

## Mechanical Modulation of Growth for the Correction of Vertebral Wedge Deformities

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**Summary:** This study tested the following hypotheses: (a) a vertebral wedge deformity created by chronic static asymmetrical loading will be corrected by reversal of the load asymmetry; (b) a vertebral wedge deformity created by chronic static asymmetrical loading will remain if the load is simply removed; and (c) vertebral longitudinal growth rates, altered by chronic static loading, will return to normal after removal of the load. An external fixator was used to impose an angular deformity (Cobb angle of 30°) and an axial compression force (60% body weight) on the ninth caudal (apical) vertebra in two groups of 12 5-week-old Sprague-Dawley rats. This asymmetrical loading was applied to all rats for 4 weeks to create an initial wedge deformity in the apical vertebra. The rats from group I (load reversal) then underwent 1 week of distraction loading followed by 4 weeks of asymmetrical compressive loading with the imposed 30° Cobb angle reversed. The rats from group II (load removal) had the apparatus removed and were followed for 5 weeks with no external loading. Weekly radiographs were obtained and serial fluorochrome labels were administered to follow vertebral wedging. After the initial 4-week loading period, the combined average wedge deformity that developed in the apical vertebra of the animals in both groups was  $10.7 \pm 4.4^\circ$ . The group that underwent load reversal showed significant correction of the deformity with the wedging of the apical vertebra decreasing to, on average,  $0.1 \pm 1.4^\circ$  during the 4 weeks of load reversal. Wedging of the apical vertebra in the group that underwent load removal significantly decreased to  $7.3 \pm 3.9^\circ$  during the first week after removal of the load, but no significant changes in wedging occurred after that week. This indicated a return to a normal growth pattern following the removal of the asymmetrically applied loading. The longitudinal growth rate of the apical vertebra also returned to normal following removal of the load. Vertebrae maintained under a load of 60% body weight grew at a rate that was  $59.4 \pm 17.0\%$  lower than that of the control vertebrae, whereas after vertebrae were unloaded their growth averaged  $102.4 \pm 31.8\%$ . These findings show that a vertebral wedge deformity can be corrected by reversing the load used to create it and that vertebral growth is not permanently affected by applied loading.

Scoliosis is a complex three-dimensional deformity of the spine. Its natural history has been separated into two stages: initiation and subsequent progression of the deformity (9). The initiating factors are unknown in most cases, but progression of the deformity is associated with the adolescent growth spurt. The primary risk factors for progression in adolescents include age at onset (the amount of remaining growth) and magnitude of the curve (8). The most prominent and serious component of the deformity is the lateral curvature, which results from wedging of the vertebrae and disks

(12,20). The progression of a bone deformity during growth is thought to be governed by the Hueter-Volkman law, which states that growth is retarded by mechanical compression of the growth plate and stimulated by distraction or reduced compression (5,17,18). On the basis of this law, a vicious-cycle hypothesis for the progression of scoliosis has been proposed (13,16), stating that an initial spinal curvature causes asymmetrical loading of the vertebrae, which results in asymmetrical growth along the physes. This asymmetrical growth results in the formation of wedge-shaped vertebrae, perpetuating the cycle by increasing the spinal curvature and thus the asymmetrical loading.

It has been shown that longitudinal growth of vertebrae in a rat tail is modulated by axial loading (16), with distraction loading increasing the growth and compression loading decreasing it as predicted by the Hueter-Volkman law. Additionally, imposed angulation and axial compression, which created an asymmetrical load across a vertebra in a rat tail, caused

Received April 20, 1998; accepted December 20, 1998.

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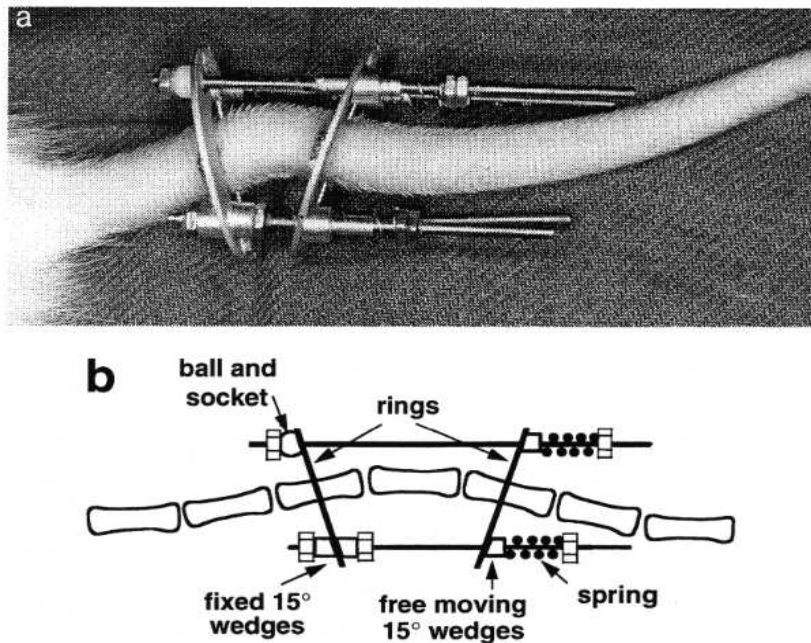


FIG. 1. Loading apparatus. **a:** Photograph of the apparatus applied to the tail of a rat. **b:** Schematic illustration of the loading apparatus.

asymmetrical growth along the physes; this resulted in significant wedging of the loaded vertebra (10). Thus, vertebral growth can be mechanically modulated and asymmetrical vertebral loading can produce differential growth along a physis, resulting in the formation of a vertebral wedge deformity. However, it remained undetermined whether a deformity created by externally applied loading could be corrected by reversal of the applied loads and whether the altered growth rates in the loaded physes could be restored to normal.

The purpose of the present study was to test the following hypotheses: (a) a vertebral wedge deformity created by chronic static asymmetrical loading will be corrected by reversal of the load asymmetry; (b) a vertebral wedge deformity created by chronic static asymmetrical loading will remain after removal of the loading; and (c) vertebral longitudinal growth rates, altered by chronic static loading, will return to normal after removal of the load.

## MATERIALS AND METHODS

An external fixator was installed at the eighth and tenth caudal vertebrae in two groups of 12 5-week-old Sprague-Dawley rats. The fixator was designed to apply a chronic static axial compression force in conjunction with an imposed angulation to asymmetrically load the intervening ninth caudal (apical) vertebra and adjacent discs. An initial vertebral wedge deformity was created in the apical vertebra of all rats by application of this apparatus for 4 weeks. After creation of the initial wedge deformity in group I (load reversal), the imposed angulation was reversed to determine if a vertebral wedge deformity could be corrected. After the initial deformity was created in group II (load removal), the compressive load and imposed angulation were removed to examine the vertebral growth response following removal of the load. Angulation and growth in the vertebrae and physes were measured with use

of weekly radiographs and fluorochrome labeling. All animal procedures were reviewed and approved by the Institutional Animal Care and Use Committee.

### Loading Apparatus

The loading apparatus and surgical procedure for its installation have been described previously (10,16). Briefly, the loading apparatus (Fig. 1) consisted of two 35-mm-diameter aluminum rings that were installed over the tail of the rat. One ring each was attached to the eighth and tenth caudal vertebrae with use of a pair of crossed 0.7-mm stainless-steel Kirschner wires percutaneously inserted through the vertebrae. Four stainless-steel threaded rods were passed through holes distributed around the rings to transmit the applied spring load between the rings. An overall Cobb angle of 30° was established between the eighth and tenth caudal vertebrae with 15° machined wedges to link the two rings to the rods. The proximal ring was fixed to the rods with use of wedges on one pair of diagonally opposed rods, and ball joints were used on the other two rods to maintain the alignment of the apparatus without overconstraining the fixation. Freely moving wedges were installed between the distal ring and the loading springs on all four rods. Force magnitude and distal-ring angulation

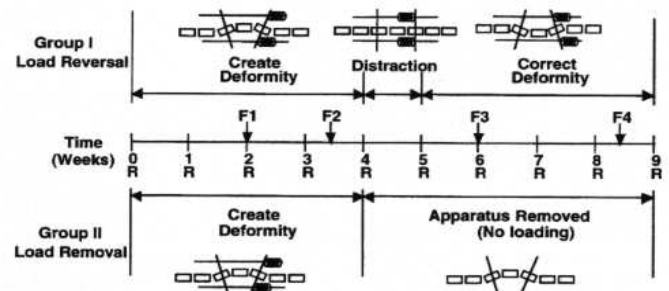
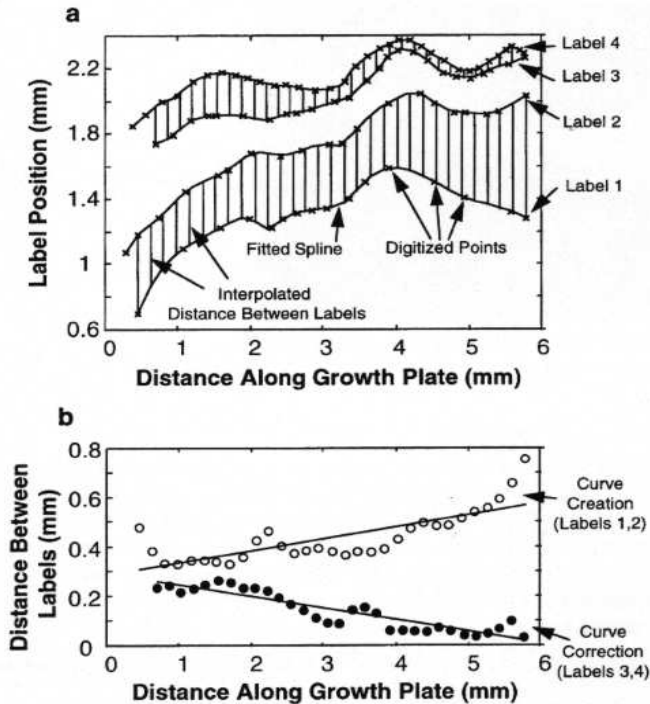


FIG. 2. Diagram of the experimental protocol, showing the three phases of loading for group I (load reversal) and the two phases for group II (load removal). The timing of radiographs (R) and fluorochrome administration (F1, F2, F3, and F4 = days 14, 24, 42, and 59) is also indicated.



**FIG. 3.** Example of analysis of the fluorochrome labels and derivation of the amount of wedge deformity. **a:** The location of the fluorochrome labels remaining in the bone on the histology prints was digitized (x), and a spline function was then mathematically fitted through these points so that the distance between the pairs of labels could be interpolated at 30 equally spaced intervals. **b:** A linear regression line was fitted to the distance between the label pairs as a function of label position. The slope of the regression line (degrees) represents the amount of angular deformity generated by the physis during the time intervals between labels.

were maintained by independently adjusting the lengths of the calibrated springs (stiffness: 0.30 N/mm) with use of lock nuts installed on each stainless-steel rod. Spring lengths were measured with a vernier caliper and were adjusted to maintain the total force at 60% of the body weight of the rat. Adjustments compensated for the growth of the tail and increasing body weight.

The loading apparatus allowed a chronic static load to be superimposed on the normal physiological forces acting on the ninth caudal (apical) vertebra. The asymmetrical loading of the growth plate resulted from the applied angulation, which displaced the apical vertebra laterally relative to the centerline of the loading; this created different moment arms for the springs on the left and right sides of the tail. The magnitude of the moment that resulted from the four spring forces was not determined because it depended on the stiffness and deformation of the intervertebral disks and the moment transmitted through the 15° wedges. The apparatus weighed 13.6 g and was tolerated very well by the animals; lifting their tails with the apparatus attached was not a problem. Although the segments within the apparatus were essentially immobilized, the rats were able to move and control their tails normally both proximally and distally to the apparatus.

### Experimental Protocol

The two loading protocols (groups I and II) are shown diagrammatically in Fig. 2. In the first phase of the experiment, which was the same for both groups, an initial wedge deformity was created in the ninth caudal vertebra by the application of asymmetrical loading for 4 weeks. A total compression load of 60% body weight was used along with the imposed Cobb angle of 30°. It had been determined in a previous study (10) that this level of loading

resulted in the creation of a significant vertebral wedge deformity by the end of 4 weeks.

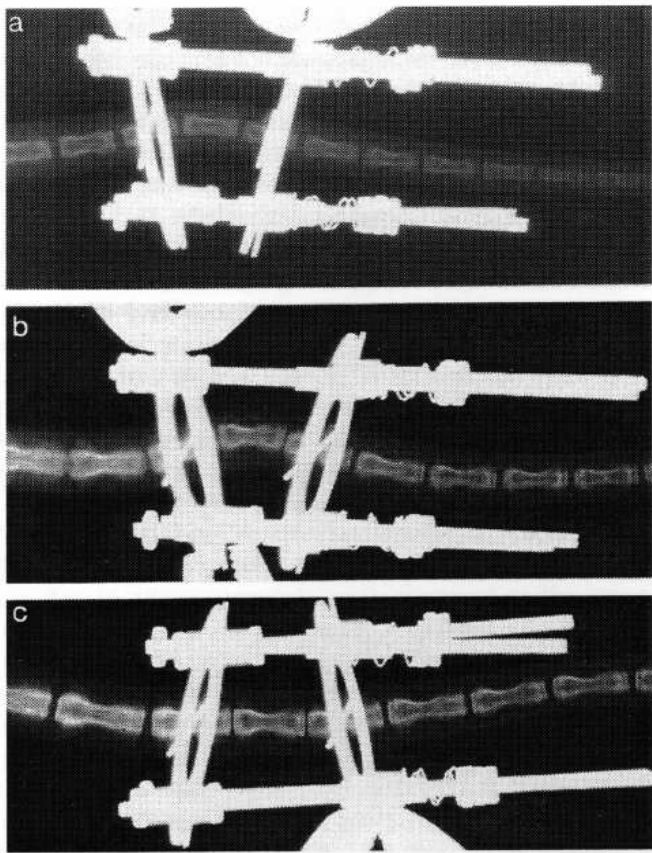
In group I (load reversal), creation of the deformity was followed by 1 week of axial distraction loading at 60% body weight and no forced angulation. At the end of week 5, the compressive load of 60% body weight was reapplied and wedges were reinstalled on the apparatus to create a 30° Cobb angle in the opposite direction to that used during the initial 4 weeks. This reversed angulation with axial compression was maintained for an additional 4 weeks. The week of distraction was necessary because the two disks adjacent to the apical vertebra become stiff during 4 weeks of compression loading and immobilization within the apparatus. The distraction loading gave the disks time to recover and allowed safe reversal of the applied angulation.

In group II (load removal), the apparatus was disassembled and removed following creation of the deformity (at week 4), leaving the two rings attached independently to the eighth and tenth caudal vertebrae. These animals were then followed for an additional 5 weeks.

Dorsal-ventral radiographs of each tail were taken weekly. Four different fluorochrome labels were administered to mark the ossifying front of the growth plate at four distinct time points. The four labels used were calcein (intraperitoneal injection, 15 mg/kg), alizarin complexone (subcutaneous injection, 30 mg/kg), xylenol orange (subcutaneous injection, 90 mg/kg) (all; Sigma Chemical, St. Louis, MO, U.S.A.), and oxytetracycline (intraperitoneal injection, 30 mg/kg) (Oxybiotic-200; Butler, Columbus, OH, U.S.A.). The labels were administered at 14, 24, 42, and 59 days (Fig. 2). The first two labels, administered 10 days apart, documented the angulation of the growth plate resulting from the initial loading (creation of the deformity). The third and fourth labels, administered 17 days apart, documented the angulation during the second part of the experiment (load reversal in group I, and load removal in group II). A longer time interval was used between the second set of labels because the growth rate slowed over the course of the experiment. The timing of injections was intended to record approximately equal growth during the two time periods on the basis of the growth rates measured in previous studies (10,16). At the end of the experiment (9 weeks after installation of the apparatus), the rats were killed by CO<sub>2</sub> gas asphyxiation and thoracotomy.

On each weekly radiograph, the four control vertebrae (caudal vertebrae 6, 7, 11, and 12) and the apical vertebra (caudal vertebra 9) were marked and digitized as described previously to measure vertebral wedging (10) and longitudinal growth (16). Average growth and angulation rates were calculated from these measurements by regression analysis of vertebral length and wedging measurements as a function of time.

Two vertebrae from each animal were fixed and sectioned (10) so that measurements could be made on the fluorochrome labels. These were caudal vertebrae 9 (apical vertebra) and 6 (control vertebra located two vertebrae proximal to the apparatus). The positions of the fluorochrome labels were digitized with use of images of a 300- $\mu$ m-thick frontal plane section that had been taken through the center of the vertebrae and printed at a magnification of  $\times 70$  (10). The digitized points were imported to Matlab (The MathWorks, Natick, MA, U.S.A.), and a cubic spline was fitted to these points to generate four curves depicting the location of the growth plate at the four time points at which the labels were administered (Fig. 3). Vertical lines were drawn between the two sets of labels (between labels one and two and labels three and four) at 30 evenly spaced locations across the width of the vertebrae. The length of these lines was plotted as a function of position along the physes. The slope of the straight line fit to these points produced the angulation of the growth plate between labels. Angulation rates ( $^{\circ}$ /day) were calculated by dividing the angulation by the number of days between labels.



**FIG. 4.** Group I (load reversal): radiographs of the rat tail. **a:** Time zero, just after application of the fixator; **b:** week 4, at the end of creation of the deformity and just before distraction; and **c:** week 9, after 4 weeks of load reversal. At time zero, the endplates of the apical vertebra were parallel. By week 4, they developed a distinctive wedge shape and by week 9, they were relatively parallel (wedge deformity was corrected).

#### Statistical Analysis

Radiographic measurements of the wedging of the apical vertebra during creation of the deformity were analyzed by a two-factor analysis of variance (ANOVA) (factors: group and week, with week as a repeated factor). Radiographic measurements of the angulation of the apical vertebra for group I during curve correction (weeks 5-9, not including the distraction week) were analyzed by a one-factor ANOVA with week as a repeated factor. A separate one-factor ANOVA with time as a repeated factor was performed for radiographic measurements for group II to test for angulation changes following removal of the load (weeks 4-9). A three-factor ANOVA was used to analyze differences in the angulation of the control vertebrae for weeks 0-9 by group, position of the vertebrae relative to the loading apparatus (repeated measure), and week (repeated measure).

The effect of loading on longitudinal vertebral growth rates was examined with the growth rate of the apical vertebra expressed as a percentage of the average growth rate of its four control vertebrae (growth rates were calculated with use of a linear regression of radiographic length measurements over the desired time period [10,16], i.e., weeks 0-4 or 5-9). A two-factor ANOVA was performed for the growth rate with group and time period as factors.

All statistical calculations were performed with use of SAS (release 6.12; SAS Institute, Cary, NC, U.S.A.). *Post hoc* analyses were performed with use of either a paired Student's *t* test or, when multiple comparisons were made, the Student-Newman-Keuls test. A level of 0.05 was used for significance. For Student's *t* tests,

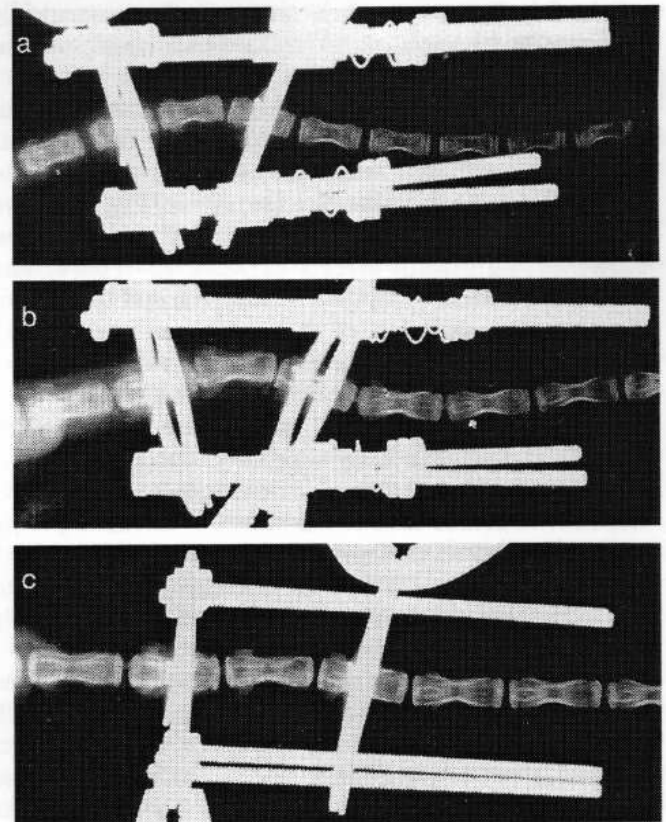
the Bonferroni criterion was used to adjust the significance level.

#### RESULTS

Four of the 24 rats were removed from the study (two from each group) because of improper placement of the Kirschner wire (three animals) or failure of the glue bond between the wire and the fixator ring (one animal). The remaining 20 rats completed the 9-week protocol without any complications.

Typical radiographs at time zero (just after application of the apparatus), 4 weeks (end of the initial loading phase), and 9 weeks (immediately after death) are shown for groups I (load reversal, Fig. 4) and II (load removal, Fig. 5). The average radiographic wedge angle of the apical and control vertebrae for both groups over the course of the experiment are plotted in Fig. 6.

During creation of the deformity (weeks 0-4), wedging of the apical vertebrae for the two groups showed a significant time effect ( $p = 0.0001$ ) but no significant group effect or group-time interaction. Over this time period, wedging increased steadily (Fig. 6), and by week 4 the average wedging of the apical vertebrae



**FIG. 5.** Group II (load removal): radiographs of the rat tail. **a:** Time zero, just after application of the fixator; **b:** week 4, at the end of creation of the deformity and just before removal of the apparatus; and **c:** week 9, after 5 weeks with no external loading. At time zero, the endplates of the apical vertebra were parallel. By week 4, they developed a distinctive wedge shape and at week 9, most of this angulation remained.

**TABLE 1.** Relative longitudinal growth rates<sup>a</sup>

Weeks	Group I (load reversal)	Group II (load removal)
0-4	64.5 (12.0) <sup>b</sup>	59.8 (13.0) <sup>b</sup>
5-9	53.9 (23.6) <sup>b</sup>	102.4 (31.8)

<sup>a</sup> Mean (SD) of the longitudinal growth rate of the apical vertebra as a percentage of the average growth rate of its four adjacent control vertebrae.

<sup>b</sup> Values for vertebrae under a load of 60% body weight.

was  $10.6 \pm 3.7^\circ$  for group I (load reversal) and  $10.7 \pm 5.2^\circ$  for group II (load removal).

For the group that underwent load reversal, average wedging of the apical vertebrae increased slightly during the week of distraction loading (week 5), from  $10.6 \pm 3.7$  to  $11.0 \pm 5.0^\circ$ . This change was not significant. With the reversal of the asymmetrical loading from weeks 5 to 9, the wedge deformity showed a highly significant correction to  $0.1 \pm 1.4^\circ$  ( $p = 0.0001$ ).

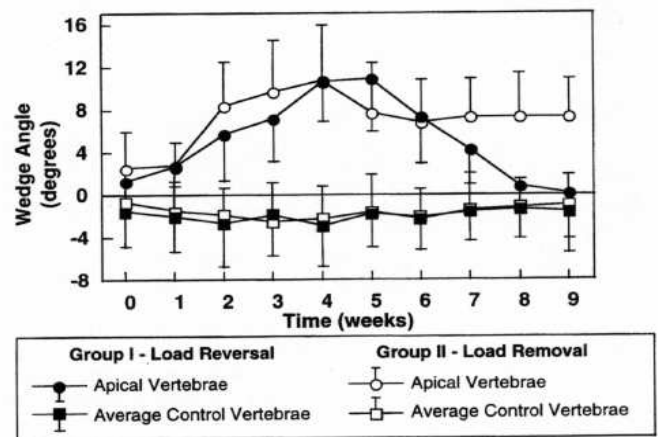
For the group that underwent load removal, there was a significant reduction of  $3.0 \pm 2.9^\circ$  in the wedging of the apical vertebrae over the first week following removal of the load (weeks 4-5) ( $p = 0.02$ ). From weeks 5 to 9, the vertebrae maintained an essentially constant angulation of  $7.3 \pm 3.9^\circ$  with no significant changes detected.

Wedging of the control vertebrae (mean of the four control vertebrae) averaged  $-1.6 \pm 3.0^\circ$  for group II (load removal) and  $-2.0 \pm 3.4^\circ$  for group I (load reversal) during the 9-week experiment (Fig. 6). There was no significant difference between the two groups. However, significant effects of week ( $p = 0.04$ ) and week-level interaction ( $p = 0.003$ ) were detected, indicating that wedging of the vertebrae varied with time and depended on their position relative to the instrumentation.

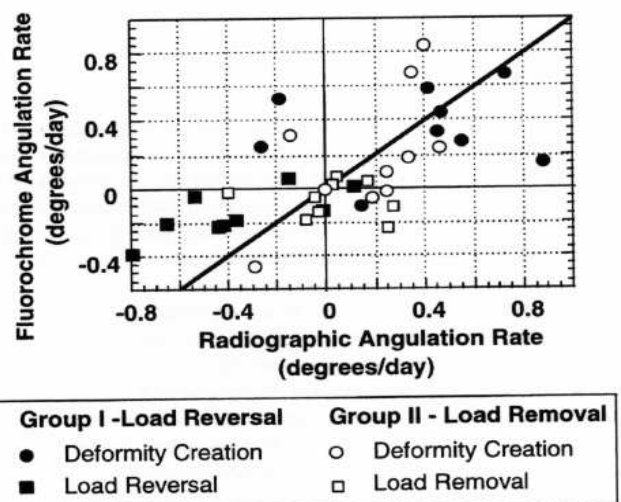
The average longitudinal growth rate for the vertebrae loaded at 60% body weight (group I: apical vertebra; group II: apical vertebra before load removal) was  $59.4 \pm 17.0\%$  that of the control vertebrae (Table 1). The vertebrae that were unloaded during weeks 5 through 9 (group II) had an average growth rate of  $102.4 \pm 31.8\%$  of the controls, which was significantly greater than the rates for the loaded vertebrae.

Longitudinal growth rates for the control vertebrae differed with location and time. The average growth rates over the 9-week study were  $57.4 \pm 33.5$ ,  $55.4 \pm 33.4$ ,  $51.0 \pm 31.4$ , and  $51.2 \pm 31.7 \mu\text{m/day}$  from the most proximal to the most distal control vertebrae. The growth rates of the two distal vertebrae were significantly lower than for the most proximal control vertebrae. The average vertebral growth rate was  $75.8 \pm 22.7 \mu\text{m/day}$  during the first 4 weeks of the study and decreased significantly to  $24.1 \pm 8.7 \mu\text{m/day}$  during the last 4 weeks.

Overall, no significant differences between fluorochrome and radiographic measurements of vertebral angulation rates were detected for the apical vertebrae (Fig. 7). For technical reasons, angulation rates measured with fluorochrome could not be made on three of the control vertebrae and one of the apical vertebra in group I during each of the two time periods (initial loading and load reversal). Similarly, angulation rates measured radiographically could not be calculated for six control vertebrae and one apical vertebra. This left 68 of 80 observations for analysis of the controls and 78 of 80 for analysis of the apical vertebrae. The magnitude of the average angulation rate during load reversal measured radiographically ( $-0.36 \pm 0.28^\circ/\text{day}$ ) was larger than that measured from fluorochrome labeling ( $-0.14 \pm 0.14^\circ/\text{day}$ ). Al-



**FIG. 6.** Radiographically measured vertebral wedge angle plotted against the time from installation of the fixator. Each point represents the average  $\pm$  SD for 10 apical (ninth caudal) vertebrae or 40 control vertebrae. The control vertebrae were the two vertebrae cephalad and caudal to the loading apparatus.



**FIG. 7.** Correlation between asymmetrical vertebral growth measured from fluorochrome labels (vertical axis) and radiographs (horizontal axis) of the loaded apical vertebra. The solid line represents the theoretical 1:1 line for equality of the two measurements.

though this difference was not significant ( $p = 0.08$ ), it was larger than the differences seen during creation of the curve ( $0.26 \pm 0.32^\circ/\text{day}$  radiographically, and  $0.27 \pm 0.32^\circ/\text{day}$  from fluorochrome labeling) or removal of the load ( $0.02 \pm 0.19^\circ/\text{day}$  radiographically, and  $-0.06 \pm 0.10^\circ/\text{day}$  from fluorochrome labeling). As expected, the average angulation rate measured from fluorochrome labeling in the control vertebrae ( $0.01 \pm 0.25^\circ/\text{day}$ ) was not significantly different from zero ( $p = 0.77$ ).

## DISCUSSION

This study showed that a vertebral wedge deformity can be corrected by reversal of the load used to create it. This implies that the principles of the Hueter-Volkman law are applicable to the correction of an existing vertebral deformity providing there is sufficient residual growth. In contrast, there was little change in angulation following removal of the asymmetrical loading. The external loading reduced longitudinal growth rates, but the rates returned to control values with removal of the load. These results demonstrate that growth is not permanently affected by applied external loading of the magnitude applied here.

There were no significant differences between the measurements of vertebral wedging made radiographically or from fluorochrome labeling. This finding is in agreement with the results found previously for wedge creation (10) and indicates that the vertebral wedging was caused by asymmetrical growth of the physes and not by diaphyseal remodeling or other mechanisms. The slower angulation rate measured with fluorochrome labels during load reversal, although not statistically significant, may indicate that other mechanisms were more active as the growth rate of the rats slowed. In this study, chronic static loading was used to modulate growth in the physes; the remodeling of mature bone is thought to be stimulated by time-varying rather than static loading (7,14).

Only small changes in angulation of the control vertebrae were measured over the course of the experiment (Fig. 6); however, time effect and level-time interaction were significant. Wedging of these vertebrae tended to increase during creation of the wedge and to reduce after load removal or curve reversal. These effects probably resulted from bending of the tail (on either side of the loading apparatus), producing compensatory curves in the opposite direction of the imposed curve. This speculation is supported by the fact that the two vertebrae adjacent to the apparatus showed the largest effect (angulation of  $2-3^\circ$  opposite that of the apical vertebra by week 4), whereas the two farther from the apparatus showed a somewhat smaller effect (angulation of  $1-2^\circ$  opposite that of the apical vertebra by week 4).

Some technical difficulties were encountered in

these experiments. For group I, the order of fluorochrome injections was calcein, alizarin complexone, xylenol orange, and oxytetracycline. For group II (all injections were performed after those for group I), the sequence of the labels was changed to oxytetracycline, alizarin complexone, calcein, and xylenol orange to separate the two reddish fluorescing labels and thus facilitate analysis of the histologic sections. Radiographic measurements were challenging despite the use of fine-resolution film. Previously, we reported the precision of the radiographic measurement technique, with intraobserver errors in marking and digitizing the wedge deformity as  $3.7^\circ$  and interobserver errors as  $3.0^\circ$  (10).

We attempted to maintain precise control over the applied force and tail angulation; however, this was not completely successful. During creation of the deformity (weeks 0-4), the average imposed Cobb angle (intended to be maintained at  $\pm 30^\circ$ ) for group I was  $28.6 \pm 8.9^\circ$ . However, during load reversal, it averaged  $-20.5 \pm 7.4^\circ$ . We believe the smaller Cobb angle achieved during load reversal was due to stiffening of the disks, a side effect of the loading apparatus that compresses and reduces the mobility of the disks (6). The intermediate week of distraction used for group I was intended to offset this effect. In group I, the wedging of the apical vertebra changed more quickly during the load-reversal phase than during the initial creation of the deformity. This occurred despite the fact that the growth rate of the vertebra slowed down over time. This may have resulted from a more asymmetrical loading of the vertebra during correction of deformity caused by the greater load transfer in the disks during correction, compared with creation, of the curve.

Mechanical modulation of growth has been studied primarily in long bones (1,2,4,19), and only a few studies have examined this phenomenon in the spine. Stefko et al. used an external fixator to distract sections of a goat spine (15) but did not report what percentage of the change in growth occurred at the growth plates relative to the discs. Stapling has been used in the spine for the correction of scoliosis deformities (3,11). Its greater success in dogs (11) than in humans is apparently because humans were treated when there was insufficient residual growth (3) and the staples may not have generated sufficient force to affect growth substantially.

A basic assumption of this work, as it relates to scoliosis, is that the progression of the lateral vertebral wedging in scoliosis is secondary to changes in growth rate across the vertebral growth plate. The results of this study indicate that mechanical modulation can be used to reverse this type of deformity. However, even when a bone deformity is not caused by mechanical loading, the results suggest that mechanical modula-

tion of the residual growth can be used to correct the deformity. These findings also have implications in the design, use, and effectiveness of bracing as a treatment method for scoliosis. If braces can be designed to modify the loading across the physes of the vertebrae in a scoliotic curve, it may be possible to halt the progression of a deformity or even reverse it.

In this study, an external fixator reversed a deformity created in the vertebra of the rat tail; however, the required level of force and responsiveness of human vertebrae is unknown. If this technique is to be successfully applied to the correction of human vertebral deformities, more work is required to determine how the forces applied to a rat vertebra can be scaled to create the equivalent effect in human vertebrae.

**Acknowledgment:** This work was supported by National Institutes of Health individual National Research Service Award Grants F32 AR08453-01 and F32 AR08484-01 and National Institutes of Health Grant R55 HD 34460. The authors acknowledge the assistance of Holly Spence for help in animal procedures and data collection.

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