These 2 cases provide additional examples of HUS occurring in sibs after a long interval. The family resided in New York City, which is not considered as an endemic area for HUS, and the clinical course of the disease in both infants was almost identical, culminating in early death.

The aetiology of HUS may indeed be multiple, and the genetic basis for a significant proportion of cases does not seem very likely. However, the familial cases with longer intervals between onset, as presented here, cannot simply be discarded as fortuitous instances of environmental causative factors. It may be that the genetic predisposition for HUS is operative in such families, implicating an autosomal recessive trait.

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Aetiology of hydrops fetalis

Sir,
Drs. Radford, Izukawa, and Rowe (1976) in their article on congenital paroxysmal atrial tachycardia happen also to contribute a great deal to the literature on hydrops fetalis. Several reviews on the conditions associated with hydrops fetalis point to the varying aetiologies of the disease (Becker, 1975; Driscoll, 1966). Most of these diseases are associated with congestive heart failure, severe anaemia (usually haemolytic), and/or cellular damage to

the liver or placenta. Unfortunately, no one process can explain all the cases. In erythroblastosis fetalis, hydrops fetalis is most related to hypoproteinaemia (Phibbs *et al.*, 1974) or low oncotic pressure (Baum and Harris, 1972) rather than universal anaemia. Whether hypoproteinaemia is due to liver disease or to congestive heart failure is uncertain; however, cases of 'pure' output failure such as those reported by Radford *et al.* offer an ideal opportunity to test the hypothesis that hydrops results from hypoproteinaemia secondary to heart failure and not vice versa. Do they have the necessary protein levels on their patients?

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Dr. Radford and colleagues comment:

Regarding the interesting letter from Dr. K. L. Harkavy, we do have serum protein data on 3 of our 12 patients (Cases 8, 11, 12). Initial blood specimens were taken on the first day of life in all 3 patients and repeated later in 2. Electrophoretic pattern analyses were also done. The relevant results are in the Table.

Although the data are limited, there was definite hypoalbuminaemia in Cases 8 and 11. This would fit with the hypothesis raised by Dr. Harkavy, i.e. that hypoproteinaemia was secondary to heart failure and led to hydrops.

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Patient age	Case 8 1 d	5 d	Case 11 1 d	18 d	Case 12 1 d	Age related normal range
Total protein g/100 ml	4.7	4.6	4.1	5.5	5.5	4.6-7.0
Albumin g/100 ml	3.1	2.6	2.6	3.6	—	3.2-4.8
α_1 "	0.3	0.4	0.2	—	—	0.1-0.3
α_2 "	0.5	0.4	0.3	—	—	0.2-0.6
β "	0.5	0.7	0.4	—	—	0.3-0.6
γ "	0.4	0.5	0.3	—	—	0.6-1.2