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A Study on Echogenic Changes of the Kidney-Spleen and Liver-Spleen Contrasts with Age in Infants and Children

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Individual echogenicities of the kidneys and liver cannot be determined using only the generally accepted right kidney/liver contrast ratio. We therefore determined 5 new contrast ratios: i) left kidney to spleen (Kl/SP); ii) right kidney to spleen (Kr/SP); iii) liver to spleen (L/SP); iv) Kl/SP – Kr/L; and v) (Kl/SP)/(Kr/L); with the last 2 being designated as modified ratios alternative to the L/SP ratio. We used the new ratios with computerized histogram sonography to assess echogenicitic changes in the kidneys, liver and spleen with age for 257 normal children ranging from birth to 15 years old. The values in Kr/L, Kl/SP and Kr/SP increased from day 0 to day 2 in early neonates (values > 1), decreased between day 2 to 1 month, and thereafter remained constant. The values were < 1 from 1 month to 15 years. The values in L/SP ratio were constant from birth through 6 months (values > 1), and thereafter decreased gradually to a value of approximately 1 and remained constant in the later periods. We found the relationship Kl/SP < Kr/L in early neonates, Kl/SP > Kr/L in infants and Kl/SP < Kr/L again in the later periods. These results suggest that the renal cortical echogenicity was higher than that of the liver and splenic parenchyma in early neonates and that the echogenicity of the liver was greater than that of the spleen in children from birth to 6 months. Combining these ratios is thus helpful for assessing renal and hepatic echogenicity individually. Moreover, significantly higher echogenicity of the liver was observed in patients with hepatic dysfunction (n = 21) for children of all age groups, as observed by the L/SP contrast ratio. Significantly increased echogenicity of the liver was observed by modified ratios in children aged 1 year or older. We suspect that the L/SP and modified ratios are useful for assessing the echogenicity of the liver in patients with liver dysfunction.

Key words: childhood; computerized histogram sonography; liver; kidneys; spleen

It is well established that the liver-kidney contrast by echographic findings varies until 2 to 3 months after birth and thereafter becomes constant, although a few authors have suggested that the period of variation lasts as long as 6 months (Haller et al., 1982; Han and Babcock, 1985) or even 1 year (Winkler and Altrogge, 1985). Using sonography, Hricak and colleagues (1983) observed the features of an adult kidney as early as the 2nd year of life, and attributed this to the gradual change of the neonatal kidney. This concept is well supported by the general belief that liver-kidney echogenicity remains unchanged after a certain period. But determination of the echogenicity of the kidneys is encumbered by inherent limitations—the standard to which the kidney is compared may not be normal itself—and a

Abbreviations: Kl < Kr, left renal echogenicity < right renal echogenicity; Kl/SP, left kidney to spleen; Kl/SP – Kr/L, Kl/SP minus Kr/L; (Kl/SP)/(Kr/L), Kl/SP divided by Kr/L; Kr/L, right kidney to liver; Kr/SP, right kidney to spleen; L > SP, hepatic echogenicity > splenic echogenicity; L/SP, liver to spleen; ROI, region of interest; SGPT,serum glutamic-pyruvic transaminase; SGOT, serum glutamic-oxaloacetic transaminase; SP > L, splenic echogenicity > hepatic echogenicity

Table 1. The values of echointensity in 6 contrast ratios in different childhood age groups (birth to 15 years) in normal children (*n* = 257)

Age group		Contrast ratio					
		Kr/L	Kl/SP	Kr/SP	L/SP	Kl/SP – Kr/L	(Kl/SP)/(Kr/L)
d0	[16]	1.084±0.164	1.037±0.186	1.185±0.278	1.092±0.189	–0.047±0.244	0.978±0.237
d2	[17]	1.211±0.175	1.129±0.169	1.332±0.224	1.110±0.177	–0.082±0.217	0.946±0.172
d5	[15]	1.072±0.124	0.977±0.167	1.207±0.140	1.137±0.169	–0.095±0.227	0.927±0.216
m1	[40]	0.918±0.195	0.938±0.213	0.994±0.261	1.096±0.235	0.020±0.276	1.059±0.288
m3–4	[23]	0.873±0.150	0.872±0.163	0.959±0.234	1.100±0.206	–0.001±0.192	1.019±0.227
m5–6	[6]	0.839±0.124	0.886±0.135	0.885±0.072	1.081±0.244	0.047±0.189	1.082±0.273
m7–8	[5]	0.853±0.170	0.834±0.167	0.772±0.086	0.918±0.100	–0.019±0.291	1.023±0.355
m9–11	[12]	0.797±0.089	0.886±0.118	0.826±0.157	1.034±0.126	0.089±0.154	1.126±0.210
y1–2	[17]	0.857±0.130	0.889±0.156	0.858±0.189	1.011±0.219	0.032±0.210	1.060±0.255
y3–4	[15]	0.871±0.169	0.855±0.171	0.876±0.178	1.018±0.160	–0.017±0.092	0.989±0.117
y5–6	[21]	0.901±0.212	0.828±0.139	0.788±0.181	0.909±0.272	–0.073±0.289	0.974±0.292
y7–8	[13]	0.883±0.146	0.883±0.188	0.928±0.203	1.070±0.287	–0.000±0.285	1.042±0.359
y9–10	[13]	0.871±0.265	0.916±0.164	0.904±0.221	1.061±0.190	0.045±0.206	1.101±0.263
y11–13	[16]	0.869±0.126	0.836±0.113	0.792±0.146	0.915±0.131	–0.034±0.111	0.971±0.123
y13–15	[28]	0.914±0.220	0.895±0.152	0.878±0.213	0.973±0.204	–0.019±0.187	1.002±0.082

Mean ± SD.
[ ], number of subjects.
Kl/SP, left kidney to spleen; Kl/SP – Kr/L, Kl/SP minus Kr/L; (Kl/SP)/(Kr/L), Kl/SP divided by Kr/L; Kr/L, right kidney to liver; Kr/SP, right kidney to spleen; L/SP, liver to spleen.
d0, 0 day old; d2, 2 days old; d5, 5 days old; m1, 1 month old; m3–4, 3 to 4 months old; m5–6, 5 to 6 months old; m7–8, 7 to 8 months old; m9–11, 9 to 11 months old; y1-2, 1 to 2 years old; y3–4, 3 to 4 years old; y5–6, 5 to 6 years old; y7–8, 7 to 8 years old; y9–10, 9 to 10 years old; y11–12, 11 to 12 years old; y13–15, 13 to 15 years old.

standard reference for kidneys is needed. If, in late childhood and adult life, the kidney is of the same echogenicity as the liver, this should not be considered an indicator of renal disease (Eggert et al., 1991). In fact, the idea that hepatic echogenicity remains constant is still only an assumption. The physiological increase in echogenicity of the liver may be disguised by pathological causes (Eggert et al., 1991). Or the echogenicity of the liver and kidneys may vary with certain physiological and pathological changes, e.g., primary viral infections of the liver and kidneys, physiological hepatomegaly in early infants, or quantitative changes in renal anatomy in early infants (Hricak et al., 1983). Structural collagenous changes also have an impact on echogenicitic changes within these organs during physiological processes (Harkness and Nightingale, 1962; Harkness and Harkness, 1963; Fields and Dunn, 1973; Rosenfield et al., 1980), as well as during pathological processes (Fields and Dunn, 1973; Simpson, 1957; Rosenfield et al., 1980). Other biological components, such as fat, calcium, proteins, the volume of cellular elements, hepatic perfusion, etc. (Rosenfield et al., 1980), may play important roles in echogenic variation with age. Clearly, it is rather difficult to construct a rigid contrast ratio, and by using only the generally accepted liver and kidney contrast, we cannot determine the variation in echogenicity for different childhood age groups. It is therefore necessary to establish the actual parameters of kidney and liver echogenicity in various childhood age groups.

To this end, we determined the i) right kidney to spleen (Kr/SP); ii) left kidney to spleen (Kl/SP); iii) liver to spleen (L/SP); iv) Kl/SP – Kr/L; and v) (Kl/SP)/(Kr/L) ratios for the purpose of establishing the changes in echogenicity of the liver and kidney individually. Kl/SP – Kr/L and (Kl/SP)/(Kr/L) are modified ratios alternative to the L/SP.

Subjects and Methods

Sonograms were performed in 278 male and female children from 0 to 15 years of age at the out- and inpatient departments of our hospital. Among the 278 children, 257 were normal and 21 were patients with hepatic dysfunction. Informed, written consent was obtained from the parents or guardians of each subject prior to the sonogram study. Children were considered normal if they only showed signs of hepatomegaly, but were negative for both hepatic dysfunction (laboratory evidence) and kidney diseases (clinical and/or laboratory evidence). Hepatic dysfunction was defined in 21 cases by a steep rise in serum transaminases. Of the 257 normal children examined, 68 were neonates, and the rest were non-neonates. Correlation with clinical status (hematuria, proteinuria, hepatomegaly, jaundice) and laboratory data [blood urea nitrogen, serum creatinine, serum glutamic-pyruvic transaminase (SGPT) and serum glutamic-oxaloacetic transaminase (SGOT)] served as the basis for defining the renal and hepatic status of each patient. Renal function was considered abnormal if the serum creatinine level exceded 1.0 mg/dL. Liver function abnormality was based on SGPT and SGOT of greater than 47 IU/L. We divided neonates into 3 age groups: 0 day of birth (d0), 2nd day of birth (d2) and 5th day of birth (d5); and the non-neonates into 12 age groups: 1 month (m1), 3 to 4 months (m3–4), 5 to 6 months (m5–6), 7 to 8 months (m7–8), 9 to 11 months (m9–11), 1 to 2 years (y1-2), 3 to 4 years (y3–4), 5 to 6 years (y5–6), 7 to 8 years (y7–8), 9 to 10 years (y9–10), 11 to 12 years (y11–12) and 13 to 15 years (y13–15) (Table 1).

We used an ultrasonogram machine ALOCA SSD 670 with either 5.00 or 3.5 MHz transducers of a convex type. A 5.00 MHz transducer was used in neonates and young children of small body size and a 3.5MHz transducer in older children, and young children of large body size. Gain settings and Sensitivity Time Control (STC) were adjusted and kept constant for the most satisfactory visualization of parenchyma of the organs throughout each examination. All patients were scanned in supine positions in the longitudinal plane. In the case of neonates, the babies were placed in the supine position and then rotated into a right lateral position with the transducer moving from back to front as the spleen and left kidney were adequately visualized. Newborn babies were examined in an incubator with minimal handling and disturbance. Babies were kept warm by radiant heat lamps. No sedation was used. Two people were present at the time of the study. One assisted by restraining the baby while the other scanned. The neonates were placed in a supine or decubitus position for examination of the left kidney and spleen, and this position was maintained for the right kidney and liver. The 5.00 MHz transducer was favored as the standard choice (Frank et al., 1979).

The histogram analysis with region of interest (ROI) was used for measuring the intensities of echogenicity. The relative number of echoes of given intensities were plotted on the vertical axis against the varying levels of echobrightness on the horizontal axis. The histogram ROIs were placed closely in the most clear and homogeneous areas of the hepatic parenchyma, renal cortex and splenic parenchyma, as these regions were nearly equidistant from the transducer (Fig. 1). Because placing an object within the near or far field of the transducer may alter echogenic properties (Carson and Oughton, 1977; Taylor et al., 1979; Jaffee and Taylor, 1979), we selected a transducer that would include the area to be studied within the focal zone of the transducer.

Histogram analysis

The histograms represent the echo-distribution intensity of the ROI area. The horizontal axis represents 0 to 63 shades of gray, and the vertical axis the distribution ratio of each shade. Here the number of picture elements (pixels) of the most common shades in the ROI areas were assumed to be 100%. (T, total number of pixels in ROI; L, gray scale level of shade component that is most common in the ROI; M, number of pixels of the shade component that is most common in the specified area).

Statistical analysis

All data were expressed as mean ± SD. Differences between the variables in multiple groups were assessed using both parametric and non-parametric analyses due to an insufficient number of data. One-way analysis of variance and a post hoc test [Fisher's Protected Least Significant difference (PLSD) method] was used to calculate between the 2 variables in multiple groups. Differences between more than 2 groups (multiple groups) were assessed using the Kruskal-Wallis test. Student's *t*-test was used to compare between patients with liver dysfunction and normal controls. Correlations between variables were performed by regression analysis. A value of $P < 0.05$ was considered significant.

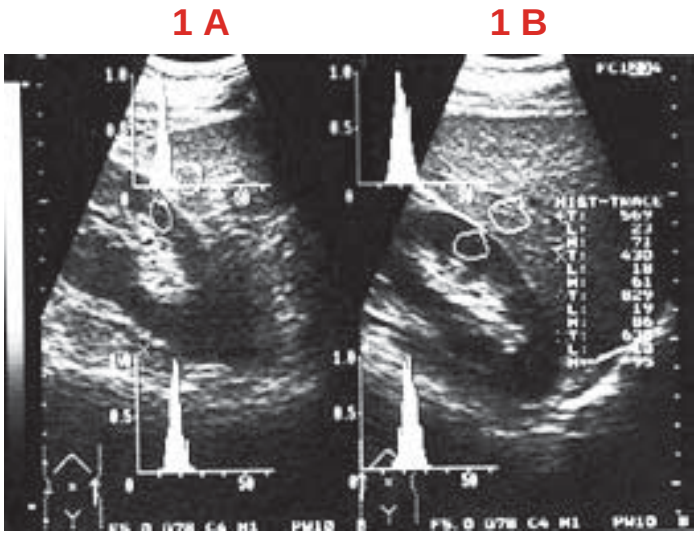

Fig. 1. A computerized histogram analysis of the right renal cortex, liver and splenic parenchyma by longitudinal scan. **A**: The portion of the left renal cortex and splenic parenchyma that is of interest is demarcated by a circle. The regions of interests (ROIs) are placed in the left renal cortex and splenic parenchyma at the same distance from the transducer. **B**: The portion of the right renal cortex and hepatic parenchyma that is of interest is demarcated by a circle. The ROIs are placed in the right renal cortex and hepatic parenchyma at the same distance from the transducer.

Results

Correlations in echogenicity between the right and left kidneys

In non-neonates ($n = 209$), statistically significant correlation in echogenicity was found between right and left kidneys ($r = 0.752$) (Fig. 2). In early neonates ($n = 48$), there was no correlation between the echogenicity of the right and left kidneys ($r = 0.075$) (Fig. 3)

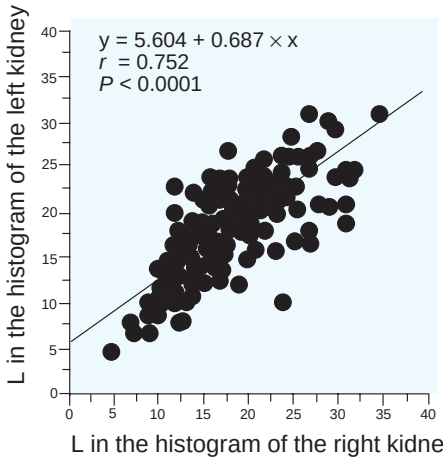

Fig. 2. Relationship in echogenicity between the left and right renal cortices during the non-neonatal period. L, the gray scale level of the shaded component that is most common in ROI; ●, L in the histogram of the left and right renal cortices.

Correlations in echogenicity between the liver and spleen

A significant correlation was found between the echogenicity of liver and spleen in non-neonatal children ($r = 0.677$) (Fig. 4). A weak correlation was seen between the echogenicity of liver and spleen in early neonates ($r = 0.334$) (Fig. 5). No significant differences in echogenicity were found between males and females.

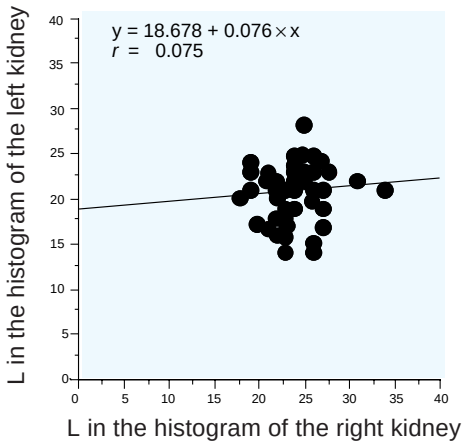

Fig. 3. Relationship in echogenicity between left and right renal cortices during the early neonatal period. L, the gray scale level of the shaded component that is most common in ROI; ●, L in the histogram of the left and right renal cortices.

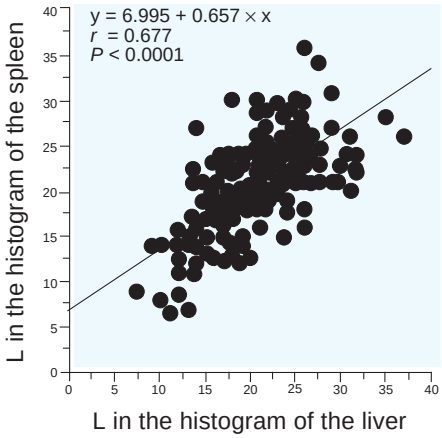

Fig. 4. Relationship in echogenicity between hepatic and splenic parenchyma during the non-neonatal period. L, the gray scale level of the shaded component that is most common in ROI; ●, L in the histogram of the hepatic and splenic parenchyma.

The Kr/L contrast ratio in normal children

The Kr/L contrast ratio in normal children (n = 257) increased from d0, peaked at d2, decreased until 1 month of age, and thereafter became constant (Fig. 6). The average value was higher than 1 between d0 to d5 and lower than 1 between m1 to 15 years. Statistically significant changes were seen from birth to 15 years ($P < 0.001$) by the Kruskal-Wallis test. Significant changes were also observed between d0 and d2 ($P < 0.05$); d0 and each group of months and years ($P < 0.01$); d2 and d5 ($P < 0.05$); d2 and each group of months and years ($P < 0.001$); and d5 and each of the following: m3–4, m5–6, m7–8, m9–11, y1–2, y3–4, y5–6 and y7–8 ($P < 0.01$) as analyzed by the Fisher PSLD test.

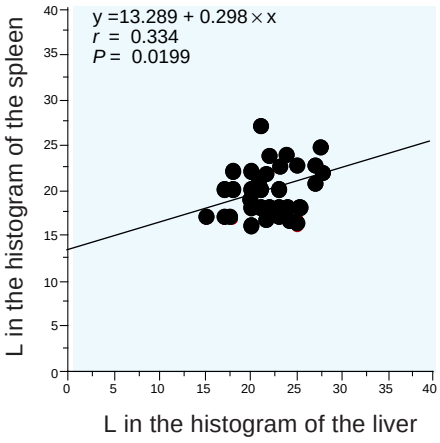

Fig. 5. Relationship in echogenicity between hepatic and splenic parenchyma during the early neonatal period. L, the gray scale level of the shaded component that is most common in ROI; ●, L in the histogram of the hepatic and splenic parenchyma.

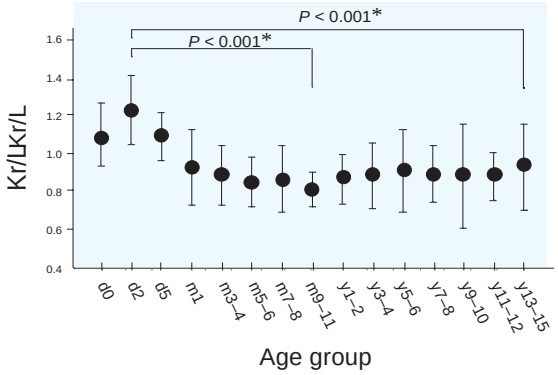

Fig. 6. Echointensity of the right renal cortex and hepatic parenchyma in the Kr/L contrast ratio versus different childhood age groups: d0, 0 days old; d2, 2 days old; d5, 5 days old; m1, 1 month old; m3–4, 3 to 4 months old; m5–6, 5 to 6 months old; m7–8, 7 to 8 months old; m9–11, 9 to 11 months old; y1-2, 1 to 2 years old; y3–4, 3 to 4 years old; y5–6, 5 to 6 years old; y7–8, 7 to 8 years old; y9–10, 9 to 10 years old; y11–12, 11 to 12 years old; y13–15, 13 to 15 years old. *Kruskal-Wallis test.

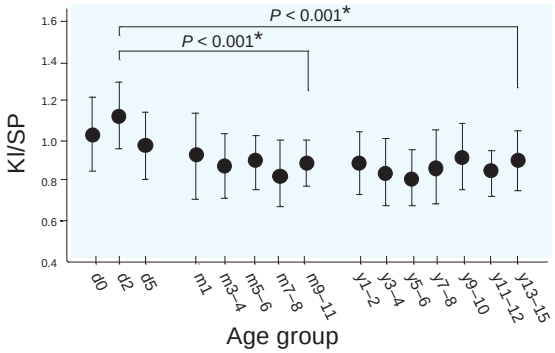

Fig. 7. Echointensity of the left renal cortex and splenic parenchyma in the Kl/SP contrast ratio versus different childhood age groups: age classification is the same as in the legends to Fig. 6. *Kruskal-Wallis test.

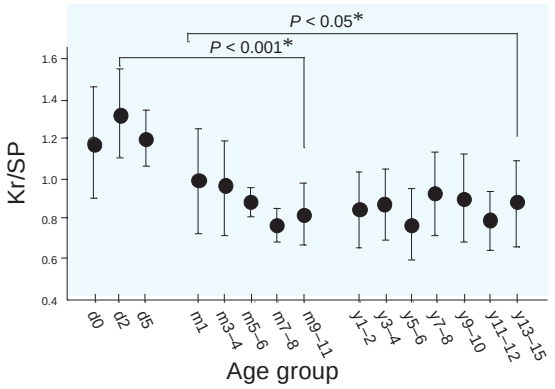

Fig. 8. Echointensity of the right renal cortex and splenic parenchyma in the Kr/SP contrast ratio versus different childhood age groups: age classification is the same as in the legends to Fig. 6. *Kruskal-Wallis test.

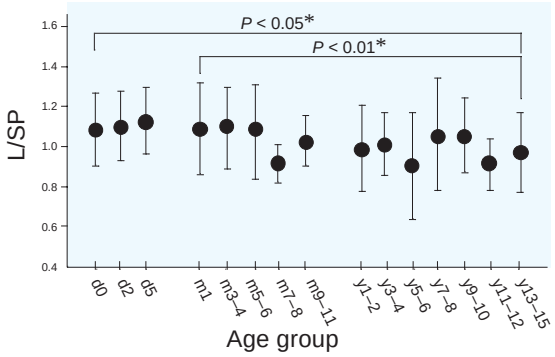

Fig. 9. Echointensity of the hepatic and splenic parenchyma in the L/SP contrast ratio versus different childhood age groups: age classification, the same as in the legends to Fig. 6. *Kruskal-Wallis test.

The Kl/SP contrast ratio in normal children

The values of this ratio increased from d0, peaked at d2 and decreased until 1 month of age (n = 257) (Fig. 7). After 1 month, the values became constant. The average value was greater than 1 between d0–d2, equal to 1 at d5, and less than 1 between m1–15 years. Statistically significant changes were seen from birth to 11 months ($P < 0.001$), and from birth to 15 years ($P < 0.001$) analyzed by the Kruskal-Wallis test. Significant changes were seen between d0 and m1 ($P < 0.05$); d0 and each of the following: m3–4, m7–8, m9–11, y1–2, y3–4, y5–6, y7–8 and y11–12 ($P < 0.01$); d2 and d5 ($P < 0.05$); and d2 and each group of months and years ($P < 0.01$) by the Fisher PSLD test.

The Kr/SP contrast ratio in normal children

The values of this ratio increased from d0, peaked at d2, and decreased until 1 month of age (n = 257) (Fig. 8). After 1 month, the values became constant. The average value was greater than 1 between d0 to d5, and less than 1 between m1 to 15 years. Significant changes were seen from birth to 15 years ($P < 0.001$), and from 1 month to 15 years ($P < 0.05$) as analyzed by the Kruskal-Wallis test. Significant changes were found between d0 and d2 ($P < 0.05$); d0 and each group of months and years ($P < 0.01$); d2 and each group of months and years ($P < 0.001$); d5 and each group (except m5–6) of months and years ($P < 0.001$); m1 and each of the following: m7–8, y1–2, y5–6, y11–12 and y13–15 ($P < 0.05$); and m3–4 and each of these: y5–6, y11–12 ($P < 0.05$) as analyzed by the Fisher PSLD test.

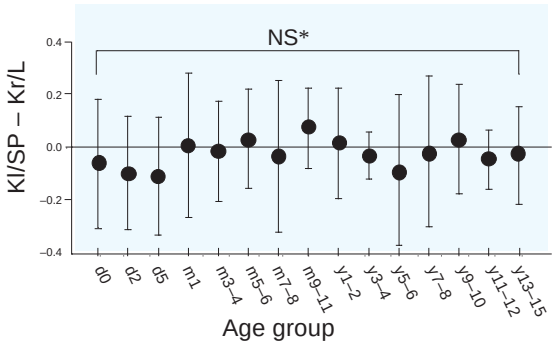

Fig. 10. Echointensity of the hepatic and splenic parenchyma in the Kl/SP – Kr/L contrast ratio versus different childhood age groups: age classification is the same as in the legends to Fig. 6. *NS, not significant in statistical difference with the Kruskal-Wallis test.

The L/SP contrast ratio in normal children

The values of this ratio increased from birth through 6 months and decreased at 7 to 8 months (n = 257) (Fig. 9). After that, the values increased slightly and became constant later on. The average value was greater than 1 between birth to 6 months, and almost equal to 1 in the later period. Statistically significant changes were observed between birth to 15 years ($P < 0.05$); 1 month to 15 years ($P < 0.01$) as analyzed by the Kruskal-Wallis test. Significant changes were also observed between d0 and y5–6 and y11–12 each ($P < 0.05$); d2 and y5–6, y11–12 and y13–15 each ($P < 0.05$); d5 and m7–8, y5–6, y11–12 and y13–15 each ($P < 0.05$); m1 and y5–6, y11–12 and y13–15 each ($P < 0.05$); m3–4 and y5–6, y11–12 and y13–15 each ($P < 0.05$); and y5–6 and y7–8, y9–10 and y11–12 each ($P < 0.05$) as analyzed by the parametric test (Fisher PSLD method).

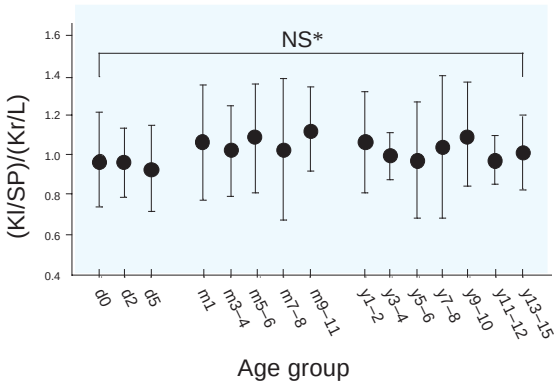

Fig. 11. Echointensity of the hepatic and splenic parenchyma in the (Kl/SP)/(Kr/L) contrast ratio versus different childhood age groups: age classification, the same as in the legends to Fig. 6. *NS, not significant in statistical difference with the Kruskal-Wallis test.

Inconsistent results were observed for correlations in echogenicity between the kidneys (left and right) as well as between the liver and spleen in the early neonatal period. Thus, the accuracy of the L/SP ratio result in which hepatic echogenicity was higher than splenic in the early neonatal period is open to question. To resolve these discrepancies for echogenicity in the early neonatal period we produced 2 modified ratios, Kl/SP – Kr/L and (Kl/SP)/(Kr/L).

The Kl/SP – Kr/L contrast ratio in normal children

We observed that the results for echogenicity was Kl/SP < Kr/L in early neonates and Kl/SP > Kr/L in the infantile period (n = 257) (Fig. 10). After the infantile period we again observed that the echopattern was Kl/SP < Kr/L, and that this pattern continued in the later periods. Significant changes were found between d2 and m9–11; d5 and m9–11; and m9–11 and y5–6 ($P < 0.05$) by the Fisher PSLD test.

The (Kl/SP)/(Kr/L) contrast ratio in normal children

The result for echogenicity in this ratio was similar to that in the Kl/SP – Kr/L contrast ratio (n = 257) (Fig. 11). Statistically significant differences were found between d2 and m9–11; and d5 and m9–11 ($P < 0.05$) by the Fisher PSLD test.

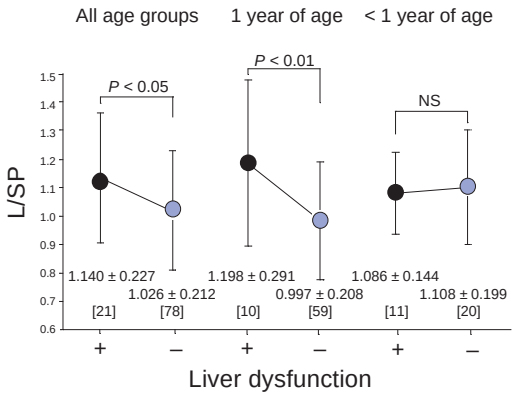

Fig. 12. Difference in echogenicity of the hepatic parenchyma in the L/SP contrast ratio between patients with hepatic dysfunction (●) and normal controls (●). Children of all age groups (left), children aged 1 year or older (middle) and children younger than 1 year (right). +, hepatic dysfunction; –, no hepatic dysfunction. [ ], number of subjects.

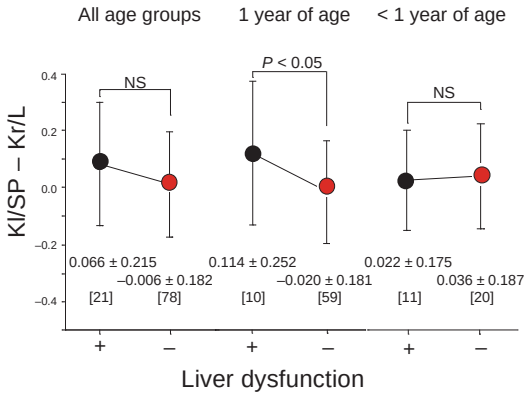

Fig. 13. Difference in echogenicity of the hepatic parenchyma in the Kl/SP – Kr/L contrast ratio between patients with hepatic dysfunction (●) and normal controls (●). Children of all age groups (left), children aged 1 year or older (middle) and children younger than 1 year (right). +, hepatic dysfunction; –, no hepatic dysfunction. [ ], number of subjects.

Echogenicitic changes of the liver in patients with hepatic dysfunction and normal controls

In the L/SP contrast ratio: A statistically significant difference in the echogenicity of the liver was observed between patients with hepatic dysfunction (n = 21) and normal controls (n = 79) ($P < 0.05$) in children of all age groups (Fig. 12). A significant difference in echogenicity of liver was observed between patients with hepatic dysfunction (n = 10) and normal controls (n = 59) in children aged 1 year or older ($P < 0.01$), but no significant difference was observed between the hepatic dysfunction (n = 11) and normal controls (n = 20) in children aged less than 1 year.

In the Kl/SP–Kr/L contrast ratio: A statistically significant difference in the echogenicity of the liver was seen between normal controls and patients with hepatic dysfunction in children aged 1 year or older ($P < 0.05$), but this difference was absent in children aged less than 1 year (Fig. 13). Differences were analyzed by Student's t-test.

In the (Kl/SP)/(Kr/L) contrast ratio: The result in this ratio was similar to that found in Kl/SP–Kr/L ratio (Fig. 14).

In the Kr/L contrast ratio: No statistically significant difference in the echogenicity of the liver was seen between the patients with hepatic dysfunction and normal controls (Fig. 15).

Discussion

Ultrasonography is being utilized with increasing frequency to evaluate renal and hepatic parenchymal diseases in both children and adults (Gosink et al., 1979; Joseph et al., 1979; Kurtz et al., 1980; Henschke et al., 1982; Winkler and Altrogge, 1985; Benbridge et al., 1986; Platt et al., 1988; Kraus et al et al., 1990).

A number of previous articles have assessed childhood kidney and liver sizes (Wladimiroff and Sekeris, 1976; Haugstvedt and Lundberg, 1980; De Vries and Levene, 1983; Dinkel et al., 1985; Scott et al., 1990; Gupta et al., 1993) and volumes (Holloway et al., 1983; Chiara et al., 1993; Sargent and Gupta, 1993). To our know-

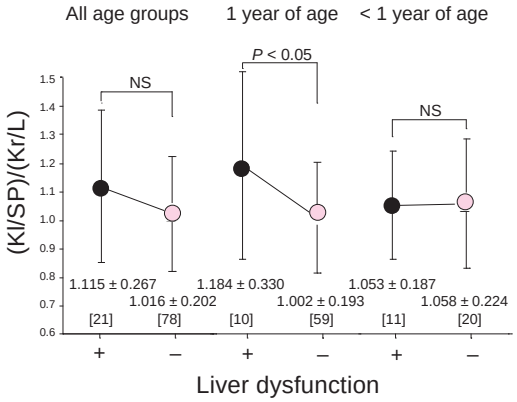

Fig. 14. Difference in echogenicity of the hepatic parenchyma in the (Kl/SP)/(Kr/L) contrast ratio between patients with hepatic dysfunction (●) and normal controls (○). Children of all age groups (left), children aged 1 year or older (middle) and children younger than 1 year (right). +, hepatic dysfunction; –, no hepatic dysfunction. [ ], number of subjects.

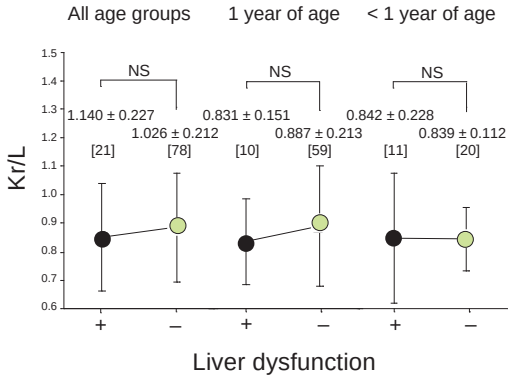

Fig. 15. Difference in echogenicity of the hepatic parenchyma in the Kr/L contrast ratio between patients with hepatic dysfunction (●) and normal controls (●). Children of all age groups (left), children aged 1 year or older (middle) and children younger than 1 year (right). +, hepatic dysfunction; –, no hepatic dysfunction. [ ], number of subjects.

ledge, however, no assessment has been made on changes in the echogenicity of liver and renal parenchyma over a broad time span in childhood (e.g., birth to 15 years). Our study was designed to determine the changes in echogenicity of the liver and renal parenchyma by computerized histogram sonography in normal children of various age groups, thus allowing the identification of variances from the normal. We also used this technique to evaluate the echogenicity of the liver in patients with hepatic dysfunction and in normal controls. Echogenicitic assessments of the renal cortex by the Kr/L contrast have already been presented in several reports (Haller et al., 1982; Hayden et al., 1984; Erwin et al., 1985; Han et al., 1985; Cramer et al., 1986). Hepatic parenchymal echogenicity, to which renal cortical echogenicity is compared in this study, has been taken as the standard in several studies using the KR/L contrast ratio. However, it is difficult to evaluate the renal echogenicity without knowing the hepatic echogenicity. We therefore used 5 new parameters to investigate the echogenicity of the liver and kidneys individually.

Our results showed a positive correlation in echogenicity between the left and right neonatal kidneys (Fig. 2), which was consistent with the results of previous studies (Han and Babcock, 1985; Emamian et al., 1993). This study was unable to show a correlation in echogenicity between the left and right early neonatal kidneys (Fig. 3). The cause of these discrepant results for the early neonatal period are unknown to us, though a few factors might possibly be implicated: i) inadequate visualization of the left kidney; ii) inconsistent renal perfusions (left and right); and iii) the differences in body positions and body-wall thickness. The inadequate visualization of the left kidney might further be associated with large stomach size, overdistention of the intestines, overcrowding of the ribs, and the short time permitted for echography, etc. A few studies have reported technical difficulties in performing sonography in neonatal babies (e.g., Frank et al., 1979), but we could find no similar reports on perfusion distributions to the early neonatal kidneys. An extensive study is needed in this area.

Our present study shows that renal cortical echogenicity (left and right) was greater than that of the hepatic and splenic parenchyma (Figs. 6, 7 and 8) during the early neonatal

period, and that with the infant's growth it then progressed through a predictable change from increased echogenicity to echogenicity less than that of the liver and spleen. It should be noted that no patients in our study manifested renal disease, hypertension, sepsis or hypoxemia, so these factors cannot, therefore, be implicated in the increased echogenicity. The above results are in contradiction to previously published reports using the generally accepted Kr/L ratio. Hricak and coworkers (1983) and Hayden and colleagues (1984) stated that renal cortical echogenicity was normally as echogenic as the liver parenchyma during the neonatal and young infantile period. Han and Babcock (1985) compared the echogenicity of the kidneys (left and right) with that of the liver and spleen in 122 children (newborn to 17 years old) and were not able to show that the neonatal renal cortical echogenicity was greater. These prior authors used only naked-eye evaluation to determine echogenicity. Eggert and coworkers (1991) proved by a densitometric study in infants that naked-eye evaluation did not permit a clear distinction between the categories of echogenicity, and also that the assessment of individual examiners varied considerably. Our ultrasound study was performed by computerized histogram analysis. To our knowledge, very few data on histogram sonography have been published until recently. Rosenfield and Siegel (1981) studied medical renal diseases by computerized histogram sonography, and graded renal cortical echogenicity by comparing the amplitude of echoes in the renal cortex with that of the liver, spleen and renal sinus. Bondestam and others (1992) examined the correlations of liver echointensity with cytology and chemical measurements of fat, water and protein content in live burbots (lota lota) fish by using computerized histogram sonography.

In the L/SP contrast ratio, we observed that the echogenicity of the liver was higher than that of the spleen from birth through 6 months, after which it gradually decreased till it was equal to that of the spleen (Fig. 9). We were uncertain of the accuracy of results concerning the echogenicity of the liver and spleen in the L/SP contrast ratio in early neonates. Factors such as weakly correlated echogenicity between the liver and spleen (Fig. 5), or negatively correlated echogenicity between the left and right kidneys during the early neonatal period (Fig. 3) might have contributed to these ambiguous results. To resolve such a discrepant relationship in echogenicity between the left and right kidneys in the early neonatal period, we produced 2 modified ratios, Kl/SP – Kr/L and (Kl/SP)/(Kr/L). The results for echogenicity of the liver and spleen in modified ratios was SP > L or Kl < Kr in the early neonatal period (Fig. 10). If the result for echogenicity of the liver and spleen in the L/SP contrast ratio, i.e., L > SP, was true in the early neonatal period, then we can say the result observed in modified ratios was Kl < Kr. A good correlation between the echogenicities of the kidneys of the left and right sides (Fig. 2), as well as between those of the liver and spleen did in fact exist in the non-neonatal period (Fig. 4). Therefore, the results for echogenicity of the liver and spleen in L/SP (Fig. 9), as well as in modified ratios (Figs. 10 and 11) during the non-neonatal period can be accepted as reliable. A study regarding perfusion distribution to the kidneys in the early neonatal period is needed. The result obtained in the L/SP ratio for echogenicity of the liver and spleen (Fig. 9) was in good agreement with previous findings that the liver is always higher in intensity than the spleen (Rosenfield et al., 1981; Sanders, 1991). Emamian and workers (1993) showed that the echogenicity of the spleen in young adults was higher than that of the liver. Our results in modified ratios were partially consistent with the reports of Emamian and workers, but further studies will be needed in this regard.

The results for our new ratios measured by computerized histogram sonography revealed that the echogenicity of the liver was significantly higher in patients with hepatic dysfunction (serum transaminases more than 47 IU/L) than that of normal controls (serum transaminases within normal limits). It should be noted that a significantly increased echogenicity of the liver was found in the L/SP ratio in children aged from birth through 15 years (Fig. 12), but only in children of age 1 year or

older using modified ratios (Figs. 13 and 14). More studies are needed to further evaluate echogenicity of the liver by computerized histogram sonography, especially in children aged less than 1 year. This study was unable to show a statistically significant difference in echogenicity of the liver in children with hepatic dysfunction and normal controls by using a traditional Kr/L contrast ratio (Fig. 15).

We can conclude that i) the values of ratios, i.e., Kl/SP, Kr/SP and Kr/L started to increase from d0, came to their peak at d2, and thereafter decreased until 15 years of age; ii) the values in the L/SP ratio increased (> 1) during the period from birth through 6 months, and thereafter decreased slightly to the level of 1 and remained constant; iii) the significance of the modified ratios in the early neonatal period remains ambiguous; iv) there is a greater possibility of increased echogenicity in hepatic parenchyma in children aged 1 year or older with high levels of serum transaminases (increased SGPT and SGOT); v) our new ratios may provide some advantages over the generally accepted (traditional) Kr/L contrast ratio for determining hepatic and renal echogenicity individually and more accurately; and vi) echogenicitic assessment of renal and hepatic parenchyma by naked-eye evaluation is not reliable.

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