Table Pyloric dimensions in infants studied. Results are mean (± 2 SD)

	Anterior-posterior diameter (mm)	Muscle thickness (mm)
Newborn infants (n=1391)*	8.8 (± 2.2)	2.1 (± 0.6)
Range	7.0–11.0	1.0–3.0
Infantile hypertrophic pyloric stenosis (n=9)	15.7 (± 0.6)	4.9 (± 0.14)
Range	13.0–23.0	4.0–6.0
Infantile hypertrophic pyloric stenosis (values on day 3) (n=9)	8.3 (± 0.17)	1.8 (± 0.09)
Range	7.0–10.0	1.0–2.0

*The nine infants later developing infantile hypertrophic pyloric stenosis were excluded from the normal range.

determine the significance of the change in pyloric dimensions from birth to presentation with infantile hypertrophic pyloric stenosis.

Results

A total of 1400 consecutive newborn infants had pyloric dimensions measured at routine postnatal examination. Nine of these infants subsequently presented to this hospital with infantile hypertrophic pyloric stenosis, which in all cases was confirmed by surgery. The pyloric dimensions recorded are summarised in the table.

The initial pyloric dimensions of the nine infants who presented with infantile hypertrophic pyloric stenosis were no different from the other 1391 healthy newborns. However, there was a highly significant increase ($p < 0.001$) in the size of the

pylorus by the time infantile hypertrophic pyloric stenosis was diagnosed.

Discussion

This study shows that pyloric muscle hypertrophy is not present in the early newborn period of infants who later develop infantile hypertrophic pyloric stenosis. It excludes the possibility of a congenital preformed 'soft' hypertrophy of pyloric muscle without constricting effects suggested by Wallgren. The incidence of infantile hypertrophic pyloric stenosis in this study group was 6.4/1000 live births and is in keeping with the previously reported high rising incidence in this area.⁵ We conclude that infantile hypertrophic pyloric stenosis should no longer be referred to as 'congenital' pyloric stenosis, although the true aetiology of this acquired condition remains to be elucidated.

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Accepted 5 August 1988

Plasma aldosterone and renin activity

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SUMMARY Eight infants had paired measurements of plasma aldosterone and plasma renin activity while being treated for congestive heart failure. There is parallelism with aldosterone and renin activity in the presence of a hyperactive renin-angiotensin-aldosterone system. Six patients had plasma renin activity and plasma aldosterone measured after commencing captopril and we have shown biochemical blockade of the renin-angiotensin-aldosterone system.

We have recently reported raised plasma renin activity in infants being treated for congestive heart failure due to congenital heart disease.¹ In that study we were unable to report the results of plasma aldosterone because concentrations fell outside the range of the assay in most instances. We have performed an additional study to determine whether parallelism between plasma renin activity and plasma aldosterone, which has been described in normal infants,² continues when the renin-angiotensin-aldosterone system is hyperactive, and whether this

system can be blocked by using an angiotensin converting enzyme inhibitor.

Patients and methods

Eight patients had coincidental measurement of plasma renin activity and plasma aldosterone. Five of these patients had sampling on three separate occasions over a period of four weeks, one patient on two occasions, and two patients on a single occasion. The eight infants, at the time of the first sampling, had an age range of 19 to 111 days and their mean (SD) weight was 3330 (400) g. All the patients had congestive heart failure due to congenital heart disease with left to right shunts and were receiving diuretics.

Other criteria for inclusion in the study, the sampling conditions, and the method of estimation of plasma renin activity were as previously reported.¹

Plasma aldosterone was measured by direct radioimmunoassay using a 'Coat-a-Count' kit with ¹²⁵I aldosterone label (Diagnostic Products Ltd). The antialdosterone antibody is coated on the walls of the assay tubes thus achieving solid phase separation of the bound and unbound fractions. Both intra-assay and interassay precision is less than 10% throughout the range of the assay. Samples with expected aldosterone concentrations greater than the working range of the assay were diluted in zero calibrator. This procedure had previously been validated by serial dilution of a high level sample that displayed parallelism with the dose response curve. One ml of whole blood was required for each of the assays.

Six patients, including two patients in the first study, were given captopril for treatment of their heart failure. The standard regimen in use at the Royal Liverpool Children's Hospital for initiating treatment was followed. Oral captopril was introduced at 0.25 mg/kg/dose three times daily. Increments of 0.25 mg/kg were made each day up to a maximum of 1 mg/kg/dose. Plasma renin activity and plasma aldosterone were measured after at least four days on the maximum dose. The same conditions were employed for blood sampling but in addition the sample was timed at two hours after the dose of captopril.

Results

A total of 14 paired plasma renin activity and plasma aldosterone values were obtained in the first part of the study. Three patients from the first batch of assays (6, 7, and 8) had only one set of paired values within the standards range (table). The results are

plotted on the figure (triangles). The correlation coefficient by Spearman's rank test was 0.78 ($p < 0.005$).

The patients in whom captopril had been administered showed an abolition of the relationship be-

Table Sample times, plasma renin activity, and plasma aldosterone concentration, in eight patients

Patient No	Sample time (days)	Plasma renin activity (ng angiotensin I/ml/hour)	Plasma aldosterone (pmol/l)
1	Zero	183	27 920
	6	81	21 070
	28	9	3630
2	Zero	88	29 910
	7	20	1690
	28	19	3010
3	Zero	69	12 810
	7	49	10 160
	28	25	2310
4	Zero	95	23 360
5	Zero	22	7770
6*	Zero	71	Outside standards range
	8	69	Outside standards range
	28	25	3026
7*	Zero	80	Outside standards range
	7	38	Outside standards range
	28	12	2359
8*	Zero	89	Outside standards range
	4	34	2388

Outside standards range: >3300 pmol/l; * = patients whose plasma aldosterone assays were performed without dilution.

Figure Plasma renin activity and plasma aldosterone in infants with congestive heart disease. Diuretic treatment without captopril ($\blacktriangle$) and diuretic treatment with captopril ($\bullet$).

tween plasma renin activity and plasma aldosterone (figure, circles). There was no significant difference between mean (SD) plasma potassium concentrations before and after instituting captopril treatment (4.27(0.39) mmol/l and 4.33(0.51) mmol/l, respectively).

Discussion

This study has shown that there is parallelism with plasma renin activity and plasma aldosterone when there is hyperactivity of the renin-angiotensin-aldosterone system in infants in heart failure. Although five plasma aldosterone samples were above the range of the assay, these corresponded with high plasma renin activity values and tend to support our conclusion.

Previous studies of normal infants have shown good correlation with plasma renin activity and plasma aldosterone in the normal range.² In older children of widely varying ages with salt depleting conditions there is a rise of both plasma renin activity and plasma aldosterone.³ By contrast exchange transfusion with acute blood volume loss results in increased plasma renin activity but no parallel rise in plasma aldosterone.⁴ In our study the patients were in a stable state compared with those undergoing exchange transfusion.

The effect of captopril is to block the conversion of angiotensin I to angiotensin II. The latter is one stimulus to the production of aldosterone and our study would suggest it is the major stimulus in infants in heart failure. We have shown that captopril at a dose of 1 mg/kg can effectively block this hormone system. Further studies of infants being treated for heart failure are required to determine the minimum dose required for effective blockade.

We thank Mrs J Start and Mrs J Radcliffe for technical help with the assay procedures and the cardiology department at Royal Liverpool Children's Hospital, Myrtle Street for providing the patients for study.

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Accepted 1 September 1988

Dysphagia due to oesophageal web

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SUMMARY An 8 year old boy developed a recurrent sensation of food sticking in his throat during meal times. Barium swallow examination showed an anterior oesophageal web at the level of C5. Symptoms disappeared after pharyngoscopy and dilatation of the web.

Case report

An 8 year old boy of mixed Asian and white parentage attended a school medical examination with a six month history of food sticking in his throat. He avoided eating meat, fish, eggs, and green vegetables, preferring soft foods that could be chewed easily. Frequent choking at mealtimes had lead to a decrease in appetite; liquids were tolerated

without difficulty. Systematic enquiry showed no additional symptoms and the medical history was unremarkable. His parents and siblings were well and there was no family history of illness of any sort. Physical examination was entirely normal.

INVESTIGATIONS

Haematological investigation showed a normal haemoglobin (128 g/l), but a moderately reduced mean corpuscular volume (75.4 fl, reference range 80-96 fl) indicated probable mild iron deficiency. More detailed investigations were not undertaken at this time. A one month course of ferrous sulphate (120 mg three times daily) was given, and as dysphagia persisted, he was referred to hospital for further assessment.

Dysphagia had been present for 12 months at the time of hospital review. Clinical examination