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Tools 1University of Liverpool School of Medicine, Liverpool, UK2Department of Women's and Children's Health, University of Liverpool, Liverpool, UKFind articles by Charlotte EM Rugg-Gunn3Alder Hey Children’s Hospital, Liverpool, UKFind articles by Mark Deakin2Department of Women's and Children's Health, University of Liverpool, Liverpool, UK4NIHR Alder Hey Clinical Research Facility, Liverpool, UKFind articles by Daniel B HawcuttReceived 2021 Mar 2; Accepted 2021 May 21. ketoacidosis (DKA) is a life-threatening complication of type 1 diabetes mellitus. Careful and timely intervention is required to optimise glycaemic control and reduce the risk of mortality and devastating complications. Of these, cerebral oedema is the leading cause of death, with a mortality rate of approximately 25%. This article highlights the recent updates to UK fluid therapy guidelines for DKA and provides clinical context for the benefit of paediatricians and junior doctors in light of this new guidance.Keywords: resuscitation, diabetesKey messagesUpdates to two UK guidelines in 2020 has improved concordance, with 0.9% sodium chloride and the Holliday-Segar formula used as a gold standard to calculate fluid maintenance requirement.Minor differences remain, such as the maximum weight for maintenance and the stratification of diabetic ketoacidosis or dehydration severity.Careful monitoring and adherence to these national guidelines is recommended and will hopefully contribute to a reduction in deaths secondary to cerebral oedema (CO).Further research is required to evaluate the efficacy of these new guidelines, and better understand the pathophysiology and risk factors for developing CO.Diabetic ketoacidosis (DKA) is a potentially life-threatening metabolic complication of type 1 diabetes mellitus. Despite interventions to reduce the incidence of DKA, the National Paediatric Diabetes Audit for 2018/2019 showed that overall 20.9% of all newly diagnosed patients in the United Kingdom (UK) presented in DKA, although there is considerable regional variability.1DKA is characterised by uncontrolled hyperglycaemia, ketosis and subsequent metabolic acidosis. As highlighted in figure 1, the clinical picture can develop insidiously due to a variable constellation of non-specific systemic signs and symptoms, including polyuria, polydipsia, weight loss, fatigue, vomiting and abdominal pain, ultimately leading to confusion, coma and death, if untreated.2 3 Due to the ambiguity of the presenting clinical features in the early stages, delays in diagnosis are common.4Globally, the International Society for Paediatric and Adolescent Diabetes (ISPAD) guidelines are widely used for management of paediatric DKA.5 However, in the UK, it is guided by the integrated care pathways of either the British Society for Paediatric Endocrinology and Diabetes (BSPED) or the National Institute for Health and Care Excellence (NICE). The BSPED guideline evolved from the NICE guideline in 2009 due to concerns over limited evidence that was used for its development. While there were a number of dual-serving board members, the input and additional evidence by the BSPED special interest group in DKA was thought to increase its safety.6 Both guidelines cover the diagnosis and management of type one and two diabetes in children and young people (CYP) aged under 18 years.7 Areas of particular benefit within a secondary care setting are centred on the requirements for fluid therapy, given the importance of resuscitation in preventing complications. Due to the difference in perceived risk of cerebral oedema (CO) due to rapid fluid administration, there was discordance between the NICE and BSPED 2015 guidelines.Specifically, the NICE 2015 guidelines were more conservative with fluid administration, in order to avoid rapid changes in osmolality. The BSPED guidelines permitted a more generous fluid allowance, and this raised concerns about precipitating CO. The conflicting advice generated challenges in the emergency setting, with arbitrary site-specific preference rather than evidence-based medicine taking precedence. In addition to generating uncertainty of treatment pathways increasing the risk of clinical error, the regional variability hampered service evaluation and audit of clinical practice.Unfortunately, there is little data to establish differences in their efficacy, especially surrounding the risk of CO in the paediatric population. However, inadequate resuscitation is likely to increase the risk of brain injury and thus must be avoided. Further research on a national scale is required to assess this.This article aims to highlight the considerable alterations made to the two main UK guidelines. Alongside these updates, this article will provide clinical context to paediatricians and junior doctors who are likely to treat these vulnerable groups.In January 2020, the BSPED published new guidance for the management of children with DKA.6 This integrated care pathway migrated further from the model of restrictive fluid replacement towards an even more flexible approach for resuscitative and maintenance fluids. The rationale for this was based on new evidence from the Pediatric Emergency Care Applied Research Network (PECARN) DKA Fluid trial, which suggested that there was no significant difference in outcomes between rapid and slower fluid administration.8 Additionally, it showed that the initial conscious level was closely related to pH and weakly to age, but not to blood glucose or plasma sodium level.8 9 While this appears to demonstrate that cerebral function is related to the severity of acidosis even in the absence of drivers of CO, it does not negate previous evidence that fluid shift may also contribute towards CO.10 11Furthermore, there are clear risks associated with inadequate fluid replacement. A hypovolaemic state will result in systemic hypotension and ultimately, cerebral hypoperfusion which, particularly in the context of acidosis, will increase the risk of a brain injury.6In 2008, following three deaths from CO, the South Thames Retrieval Service introduced a ‘restrictive fluid’ DKA guideline in the region. Subsequently, no deaths have been reported, but this is insufficient evidence on which to make definitive recommendations or guidelines.12More recently, in December 2020, NICE released updated guidance on the management of DKA in CYP, which was broadly concordant with BSPED, particularly with respect to more liberal fluid restriction. This was prompted by new evidence identified by NICE’s surveillance team in a review of 12 studies set to determine optimal fluid therapy in CYP with DKA. The fluid protocols followed in the PECARN study were fundamental to these updates,8 as ‘neither the rate of administration nor the sodium chloride content of intravenous fluids significantly influenced neurologic outcomes in children with DKA’.8 Therefore, restrictions to the fluid administration, which were recommended in the 2015 NICE guidance, ‘were not necessarily required’.13 Additionally, as with the BSPED and ISPAD guidelines, the updated NICE guidance was amended to include the Holliday-Segar formula, used to calculate fluid maintenance.6 7 Updates to both the BSPED and NICE guidelines in 2020 have a more permissive fluid rate than in the previous DKA fluid therapy guidelines.Both BSPED and NICE recommend the following fluid maintenance requirements, using 0.9% sodium chloride without added glucose, and the Holliday-Segar formula:100 mL/kg for the first 10 kg (0–10 kg body weight).50 mL/kg for the second 10 kg (10–20 kg body weight).20 mL/kg for each subsequent kilogramme (>20 kg body weight).When calculating the total fluid replacement, subtract any initial bolus volumes from the total fluid deficit (unless the child or young person is in shock).For insulin and electrolyte replacement requirements, both recommend6 7:The addition of 40 mmol/L potassium chloride to all fluids (apart from the initial intravenous bolus) unless the patient is anuric or hyperkalaemic. Include this before starting the insulin infusion if hypokalaemia is observed at presentation.0.9% sodium chloride should be used without added glucose unless the plasma glucose is

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