baseline were available for follow-up at 18 months; and not all children whose 18 month data were collected, had data collected at baseline. Thus, children whose data were collected at 18 months of age (presented in table 2 of the article) [1], comprised an unknown proportion of those who were present at baseline, plus an unknown proportion of those who were not present at baseline.

(*v*) The authors [1] found that children in the comparison group fared worse than children in the intervention group. Notwithstanding the methodological limitations compromising validity, they assumed this to mean that under natural circumstances, nutritional status of children would decline, and the intervention partially mitigated this. But they have not provided any data from any study, anywhere in the world, that can support this view. This suggests that the explanation offered for the unusual finding in this study [1] is erroneous. This view is strengthened by the other points mentioned in the commentary [2].

(*vi*) Figure 3 in the study [1] shows that only about 12% of the loans were for 'food and supplies' and the total amounted to less than Rs. 10,000 across the *tola*s. In the face of food insecurity (*i.e.,* starvation), one would expect people to take loans to purchase food (to tide over the immediate scarcity) rather than invest in capital for agriculture or medical supplies (that have no short-term impact on starvation).

(*vii*) The table of baseline characteristics in the study [1] showed statistically significant differences in three anthropometric parameters between the intervention and comparison groups. Two of these were better in the intervention group viz HAZ (*Z* score -2.00 *vs* -2.14) and proportion with MUAC <12.5 cm (13% *vs* 16%). In contrast, the proportion with wasting was higher in the intervention group (20% *vs* 15%). These data suggest that children in the intervention group had (statistically) better HAZ. Since height *Z* score is an indicator of longer-term nutritional status and does not decline immediately in acute malnutrition (unlike wasting or MUAC), it suggests that children in the interventional group had a statistically superior indicator of longer-term nutritional status (at baseline).

(*viii*) Since only one-third of the mothers in the intervention group actually received the intervention, it is difficult to believe that the comparable outcomes in offspring of those who did (and did not) receive the intervention was based on a spillover effect. The authors have not demonstrated how/why financial empowerment of a limited number of women in the community could create a spillover effect to other mothers and families.

In summary, methodological limitations compromise the validity of the trial [1], and the authors' recent comments do not change the viewpoint that this trial is insufficient to support further similar studies or launch a community-wide intervention with the specific microfinance scheme described (for the purpose of improving nutritional status of children). Whether the scheme could have any other positive social or cultural or health-related impact, is outside the scope of discussion.

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Acute Peritoneal Dialysis in Premature Infants: Few Concerns

We read with great interest the recent article by Okan, *et al.* [1] published in *Indian Pediatrics* which concluded that peritoneal dialysis (PD) is technically feasible in very low birthweight (VLBW) and extremely low birthweight (ELBW) neonates despite a high mortality rate in the studied population (81%). We also agree that peritoneal dialysis in neonates, and particularly in preterm neonates, is challenging and is still evolving with only few anecdotal case report and case series till date indicating its feasibility in preterm neonates. Further, due to the physiological compromise (small size, poor hemodynamic stability and tendency of coagulopathy), overall prognosis in preterm neonates undergoing peritoneal dialysis is grimmer as compared to their term counterparts as well as older children. This study was need based and addressed a very important and clinically relevant issue. However, we have few concerns related to the article which we would like to get the clarification from the author.

1. In Table I of the article, we were intrigued to note that patent ductus arteriosus (PDA) led to acute kidney injury on day 1, and that too requiring PD [1]. We would like to know the exact clinical/ laboratory criteria for doing peritoneal dialysis in that baby.

2. Many babies (50% of the study population) had undergone PD due to necrotizing enterocolitis (NEC) as one of the underlying causes (***Table* I**) [1]. The result section also mentions that 5 (23.8%) of babies had perforated NEC (stage IIIb) [1]. As the presence of NEC, particularly perforated NEC is a contraindication to do PD [2], why was it carried out in these babies? This is important, as approximately 80% of the babies who had undergone PD with NEC as underlying cause, ultimately died [1].

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AUTHORS' REPLY

We would like to thank the authors for their interest in our article [1]. The comprehensive criticism of methodological and pathophysiological issues presented in their letters provides an illuminating framework for our study. We would like to offer some clarifications regarding the points they have raised.

Peritoneal dialysis was indicated according to the Neonatal RIFLE Criteria for acute kidney injury (AKI) [2] *i.e.,* oliguria/anuria (urine output of <0.7 mL/ kg/h for 24 h or anuria for 12 h), failure of conservative treatment (furosemide or water restriction in cases without hypovolemia), signs of uremia (impaired cardiac and respiratory functions, or seizures), refractory hyperkalemia, metabolic acidosis or fluid overload. In our study, the patient who was started peritoneal dialysis (PD) at the earliest time had a gestational age of 27 weeks and weighed 1060 g, with a hemodynamically significant patent ductus arteriosus (PDA) and history of anhydramnios. PD was initiated at the end of the first day of life for anuria, failure of conservative treatment, signs of uremia and was performed for four days. Urine output was obtained on the third day of life. The patient responded successfully to PD and survived thereafter. The literature on AKI in premature infants with a diagnosis of necrotizing enterocolitis (NEC) is limited. The incidence of AKI in NEC is very high and the mortality is two-fold higher than of infants with no AKI [3]. Downard, *et al*. [4] demonstrated in rat pups with NEC that the utility of direct peritoneal resuscitation (DPR) increases the intestinal blood flow significantly and speculated DPR may be a novel strategy to improve intestinal blood flow in NEC. Another study [5] reported that topical 1.5% dextrose solution enhanced significantly the blood flow in the terminal ileum to the same degree as 2.5% dextrose solution in Sprague-Dawley rats. Direct peritoneal resuscitation as a treatment modality is applicable in any disorder with decreased intestinal blood flow. The maintenance of intestinal blood flow takes control of the multi-system inflammatory response and decreases the overall risk of multiple organ dysfunction and death [5]. Peritoneal dialysis is also an alternative and rescue method to treat infants with NEC complicated with intestinal perforation. Peritoneal dialysis can be used as a type of peritoneal lavage in NEC for the removal of inflammatory cytokines, toxins, and may help in remodeling and healing of intestine [6]. We reiterate that initiation of early PD in sick extremely low birthweight infants with NEC and AKI may save lives [7].

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