement. Setting aside visual tests of active movement, we selected subjects for our symmetric and asymmetric groups on the basis of a single palpatory test for *passive* cervical sidebending to provide a distinguishing positive physical sign of subclinical disturbance in the cervical region. The

Table 3
Total Distance Traveled: Summary of Primary and Secondary Ranges (n = 17)

Individual motions and symmetry group	Distance traveled (degrees of motion)	
	Mean	±SD
Forward flexion		
Symmetric	148.34	24.58
Right asymmetric	121.51	14.43
Left asymmetric	122.51	18.78
Passive	134.07	19.34
Active	127.50	19.16
Hyperextension		
Symmetric	99.23	19.66
Right asymmetric	99.72	10.62
Left asymmetric	89.02	11.30
Passive	99.24	12.32
Active	92.73	15.39
Flexion right		
Symmetric	84.34	14.25
Right asymmetric	69.51	9.50
Left asymmetric	79.20	10.13
Passive	75.16	9.63
Active	80.20	12.95
Fexion left		
Symmetric	91.31	19.22
Right asymmetric	75.79	11.89
Left asymmetric	82.05	10.96
Passive	81.58	12.87
Active	84.52	15.17
Rotation right		
Symmetric	163.36	21.11
Right asymmetric	137.00	21.86
Left asymmetric	150.23	15.53
Passive	153.78	25.30
Active	146.61	13.69
Rotation left		
Symmetric	156.40	19.06
Right asymmetric	135.86	18.99
Left asymmetric	138.66	18.55
Passive	143.92	14.36
Active	143.35	23.36

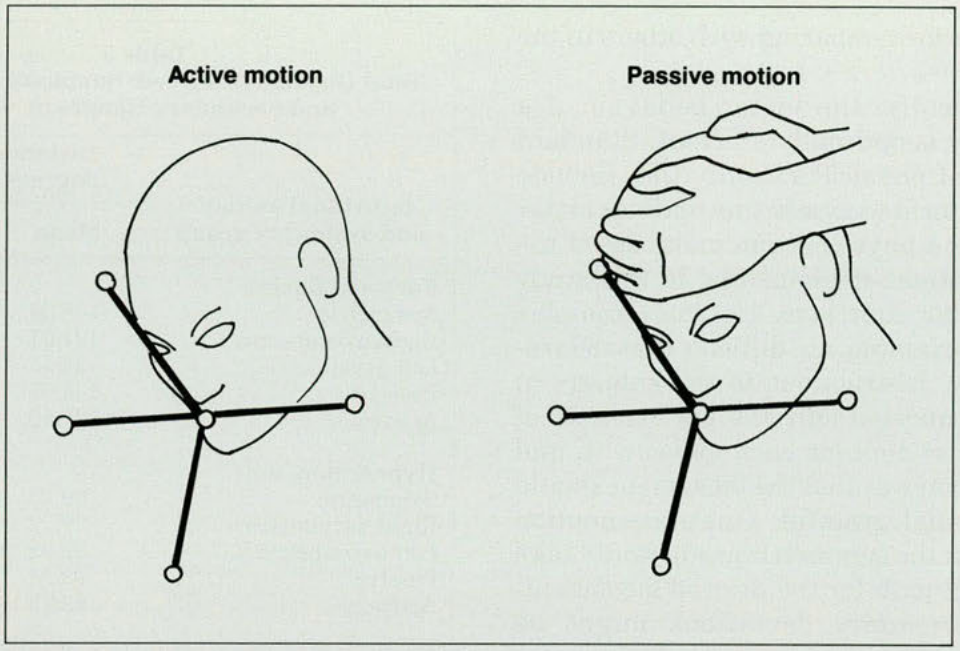

Figure 2. *Comparable three-dimensional motor patterns. Forward flexions are illustrated as examples of motion tests. Whether directed actively or passively, the process of three-dimensional movement in the cervical region was remarkably similar.*

kinematic and myoelectric data have confirmed the presence of measurable physiologic correlates for such regional motor asymmetries: limitation of range and disturbed muscle function.

Passive gross motion tests on physical examination elicit *early* signs of neuromusculoskeletal dysfunction, before symptoms of acute limitation and pain become more obvious and disabling. Patients with conditions such as acute torticollis, brachial neuritis, fibromyalgia, occipital cephalalgia, thoracic outlet complaints, or the trigger-point pain patterns of myofasciitis, when first seen, already have physical evidence of advanced motor restrictions and painful muscular spasm. Their acute episodes are often precipitated by even minor events of physical/emotional stress to already existing, but unrecognized and unresolved, early motor asymmetries. The events provide the stressor, but a motor response that is already asymmetric heightens the functional aberration. Over time, continuing stressful events move asymmetric motor function into a clinical phase with acute presenting complaints. Therefore, early recognition of subclinical signs of regional asymmetry should be a first step toward prevention of the neuromusculoskeletal dysfunctions that so greatly impair the quality of life.

Professionals concerned with patients who have had more *major* neck injury, such as from auto or sports accidents should recognize that they need to assess not only *tissue damage* resulting directly from the traumatic forces on impact, but also *the patient who has been injured*. Specifically with regard to the neck region, did factors such as malignancy, degenerative joint disease, osteoporosis, or asymmetric function precede the trauma? In short, the physical stress always affects an ongoing, already compromised system. When subclinical motor asymmetries and muscular dysfunctions precede an accident, they serve as points of lowered threshold to the physical stress of major trauma. These predisposing problems focus stress, intensify disability, and exaggerate pain patterns that delay recovery. Our data on asymptomatic subjects suggest that future research involve the selection of an experimental group of patients with more acute symptoms, that is, painful clinical problems with associated asymmetric motor behavior.

One additional result deserves comment.

When data on secondary movements that occurred during actively and passively induced primary movements are compared, these secondary movements are remarkably similar (Fig 2). This observation supports a major aspect of the results described previously for active versus passive total primary motions at their end points.[1] We have found continued evidence that, through palpation, the trained examiner introducing passive motions was as aware of the reflex controls and patterns of movement best suited for those subjects as the subjects themselves, when they actively performed the same primary rotations. These findings contribute further confidence in the clinical performance of a trained examiner, especially in regard to trainable sensitivity for detecting disturbance in motor function. Items of technique for such training have been described[4(p300)]:

Technique is an important factor in passive motion testing. A motion test is introduced by a skilled operator who controls a specific direction of simple motion in testing, rather than a more complex torsion, for example. Patient cooperation is gained by the physician's briefly describing the procedure and asking the patient to allow the movement, that is, to go along with it and not offer resistance that will interfere with test results. Going through several preliminary motions often will gain the patient's confidence in the procedure.

Placement of hands is light, to avoid having the patient react to uncomfortable pressures. For example, when the patient is seated, the operator does not grasp the patient's head as if he or she was going to *do* the turning; the operator's active role is merely to initiate and guide the motion. Light contact also complements the operator's need to sense response to motion throughout the test. In testing to compare rotation right with left, hand placements should be sufficiently similar during each test to standardize the demand for motion equally in opposing directions.

Attention to these features provides teachers with an orderly process of instruction for developing physical examination skills in students. During a physical examination or as a demonstration to students, a physician initiates and directs procedures that permit the sampling of motor behavior, that is, to monitor for range and quality of motion. In judgments of symmetry, criteria are applied that are based on sensory perception, specifically on palpable features of varying resistance to the contact of pressure sensors in the examiner's finger pads. Our kinematic and myoelectric data support the premise that a similar response to the testing procedures is being monitored by the trained examiner's hands in control of the procedure passively, as by the subject in control of the motion actively. Students now have a reference for success in acquiring the sensory and motor skills necessary for confident clinical performance in tests for motor function.

Agreement between the physician's performance and these kinematic measurements of both primary and secondary motions demonstrates that this training provides sensitivity not only to the end point of motion but also to the progress of the head and neck through three-dimensional space.

These results support the use of passively induced gross motion tests to gather medical information about asymmetric motor behavior during the evaluation of health status on physical examination.

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