REPLY:

With great interest we read the letter by Drs Adamsbaum and Rey-Salmon concerning our article “Subdural Hygromas in Abusive Head Trauma: Pathogenesis, Diagnosis, and Forensic Implications.”¹ We are very grateful for their interest in our work.

Because during neuroimaging we frequently observe the concomitant occurrence of hyper- and hypodense subdural collections (mixed-density pattern), in both the same location and at least 2 different locations, we were interested in the possible pathophysiologic mechanisms that underlie the formation of the hypodense component (ie, subdural hygromas [SDHys]). The currently available literature reveals the presence of the 2 major hypotheses that we outlined in our review article (delayed and rapid formation of SDHys).¹ Of course, we are aware that the pathophysiology of SDHy formation is still an ongoing research process, and it seems very likely that multiple mechanisms exist and coexist.

Dating the incident by estimating the age or stage of a subdural collection is an even more controversial issue and has a high potential for confusion, especially the question of how many shaking events might have occurred. Repeated shaking, of course, may definitely cause corresponding injuries such as seen in the “age-different” pattern described by Adamsbaum et al.^{2,3}

In regard to the case example shown in Fig 3 of our article¹ however, we can exclude, due to the child’s medical history, a previous subdural hematoma (SDH) being present 4 weeks earlier. The infant had been clinically examined at relatively short intervals since birth, and the clinical records did not reveal any abnormalities 1 month before the MR imaging, in either cranial sonography or clinically. Membranes organizing the subdural collection and supporting the assumption of an earlier formation of the SDHy⁴ were not discernable in the MR imaging either. We totally agree with the opinion that in the context of violent acceleration-deceleration, the occurrence of an acute SDHy without any hemorrhagic component or sediment could be considered unusual. We hypothesize that in addition to a tear in the arachnoid membrane, a hemorrhagic component might not be missing but is possibly only a minor part of the subdural collection, suggesting only a minor vascular injury in the frontoparietal region and a rapid influx of a significant amount of CSF and/or CSF-like liquid. This could explain not only the sudden increase in head circumference from the 50th to the 97th percentile but also the slightly higher signal intensity of the SDHy in Fig 3 compared with the “pure” CSF within the subarachnoid space. The formation of an acute SDHy within 20 hours in a different location than the acute SDH has also been directly observed during serial neuroimaging.⁵ In general, subdural hypodensities (ie, SDHys) have been shown to appear much sooner than the 1–4 weeks accepted previously.^{5–7}

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These data exhort us to be careful with the age estimations of subdural collections, not only with regard to mixed densities in a single location (eg, due to serum separation and sedimentation of erythrocytes^{4,7}) but also in different locations. Accordingly, we think that the subdural pathologies of the infant presented in Fig 3 do not necessarily indicate an additional abusive head trauma (AHT) that occurred 4 weeks earlier. Instead, the clinical findings, serial neuroimaging, and the available literature suggest more recent trauma including, of course, the possibility of repeated shaking within a short interval. Hence, we still believe that the SDHy in this case “can be regarded as a result of acute injury.”¹

We definitely agree with Drs Adamsbaum and Rey-Salmon that recognizing early clinical signs and symptoms of AHT is crucially important. In this context, we would also like to stress the vital importance of close and efficient cooperation among all medical disciplines involved in AHT cases (ie, pediatrics, ophthalmology, forensic medicine, and radiology in particular). Only a fast and reliable diagnosis facilitates legal certainty and appropriate therapy for infants with AHT.

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