Influence of Some Biomechanical Factors on Low-Back Pain in Pregnancy

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Several biomechanical factors were recorded intermittently in 855 pregnant women from the 12th to the 36th week of gestation and were related to back pain occurrence during pregnancy. The three factors related to the development of back pain were abdominal sagittal diameter, which correlated with back pain, with a coefficient of 0.15 ($P < 0.01$); transverse diameter ($r = 0.13$, $P < 0.01$); and depth of the lumbar lordosis, which correlated with a coefficient of 0.11 ($P < 0.01$). In the group of women who were pregnant for their first time, there was a significantly lower peripheral joint laxity in the 12th week in those women who, later in pregnancy, developed back pain. These correlations suggest that back pain in pregnancy can not be explained primarily by biomechanical factors. [Key words: pregnancy, back pain, biomechanics, spinal loading]

Back pain is common in women with normal pregnancies. Earlier studies report that 50% or more of all pregnant women experience back pain during their pregnancies.[1,4,11,12] Two widely accepted explanations are that back pain during pregnancy is caused by the increase in load on the back due to the total weight gain during pregnancy and the weight of the fetus, and by hormonal changes in the pregnant woman, which make the spine and sacroiliac joints "less stable."[6,7] A third suggested explanation of pain in the sacroiliac region is connective tissue microtrauma as a consequence of the trunk extensor muscle forces to balance the anterior flexion moment caused by the growing uterus.[9] However, these assumptions are still open to discussion because no scientific proof exists. Our knowledge of back pain in pregnancy is limited to epidemiologic descriptions of its natural course, but why back pain is not found in all pregnancies or whether any biomechanical risk factors can explain existing pain is still unknown.

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The aims of this prospective study were to identify some simple biomechanical factors specific to pregnancy, follow their development, and evaluate their influence on back pain during pregnancy.

Patients and Methods

Pregnant Swedish women register at a maternity-care unit in the 12th week of pregnancy and are followed regularly throughout their pregnancies. The nature of the social medical system is such that all pregnant women register. This study was based on all those women who registered at one specific maternity care unit in Göteborg and who later gave birth in the Göteborg area. At the time of registration (week 12), the women completed an extensive questionnaire containing personal information, including questions about present and previous back pain, working conditions, social background, level of education, earlier sick leave taken for back pain, and number of earlier pregnancies. Women with back pain were also asked to fill out a pain drawing and to describe their pain intensity on a visual analogue scale. At each of nine subsequent visits, they were asked if they had experienced pain in the previous period, and if so they completed another questionnaire, a pain drawing, and a visual analogue scale. Sick leave taken because of back pain during pregnancy also was recorded.

A number of biomechanically relevant parameters were recorded in weeks 12, 20, 24, 30, and 36. They included weight gain, abdominal circumference, sagittal and transverse abdominal diameters, the amount of lumbar lordosis, finger laxity, and striae distensae in the skin of the abdomen, thighs, and breasts. The abdominal circumference and diameters were recorded in centimeters using tape measures and protractors. The lumbar lordosis was estimated by measuring the perpendicular distance to the apex of the lumbar lordosis from a straight line connecting the apex of the thoracic kyphosis and the posterior part of the sacrum (Figure 1). Finger laxity was measured with the arm and hand in a relaxed, horizontal position by recording the maximal ulnar deviation angle of the fourth finger when a constant force of 1.7 N was applied to the distal phalanx of the finger (Figure 2). The laximeter for recording ulnar finger deviation was evaluated in two pilot studies. In one study, 10 nonpregnant women who were briefly instructed measured each other. Analysis of variance showed that the standard error of the mean was 0.48 degrees or 1.2% of the mean in that study. In another study,[13] 34 teenagers were measured by one trained physiotherapist and the results were related to the patients' Beighton scores. This score, which is considered a general measure of laxity, is based on the hypermobility of the thumb and fifth finger, the hyperextension of the knee and elbow, and on the ability to perform forward flexion of the spine. A significant correlation was found ($r = 0.55$, $P < 0.001$). From the first pilot studies, we concluded that the finger laximeter had acceptable reliability and from the second that its measures correlated well to another more complicated method of laxity measurement. Striae distensae were recorded as either present or not.

Several parameters at delivery, which were thought to be related to collagen tissue laxity, also were recorded. They included the softness of the uteral cervix, the so-called Bishop score (1–10), the time and mode of delivery, and the need for analgesia. The height and weight of the child at birth was also recorded. All mechanical parameters were related to back pain during pregnancy using the Pitman correlation test and comparison of means.[2]

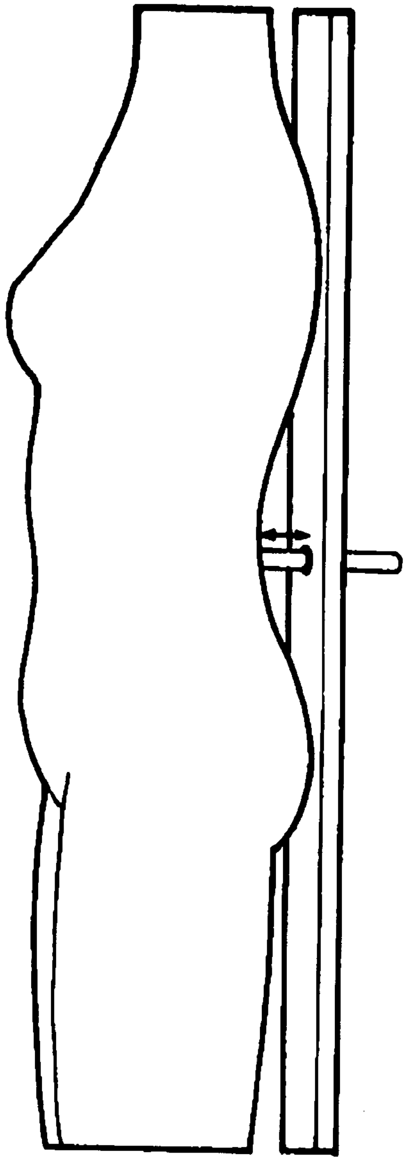

Figure 1. The depth of the lumbar lordosis was measured with a ruler.

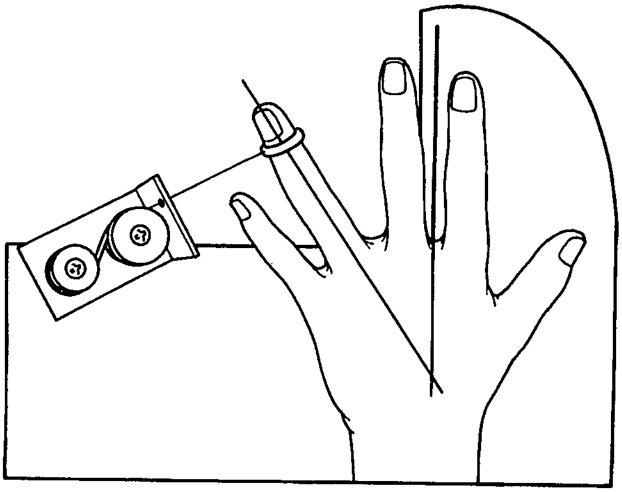

Figure 2. The ulnar deviation was measured by applying a constant normal force to the distal phalanx of the fourth left finger.

Results

From July 1, 1984 to the July, 1985, 950 women entered into the study. Thirty-three patients had late miscarriages, late abortions, or were actually not pregnant, reducing the target group to 917 women. During their pregnancy, 62 patients moved to other maternity care units and could not be followed continuously, reducing the study group to 855 women. The dropout group was analyzed based on the week 12 data and were found not to differ from the study group in parameters relevant to the study.

Forty-nine percent of the women complained of back pain one or several times. The mean values of the biomechanical parameters in weeks 12, 20, 24, 30, and 36 are listed in Table 1. Table 2 illustrates the change in biomechanical parameters and back pain during pregnancy. The lumbar lordosis was the only parameter that did not change significantly from the 12th to the 36th week.

Complaints of back pain in this pregnancy were found to correlate significantly with large sagittal ($r = 0.15$, $P < 0.01$) and large transverse abdominal diameters ($r = 0.13$, $P < 0.01$). Back pain was also statistically significantly correlated ($r = 0.11$, $P < 0.01$) to a large lumbar lordosis (Table 3).

The ulnar deviation angle behaved differently in primi- and multipregnant women. Primipregnant women began, on average with 34.5 degrees in week 12, which increased significantly to 36.3 degrees in week 24 ($P < 0.02$). Multipregnant women started with 36.4 degrees and showed no increase. In the group of primipregnant women, the mean ulnar de-

Table 1. Mean Values SEM for the Biomechanical Parameters Recorded and Percent of Women with Back Pain at Each Measurement Week (n=855)

Parameter	Week 12	Week 20	Week 24	Week 30	Week 36
Striae distensae (%)	40	40	42	42	46
Abdominal circumference (cm)	77 ± 0.3	83 ± 0.3	89 ± 0.9	95 ± 0.6	105 ± 1.9
Transverse diameter (cm)	25 ± 0.1	25 ± 0.1	26 ± 0.1	27 ± 0.1	28 ± 0.1
Sagittal diameter (cm)	18 ± 0.1	21 ± 0.1	23 ± 0.1	26 ± 0.1	28 ± 0.1
Lumbar lordosis (mm)	41 ± 0.3	41 ± 0.5	42 ± 0.3	42 ± 0.3	42 ± 0.3
Ulnar deviation (%)	36 ± 0.3	36 ± 0.3	37 ± 0.3	37 ± 0.3	37 ± 0.1
Weight (kg)	60	—	—	—	74
Back pain (%)	21	31	34	41	47

viation angle of the fourth finger in the 12th week was 35.5 degrees in the "no pain" group compared to 33.4 degrees in the group that had back pain in this pregnancy. This difference is statistically significant ($P < 0.05$). In the multipregnant group, however, no difference was found.

None of the other observations listed in Table 1 correlated significantly to back pain in this pregnancy. Dividing the women into primipregnant and multipregnant added some further information. The circumference of the abdomen increased earlier in multipregnant than in primipregnant women. In week 12, the measures were 78 cm and 76 cm, respectively; and in week 20 the multipregnant women measured 84 cm compared to 82 cm for the primipregnant women. Both differences are statistically significant ($P < 0.01$).

Back pain history, back pain intensity, type of back pain, height, weight, age, number of previous pregnancies, and previous use of oral contraceptives did not correlate to any of the biomechanical parameters.

Table 2. Change in Percent of the Biomechanical Parameters Listed in Table 1 from Week 12 to Week 36 (=855)

Parameter	Change (%)	*P* Value	Difference Significant in Week
Striae distensae	12	< 0.05	20
Abdominal circumference	36	< 0.05	20
Transverse diameter	12	< 0.05	20
Sagittal diameter	55	< 0.05	20
Lumbar lordosis	2	< NS	—
Ulnar deviation	4	< 0.05	24
Weight gain	22	< 0.05	—
Back pain	26	< 0.05	20

Height, weight at the beginning of pregnancy, rural background, use of oral contraceptives, time of onset of back pain in pregnancy, striae distensae, abdominal measures, lumbar lordosis, ulnar deviation angle, weight of the child, and maternal weight gain during pregnancy did not correlate with sick leave because of back trouble during pregnancy.

None of the parameters registered at delivery correlated with any of the biomechanical observations. Bishop score correlated with sick leave taken for back pain during pregnancy ($r = 0.17$, $P < 0.05$), but not with any biomechanical parameters.

■ Discussion

Back pain in pregnancy can not be explained solely by weight gain during pregnancy or by the general laxity of collagen tissue induced by pregnancy hormones. The theory that the large flexion moment on the lower back produced by the growing uterus and fetus contributes to the development of back pain was supported to some extent, however, as large sagittal and lateral diameters both correlated to back pain in pregnancy, although the correlations were weak. Biomechanical calculations were made to gauge the additional loads imposed on the spine by near-term pregnancy if loads were not compensated for by carrying the head, neck, and upper trunk in extension (see Appendix). The calculations suggest that the additional loads due to pregnancy are approximately equivalent to the loads that would be imposed on a nonpregnant woman who carried her trunk continuously flexed forward by 22.3 degrees.

The correlation found between back pain and lumbar lordosis is interesting in that other studies have failed to show this correlation in a nonpregnant population.[5] Because the lumbar lordosis was one of two biomechanical factors that did not change during pregnancy, we conclude that women with a naturally large lumbar lordosis are particularly susceptible to back pain when pregnant. This conclusion needs confirmation and may not be sound in every case because our method of measuring lacks precision. It is possi-

Table 3. Correlations Between Back Pain During Pregnancy and the Biomechanical Parameters.

Parameter	R Value	*P* Value
Striae distensae	0.02	NS
Amdominal circumference	0.08	NS
Transverse diameter	0.13	< 0.01
Sagittal diameter	0.15	< 0.01
Lumbar lordosis	0.11	< 0.01
Ulnar deviation	0.05	NS
Weight gain	0.05	NS
Oral contraception	(-)0.05	NS

ble that measures made with a kyphometer could give more precise information in this area.[10] Combining large lordosis with large abdominal sagittal diameter in the statistical analyses did not increase the correlation.

Striae distensae were found in one of every two subjects but did not correlate with either back pain in this pregnancy or to previous back pain. Striae are caused by insufficiencies in the subcutaneous collagen tissue during the rapid growth of the abdomen and were expected to correlate with large abdomens and high peripheral laxity. This proved to be the case, which provides indirect validation of our measures. The correlation between lumbar lordosis and striae can be explained by the correlation of lumbar lordosis with large abdomens.

The abdominal sagittal diameter was strongly correlated with peripheral laxity, perhaps as a result of collagen insufficiency caused by the hormones estrogen and relaxin. Women with high peripheral laxity may develop larger abdomens during pregnancy because of increased elasticity in the abdominal wall. This, in turn, increases the flexion moment on the back and may increase the risk of back pain. The fact that weight gain and the weight of the child were both correlated to abdominal sagittal diameter needs no further explanation.

Sick leave taken during pregnancy because of back pain in Sweden is not directly related to physically heavy work, because women who do heavy work are allowed to stop working at the beginning of the 32nd week of pregnancy without being required to take sick leave. The women taking sick leave because of back pain in this study, therefore, are women who were unable to continue doing light or moderately heavy work. This type of sick leave was found to correlate with pain intensity, which, of course, was expected. It was also primarily correlated to pain in the sacroiliac joint areas as opposed to other types of back pain.[1,11,12] This indicates that pain in the sacroiliac joint areas is more disabling than other types of back pain in pregnancy.

Age at first pregnancy also was correlated with sick leave taken during pregnancy, which correlates with obstetrical observations that younger women complain more often of pregnancy-related side-effects than do older pregnant women. We have no direct explanation for this. Younger women may be more sensitive to hormonal changes, and older women may have a different attitude to pregnancy.

We found a negative finding in this study interesting. We expected the lumbar lordosis to increase during the enlargement of the abdomen but this did not occur. The biomechanical significance of this is discussed in the Appendix.

The change in finger laxity was presumed to reflect the increased laxity in the pelvic joints found in all pregnant women as a necessary preparation for delivery.[3] Indeed, the ulnar deviation angle did change significantly as pregnancy progressed, particularly from the 12th to the 20th week. The increase in peripheral laxity during pregnancy was not uniform among the women, however, and was found primarily in the primipregnant group. In this group, the laxity in the 36th week was the same as in the 12th week for multipregnant women, indicating that increased peripheral laxity does not return to its prepregnancy state after delivery. Back pain did not correlate with laxity in multiparous women; the only correlation was found in primiparous women in the 12th week. An increased collagen laxity, if general in nature, would influence the ability of collagen tissue, including the ligaments and discs of the spine, to resist stretching. The data indicate that this change in stiffness is a minor risk factor for back pain in pregnant women. Bishop score correlated with sick leave taken for back pain during pregnancy but not with the occurrence of back pain as such. This is not entirely surprising because the Bishop score was correlated with pain intensity.

■ Conclusion

Back pain in pregnancy is a condition caused by many different factors. The primary biomechanical risk factor identified was the change in abdominal sagittal diameter. This parameter increased on average 55% from the 12th to the 36th week of pregnancy. It should be noted that even this factor was only weakly correlated with back pain in pregnancy. However, no other biomechanical factors had greater effect on the development of back pain in pregnancy. An initially large lumbar lordosis was a risk factor, although it did not change during pregnancy. Peripheral joint laxity was important only among primipregnant women, in whom the back pain group had decreased laxity.

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Appendix Estimate of the Mean Additional Loads Imposed on the Lumbar Trunk by Uncompensated Carriage of the Near-Term Fetal Material

A 22% gain in weight during gestational weeks 12 to 36 (Table 2) by a woman with a 60-kg body mass equals a 130 N absolute weight gain. At week 36, the mean sagittal diameter of the abdomen was 28 cm (Table 1). If the center of the growing fetus lies halfway along this diameter, then that center of mass lies 14 cm anterior to the spine. The additional flexion moment imposed on the lumbar spine at approximately the L3 level by uncompensated carriage of the near-term placenta and fetus is then 130 N times 14 cm, or 18.2 Nm.

Assuming the center of the extensors of the lumbar spine at the L3 level lies approximately 7.5 cm posterior of the center of the L3 vertebral body.[8] These extensors must then contract by 18.5 Nm/7.5 cm, or by 243 N, to balance this additional moment whenever the upper body segments are in an upright configuration. Furthermore, the additional 243 N contraction force in the extensors must be balanced by a 243 N lumbar spine compression increase.

To place these numbers in perspective, consider by how much a nonpregnant woman would have to flex her body segments above the L3 level to produce these same load increases. Assume that approximately 40% of the body weight, or 240 N in the mean, is superior to the L3 level and that the center of this mass lies approximately 20 cm superior to the L3 level. The increase in flexion moment to be balanced by leaning the upper body segments forward is then 240 N × 20 cm × the angle of flexion. Flexing those segments 22.3 degrees would then impose a 22.3 Nm additional flexion moment on the lumbar spine. Thus upright carriage of the near-term fetus in an average-sized pregnant woman, if not compensated for, would impose the same additional loads on the trunk structures that a continuous 22.3 degree forward lean of the body segments superior to the L3 level would impose on a nonpregnant woman. Pregnant women can, in fact, compensate at least in part for the additional flexion moment by counterbalancing it through extension of the upper trunk head and neck. The fact that we detected no change in the depth of the lumbar lordosis during pregnancy indicates that any increase in lumbar lordosis is subtle. Apparently pregnant women compensate for the flexion moment by hip joint extension rather than lumbar spine extension.