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Cognitive, behavioural and psychological barriers to the prevention of severe hypoglycaemia: A qualitative study of adults with type 1 diabetes

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Abstract
Objectives: Severe hypoglycaemia affects approximately one in three people with type 1 diabetes and is the most serious side effect of insulin therapy. Our aim was to explore individualistic drivers of severe hypoglycaemia events.
Methods: In-depth semi-structured interviews were conducted with a purposive sample of 17 adults with type 1 diabetes and a history of recurrent severe hypoglycaemia, to elicit experiences of hypoglycaemia (symptoms/awareness, progression from mild to severe and strategies for prevention/treatment). Interviews were analysed using an adapted grounded theory approach.
Results: Three main themes emerged: hypoglycaemia-induced cognitive impairment, behavioural factors and psychological factors. Despite experiencing early hypoglycaemic symptoms, individuals often delayed intervention due to impaired/distracted attention, inaccurate risk assessment, embarrassment, worry about rebound hyperglycaemia or unavailability of preferred glucose source. Delay coupled with use of a slow-acting glucose source compromised prevention of severe hypoglycaemia.
Conclusion: Our qualitative data highlight the multifaceted, idiosyncratic nature of severe hypoglycaemia and confirm that individuals with a history of recurrent severe hypoglycaemia may have specific thought and behaviour risk profiles. Individualised prevention plans are required, emphasising both the need to attend actively to mild hypoglycaemic symptoms and to intervene promptly with an appropriate, patient-preferred glucose source to prevent progression to severe hypoglycaemia.

Keywords
Severe hypoglycaemia, type 1 diabetes, prevention, experience, qualitative

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Introduction
Severe hypoglycaemia (SH) affects approximately 30% of individuals with established type 1 diabetes (T1DM) each year1,2 and remains one of the most serious and feared complications of insulin therapy. It is defined as ‘an event requiring assistance of another person to actively administer carbohydrate, glucagon or other resuscitative actions’3 and
can result in collapse without warning, fits or even sudden death. SH is often a limiting factor to attaining optimal diabetes outcomes, as blood glucose targets may be relaxed in order to avoid hypoglycaemic episodes. In addition to safety concerns (for self and others), people with T1DM often fear SH, as it can involve unpleasant symptoms, mood changes, embarrassment, loss of personal control and independence, resulting in loss of ability to drive safely and fulfill family, work and social responsibilities.

The incidence of SH varies in research studies between 0.2 and 5 events per person per year depending on individual characteristics. The distribution of events is markedly skewed, with a few individuals experiencing a disproportionately large number of severe events. Established risk factors for SH include age, duration of diabetes, tight glycemic control, impaired awareness of hypoglycaemia (IAH) and previous severe events. IAH is estimated to affect 20%–25% of unselected clinic populations. It is characterised by diminished and delayed warning symptoms of impending hypoglycaemia and is associated with a sixfold increased risk of SH when compared to people without IAH. Of those with IAH, 60% experience at least one episode of SH every year, with an average event frequency of once every 10 weeks and some people having many more events.

Hypoglycaemia is often referred to in ways that suggest it is a dichotomous variable (mild or severe), but in reality, individuals experience hypoglycaemia along a continuum. Mild hypoglycaemia, which can be readily self-treated, will progress to SH if left untreated. This is particularly true in those with long-standing T1DM due to the absence of endogenous insulin secretion (which would be reduced during hypoglycaemia) and glucagon secretion (the primary glucose-raising hormone). Together with progressive failure of counter-regulatory hormone responses, particularly sympathoadrenal activation, which also generates symptoms such as sweating and tremor, this leads to marked vulnerability in those with a duration of diabetes over 15 years. As glucose remains low or falls further, cognitive and functional impairment deepen, until SH ensues and the assistance of others becomes essential to administer glucose and facilitate recovery. Underlying drivers and potential strategies for prevention of SH have been reviewed in more detail elsewhere.

Quantitative and experimental studies have investigated the physiological, pathological and (to a lesser extent) psychological processes underlying hypoglycaemia, resulting in clinical emphasis on absolute avoidance of biochemically low glucose levels with the goal of restoring awareness and preventing severe events. Experimental evidence derives largely from ‘clamp’ studies, in which hypoglycaemia is induced artificially in a gradual or step-wise manner through manipulation of exogenous insulin and dextrose administration. However, hypoglycaemia, as it is experienced in everyday life, may differ in terms of onset, symptomatology and progression.

A biopsychobehavioural model of SH has been developed, which proposes that people with a history of SH probably have very specific profiles of risk factors. Yet, there remains only limited understanding of the reasons why people who retain some awareness of warning symptoms are not always able to prevent SH. Recently, qualitative studies have been conducted in this field, but they have focused either on the experiences of people with IAH, or their family members, or on treatment preferences for mild hypoglycaemia among adults with T1DM who have attended structured education. Given the array of established demographic, clinical and idiosyncratic behavioural risk factors involved, we considered that a qualitative study of adults with T1DM who have experienced recurrent SH would offer valuable insights, as drivers are highly personal and idiosyncratic. While each may be relevant to a subset of people with T1DM, the first step in understanding these psychological mechanisms is to describe them at the individual level.

Our aim was to conduct a qualitative study of adults with T1DM, exploring individual experiences of SH occurring in daily life, to understand barriers to the prevention of SH.

Methods

Study design

Qualitative study involving in-depth interviews.

Setting

Two multidisciplinary specialist diabetes clinics (Newcastle and Manchester, the United Kingdom).

Sample and recruitment

Purposive sampling was used to recruit 17 adults with established T1DM and a history of recurrent SH. In each participating centre, a medical professional (S.A.L. or M.K.R.) approached potential participants to ascertain interest in the study and obtain informed consent. Assurance was given that consent or refusal to participate would not affect the quality of diabetes care or eligibility for any future clinical studies and that all information provided would be confidential to the study team. A total of 20 adults with T1DM were approached, and all were initially willing to participate, though three declined (all from Newcastle) for logistical reasons.

Ethics

Ethical approval was provided by the National Research Ethics Service.

Interviews

Interviews were arranged at a time and location convenient to the participant; 12 of the 17 (71%) were conducted in a
private room in the hospital, and 5 (29%) were conducted by telephone. Mean interview duration was 52 min (range: 34–69 min). The interviews were conducted by health psychologists (J.S. and S.M.B.), with no prior relationship to the participants. Participants’ demographic data and most recent HbA1c (average blood glucose) were obtained from their medical records.

Exploratory semi-structured interview techniques were used to investigate participants’ experiences of hypoglycaemia through open and non-directive discussions about symptom awareness and experience, severity and progression to SH. Each interview was guided by an interview schedule (Box 1), informed by the literature and professional experience, which was used flexibly to allow inclusion of new issues raised by participants. Interviews were conducted in sets of three or four, and the schedule was amended between sets to include new issues arising.

Analysis

All interviews were digitally audio-recorded and transcribed verbatim. We used an adapted grounded theory approach to analysis,21,22 to explore discrete and overlapping themes in participants’ accounts of their experiences. Transcripts were read individually by health psychologists (S.M.B. and H.S.) and discussed at length (S.M.B., H.S. and J.S.) in an iterative process to allow identification and coding of emergent themes. Memos were used to cross-check and clarify codings between the three psychologists (S.M.B., H.S. and J.S.) to develop a cohesive conceptual description. All quantitative data are presented as mean ± standard deviation.

Results

The sample included 17 participants, nine (53%) of whom were women. Participants were aged 46 ± 11 years and had lived with T1DM for 26 ± 14 years. All had previously attended multidisciplinary structured T1DM education, which included specific advice to treat all hypoglycaemic episodes primarily with fast-acting refined carbohydrate. A total of 13 participants (76%) were currently using insulin pump therapy and reported experiencing fewer hypoglycaemic episodes since migration from multiple daily injections (MDI) to pump therapy. In the previous year, 15 of the 17 participants had experienced at least one episode of SH (with range between 1 and more than 50 events); one had experienced episodes of nocturnal hypoglycaemia that were disabling but from which recovery occurred without the assistance of others, and one had not had an episode of SH since commencing pump therapy (despite many previous episodes). Hypoglycaemia awareness was not formally recorded at the time of the interview, but a broad spectrum was evidenced by previous assessment undertaken as part of

Box 1. Discussion guide for interviews.

1. Experience of recurrent severe hypoglycaemia:

(a) So, let us start then by talking about your experience of hypos. What, for you, is the difference between a ‘mild’ hypo and a ‘severe’ hypo?
(b) Doctors talk about severe hypoglycaemia being those that mean you are unable to treat yourself and need someone else’s help. In the past month, how often have you had that type of hypo? (What about in past 6 months? Can you remember that accurately?)
(c) Are you always aware of going hypoglycaemic? How often do you check your blood glucose (BG) levels?
(d) What happens when you have a severe hypoglycaemia? Can you talk me through a ‘typical’ episode – before, during and after? (What is that like? How does it make you feel?)
(e) Which symptoms do you experience? (Which are most frequent, bothersome, help in identifying or treating hypo early?)
(f) How do you remember your recent severe hypoglycaemia?
(g) Do you live alone/with someone/do you have a pet? How does that impact your experience?

2. Drivers of recurrent severe hypoglycaemia:

(a) How do you feel about … managing your diabetes? … controlling your blood sugar levels?
(b) At what sort of glucose levels do you feel happiest?
   • How do you feel when they are lower/higher than that? Do higher or lower levels make you feel anxious/unhappy and so on; why is that? Was it something that your health professionals said (e.g. at diagnosis) or does something else influence it? Do you avoid high glucose ‘at all costs’?
(c) Why do you think your blood sugars go low?
   • (Does the person make a link between what he or she does and going hypo? Does the person purposefully keep BG low (bordering on SH) or does BG go low as part of erratic management/lifestyle etc?)
   • (What controls their hypoglycaemia? Chance, medics, self? Simple and basic factors (e.g. less than adequate knowledge about diet/exercise, lifestyle changes))
(d) If they purposefully run BG low, Why?
   • (Fear of high BGs/complications? Lack of fear of hypoglycaemia? Anxieties about weight (not snacking, etc.)? Depression (people may prefer being out of control than responsible for self)? Risky behaviours, chaotic control (emotive issues)?)

Is there anything else you would like to add?
routine clinical care in both participating centres (including individuals with Gold scores\(^1\) between 2 and 6). Participants’ mean HbA\(_1c\) was 61 ± 1 mmol/mol; range 45–76 mmol/mol (7.7% ± 0.8%; range 6.3%–9.1%).

Participants gave rich and detailed accounts of their individual experiences of hypoglycaemia and of the detrimental impact of SH on their safety and physical comfort, well-being and quality of life. The extent of potential distress is well illustrated by the words of one: ‘I could be having forty winks but the trouble is … those forty winks could turn into a coffin’ (M13).

The participants’ own words in Boxes 2–5 illustrate common themes. For reasons of brevity, succinct quotations have been selected, and no attempt has been made to give equal representation to the experiences of each participant, although all accounts have been given equal attention in our analyses.

### Symptoms

Participants described hypoglycaemia mostly in terms of progression or deterioration, in which symptoms occurring early during an episode differed from later symptoms in terms of type and/or severity (Box 2). It was notable that participants experienced impaired concentration or attention as an early symptom, with or without additional symptoms such as weakness, anxiety, shaking, visual disturbance, palpitations or difficulty breathing. Symptoms reported as manifesting early in an episode spanned all recognised categories: autonomic (e.g. palpitations, sweating, trembling), neuroglycopenic (e.g. weakness, confusion) and general malaise (e.g. headache, nausea). Later in the progression of an episode, autonomic and neuroglycopenic symptoms were also widely reported, but general malaise was not.

**Box 2.** Individuals’ experiences of symptoms, early and late, in the progression of hypoglycaemia.

| Early symptoms | ‘I might start to feel my brain’s not quite working properly … It’s the mental side of things when I’m slowing down and I notice that things aren’t quite right’. (F5) |
|               | ‘I find it difficult to breathe because my diaphragm just didn’t seem to work very well and I can’t fill my lungs with air’. (M4) |
|               | ‘I get coloured blotches in front of my eyes’. (M3) |
|               | ‘I get confused and things don’t make sense. I get hot, dizzy sweaty and I sometimes get the shakes … My vision gets slightly blurred’. (F2) |
|               | ‘The tiredness hits … crying … basically, not quite there’. (F1) |

| Late symptoms | ‘It’s just like a feeling of weakness … I tend to be a bit silly’. (F6) |
|              | ‘I would just feel kind of slightly shaky’. (F7) |
|              | ‘Sweaty palms and I’ll feel a bit shaky … I get a bit snappy’. (M8) |
|              | ‘Feeling of anxiety … light palpitation … concentration wanders …’. (M9) |
|              | ‘Lack concentration, grate my teeth’. (F10) |
|              | ‘My gums were ideal, because as soon as they started tingling I knew straightaway’. (F12) |
|              | ‘With me it’s usually hunger’. (M11) |
|              | ‘My brain would turn to jelly’. (M13) |

| Late symptoms | ‘My arms and legs jerk … my eyes start to flicker from side to side’. (F5) |
|              | ‘It just clouds over and I don’t know what’s going on’. (M4) |
|              | ‘I was twitching and sweating’. (M3) |
|              | ‘I get the most horrendous hot sweats, which turn into cold shivers and proper muscle convulsions’. (F2) |
|              | ‘I … close myself down’. (F1) |
|              | ‘It was bright and sunny and everything was wonderful, and it was just a lovely feeling’. (F1) |
|              | ‘Sweaty … disorientated … panicky’. (F6) |
|              | ‘Very shaky … or a cold sweat … my whole body aches’. (F7) |
|              | ‘Trouble concentrating and a bit agitated’. (M8) |
|              | ‘He [husband] ended up with eight stitches in his hand because I bit right through his hand’. (F12) |
|              | ‘It’s like being on an LSD trip’. (M13) |
|              | ‘I crawled up the street before’. (M14) |
|              | ‘You struggle and fight people’. (M13) |

The code in parentheses refers to participant gender and ID number.

### Barriers to prevention

Participant narratives revealed a number of recurring barriers to prevention of SH, relating to cognitive impairment, behavioural barriers and psychological factors.

**Cognitive impairment** (due to neuroglycopenia) was described (Box 3) as participants experiencing a ‘window of opportunity’ in time during which they could intervene to raise blood glucose concentrations and thus prevent progression to SH. Confusion was evident, with participants describing not knowing where their own kitchen was or getting to the cupboard only to forget why they were there. Some, however, also reported that they delayed acting to raise blood glucose levels (for as long as an hour), on the basis that they ‘still had time’ or that the hypoglycaemia was ‘only mild’.
Box 3. Cognitive impairment as a barrier to prevention.

'I can’t find my way from A to B, so when people say “go into the kitchen and get something to eat,” that’s fine but I don’t know where my kitchen is’. (F1)

'I stand in front of the cupboard and think “why am I here?”’. (F7)

'I had a kitkat in my pocket but I took half an hour trying to open it before I could get it in my mouth to eat’. (M14)

'The lower it goes, you get confused’. (M3)

'It’s just a fuzzy head type of thing. Lack of interest in anything but just total lack of concentration’. (F10)

'I go sort of … in his words … “a bit glakey”’. (F1)

'You say, “I’m fine, I’m fine, there’s nothing wrong”’. (F12)

'You just can’t get your words out’. (F12)

The code in parentheses refers to participant gender and ID number.

Box 4. Behavioural barriers.

<table>
<thead>
<tr>
<th>Behavioural barriers</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overtreatment with insulin</td>
<td>‘I probably overestimate rather than underestimate [the amount of insulin needed to cover treats/celebration meals] … Possibly I might … be taking too much insulin … It’s always at the back of my mind’. (F10)</td>
</tr>
<tr>
<td>Not acting on symptoms/acting too late</td>
<td>‘Sometimes I’ll get warnings and I don’t always react to them … There’s some times you don’t treat a hypo … you sort of block it out, “in a minute, in a minute …”’. (F6)</td>
</tr>
</tbody>
</table>

The code in parentheses refers to participant gender and ID number.

Psychological barriers (Box 5) were multifactorial. The first set of barriers can arguably be described as an absence of protective fear of hypoglycaemia. An absolute focus on hyperglycaemia avoidance was a major underlying issue for some, who described complete aversion to ‘high’ blood glucose (generally reported as above 10 mmol/L) and somewhat phobic behaviour (including overzealous ‘fiddling’ with insulin doses) as a result. There was strong overlap, though sufficient distinctions were made, between psychological burnout, lack of perceived control and preferring the state of hypoglycaemia. Those who mentioned these issues had experienced many episodes of SH and no longer felt able or inclined to deal with them successfully. For one individual,
hypoglycaemia was almost a ‘happy place’, one in which diabetes became someone else’s problem. This individual described liking ‘that better place’ – a detached state of mind experienced during hypoglycaemia in which reality faded and life’s difficulties could be forgotten. Social embarrassment was a concern, with participants indicating they would rather wait to treat a hypoglycaemic episode than leave a business meeting early.

**Box 5. Psychological barriers.**

<table>
<thead>
<tr>
<th>Hyperglycaemia fear/avoidance</th>
<th>Liking ‘that better place’</th>
</tr>
</thead>
<tbody>
<tr>
<td>‘I’d rather be in a hypo than blind’. (F1) ‘So, for my blood sugar to go up is the most frightening thing ever because that means I’m going to lose my sight … blindness, I really couldn’t deal with. Please just shoot me now, don’t let me go through that’. (F1) ‘I tend to worry more about hypers than hypos’. (M4) ‘You don’t want high blood sugars, going to clinics where people have had poorly maintained diabetes and all the complications, you know, it’s like psychological really’. (F6)</td>
<td></td>
</tr>
<tr>
<td>‘I probably worry more about going high than low, to be honest’. (F7) ‘Hypos doesn’t concern me at all in the scheme of things because I understand that that’s one of the things that I have to put up with. To have good blood sugar control is to have hypos, so I just don’t think it’s realistic to aim for good blood sugar control without having them, so to me they’re a kind of necessary evil. So, it’s the highs that I would get more upset and worried and concerned about’. (F7) ‘You know, I don’t want gangrene and I don’t want something to go wrong with my eyes’. (M10)</td>
<td></td>
</tr>
<tr>
<td>‘It could be very tricky because I’d need to leave the room’. (F6) ‘You think, oh, I can’t come out of this meeting to go and get my blood tests or whatever and you think, I’ll just finish the meeting’. (P12)</td>
<td></td>
</tr>
<tr>
<td>‘I don’t feel that it is in my control’. (M11) ‘It’s like walking a tightrope. As you go and keep walking on it, it gets thinner and thinner until eventually you’re in the middle, so you’re balancing all the time. You know, which way to go … You either keep it up or you go hypo. It’s a no win situation’. (M14) ‘Sometimes I just don’t want to bother any more. It’s a pfaff’. (M13)</td>
<td></td>
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<tr>
<td>'The gel) you know, it’s so bad that it makes you retch’. (F10) ‘I don’t really like glucose tablets. I find them quite difficult to eat’. (F7) ‘I avoid the glucose tablets like the plague. I really don’t like them’. (M4) ‘You know the glucose ones, they’re horrible. They make me sick. I struggle to eat them. I struggle to eat one, never mind have three or four of them’. (M3)</td>
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<tr>
<td>‘Having hypos doesn’t concern me at all in the scheme of things’. (F7) ‘I don’t have any fear of the hypos at all’. (F1)</td>
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<tr>
<td>‘I never feel, I never feel panicked when I have them. I always feel, you know, you’ll be alright but it’s only when it gets to that severe stage where I think, oh, I’d better do something now, it’s getting serious’. (F6)</td>
<td></td>
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<tr>
<td>‘prefer to let my blood glucose levels be a bit too low than to risk putting on weight because of snacking’. (F10)</td>
<td></td>
</tr>
<tr>
<td>‘I will make a manual change even if the pump tells me zero (if the wizard tells me give yourself no correction) … I’ll say, “well, I think you’re wrong there. I’m going to give this a little bit” … In the past what I would do is manually apply some compensation myself. I now realise that’s wrong … I was worrying so much about this hyperglycaemia that I was probably over compensating and then I was dragging myself into hypos’. (M4)</td>
<td></td>
</tr>
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</table>

Participants also reported significant dislike of the taste of glucose tablets, gel or drinks. They described situations where they had one of these recommended glucose sources available but did not use it, waiting instead until they had found a preferred alternative. Additionally, when experiencing symptoms indicative of rapidly progressing hypoglycaemia, some participants did not use a short-acting glucose source but relied instead on a longer acting alternative such as chocolate. Some appeared to regard the occurrence of hypoglycaemia as an opportunity to eat chocolate, which they would otherwise
‘forbid’ themselves from eating, while others evidently regarded chocolate as indistinguishable from short-acting hypoglycaemia treatments. Indeed, participants whose lives were significantly impaired by both recurrent SH and IAH used chocolate bars as a preferred primary recovery source. It was evident from their narratives that this strategy was the result of feeling deprived by a ‘diabetic diet’ and was not effective in halting the progression of hypoglycaemia before collapse or loss of independent function occurred.

**Discussion**

**Summary of main findings**

This is the first qualitative study, to our knowledge, that has explored the progression to SH, and barriers to prevention of SH, from the individual’s perspective, among adults with long-standing T1DM and a history of recurrent SH. A number of observations arose from these accounts. Awareness of hypoglycaemia symptoms, frequent self-monitoring of blood glucose and detailed knowledge of how best to raise blood glucose concentrations were necessary but not sufficient to prevent SH. Beyond this, SH can be understood as a complex phenomenon requiring cognitive capacity; identification of activities/times associated with higher risk; recognition of early symptoms; a tailored glucose self-monitoring plan to minimise risk/maximise early recognition; a specific action plan for treatment of low blood glucose levels; the will to intervene (positive psychological adjustment); and timely intervention. If early symptoms of hypoglycaemia are associated with mild cognitive deficits (e.g. lack of concentration or attention), it becomes even more imperative to have a clearly formulated action plan/intervention. Such an action plan will allow automaticity to compensate for impaired concentration. This will capture the value of acting to raise blood glucose concentrations early in the ‘window of opportunity’ to thereby prevent progression to SH.

Furthermore, the choice of intervention to raise blood glucose concentrations was likely to be more successful if the individual identified a treatment that was preferred (or at least palatable). For example, dislike of glucose tablets meant some individuals were reluctant to use them despite regarding them as a recommended treatment, while other participants successfully used alternative, more palatable, treatments. The choice of intervention also influences progression to SH. While fast-acting treatments (e.g. glucose, fruit juice, jelly beans) will be suitable either early or late in the progression, slow-acting treatments (e.g. chocolate, biscuits) may take too long to raise blood glucose concentrations and be particularly problematic if used later in an episode. The choice of intervention will influence blood glucose levels post episode. If an excessive dose of fast-acting glucose is used (or excessive quantities of slow-acting treatments are eaten), this is likely to result in rebound hyperglycaemia and a consequent delay in achieving glycaemic stability.

The validity of these findings is supported by our clinical observations and evidence from other studies. The idiosyncratic nature of hypoglycaemia is well recognised, as is the occurrence of transient hypoglycaemia-induced cognitive impairment or poor concentration as a reliable indicator of low blood glucose concentrations. Historically, much emphasis has been placed on autonomic symptoms as key to the detection and prevention of hypoglycaemia. However, our findings suggest that the earliest warning sign experienced by many people was mild cognitive impairment and that progression to SH occurred despite recognition of these symptoms. This is consistent with findings elsewhere. Gonder-Frederick et al. emphasised that neuroglycopenic symptoms should be regarded as of equal value to autonomic symptoms in detection of hypoglycaemia, and cognitive impairment is a key driver in the biopsychosocial model of hypoglycaemia. Likewise, it has been reported previously that people with insulin-treated diabetes who have optimal glycaemic control experience a decline in mental performance before the onset of warning symptoms of hypoglycaemia. As others have noted, education is a pre-requisite for an appropriate response to impending hypoglycaemia. Furthermore, as noted elsewhere, dislike for recommended treatments, such as glucose gel or dextrose tablets, suggests that the range of treatment options needs to be explored fully by an understanding clinician to minimise habitual choice of less immediately effective primary interventions. Finally, recent neuro-imaging studies indicate that IAH is associated with a reduced ability to perceive hypoglycaemia as unpleasant or dangerous, which may undermine motivation and ability to act in appropriate ways to prevent or delay SH. Narrative accounts of delays in taking appropriate actions may be explained, at least in part, by these latest findings. A psycho-educational programme has now been developed which may offer benefit to those with an inappropriate lack of concern about hypoglycaemia, for whom traditional educational approaches have not been sufficient.

**Strengths and limitations**

The validity of our findings is further supported by the number of interviews conducted and by the frequency with which similar themes emerged during our careful cross-checking of the data. It is a strength of the grounded theory approach to analysis used in this study that the themes themselves (in this case barriers to the prevention of hypoglycaemia) derive directly from the narrative of individual participants and, as such, are more likely to represent an accurate reflection of hypoglycaemia progression than would interviewer- or questionnaire-driven accounts. The way in which people are asked to describe their individual symptoms of hypoglycaemia influences their accounts, and rank order of symptoms has been shown to vary if people are asked to attribute relevance ratings to their reporting of symptoms. By eliciting unprompted narratives about the way hypoglycaemic
episodes manifest through time, this study offers valuable insights into the relative usefulness of specific symptoms as cues to prompt self-treatment. Moreover, the processes by which symptoms are ignored or not acted upon in everyday life can be better understood.

Given the qualitative nature of this study, we are cautious not to overgeneralise the findings. The insights reported here have implications for the prevention of recurrent SH but may be less relevant to those with T1DM who have little history of SH and experience only isolated episodes with clearly identifiable precursors. Hypoglycaemia awareness status was not formally assessed using quantitative measures at the time of the interview or by recruiting physicians. Given the focus on a qualitative approach by independent interviewers, we aimed to minimise potential for bias and encourage open and honest interviews by avoiding immediately preceding health-care professional assessments/questionnaires, which could be perceived as directing ‘acceptable’ responses. Nevertheless, participants were selected to encompass a broad range of preceding hypoglycaemia awareness despite their shared experience of SH.

Several other caveats apply: this study included a largely Caucasian sample. It was conducted in only two UK tertiary diabetes centres, although the interviewees comprised a substantial proportion of the people with recurrent SH in each centre. Recall of SH is considered highly reliable for up to 12 months; while most participants had experienced at least one episode within this time frame, there was unavoidable reliance on participant recall, in addition to participants’ and interviewers’ interpretation of symptoms and factors influencing progression to SH. It follows that we can only consider these findings to be useful insights rather than facts. Nonetheless, at least some of the barriers identified may be contributory factors whenever SH occurs in the context of retained symptom awareness, and recognition of these barriers may contribute to avoidance of SH.

**Implications for future research or clinical practice**

Clinical practice guidelines for the management of adult hypoglycaemia state the imperative to address the issue of hypoglycaemia ‘in every contact with patients with drug-treated diabetes, particularly those treated with insulin secretagogue or insulin’ (p. 721). Moreover, it is accepted that early intervention to stop progression towards a more severe event is always preferable. Unfortunately, the ‘window of opportunity’ to act diminishes over time. The findings of this study serve to identify individualistic barriers to hypoglycaemia prevention and to suggest ways in which these barriers can be overcome through patient-centred clinical care.

The value to the individual of having intact symptomatic awareness of hypoglycaemia is well recognised. However, transient neuroglycopenic symptoms of mild cognitive impairment such as lack of concentration (or ‘fuzzy thinking’) are likely to be particularly useful cues to prompt a ‘just in time’ dose of glucose since further deterioration in cognitive function makes successful self-treatment increasingly less likely. Indeed, it may be possible to identify a specific ‘complex’ cognitive task that the individual usually finds straightforward but becomes difficult during hypoglycaemia (such as a mental arithmetic calculation or writing test), which an individual can adopt, as a check to confirm or exclude insipient hypoglycaemia requiring treatment. Such a task would have the added benefit of screening for potential hyperglycaemia in individuals for whom subtle symptoms can be experienced during both hypo- and hyperglycaemia but may not be otherwise differentiated. Moreover, the development of a clear personalised action plan for early intervention is also central to prevention, particularly where impaired concentration/attention may cause delay in treatment. Individuals can be encouraged to consider and prepare for possible hypoglycaemia so that implementing their own personal action plan to raise glucose becomes both automatic and immediate in order to prevent progression to SH.

Likewise, it may be of significant value to reinforce the importance of using a minimal dose of fast-acting treatment (e.g. glucose, fruit juice). This recommendation is part of many established diabetes structured education programmes, such as Dose Adjustment For Normal Eating (DAFNE), which have demonstrated significant reductions in SH but may not be reinforced sufficiently in individual consultations, informed by sensitive exploration of potential barriers to following this advice. While all glucose sources will eventually achieve the goal of normoglycaemia, use of slow-acting glucose late in the progression of a hypoglycaemic episode may not raise blood glucose concentrations in time to prevent loss of function or collapse. Moreover, a larger than necessary dose of glucose at any stage will complicate the return to optimal blood glucose levels, leading to rebound hyperglycaemia and the use of additional short-acting insulin, thereby precipitating further hypoglycaemic episodes. For some individuals, fear of rebound hyperglycaemia after treatment creates reluctance to treat hypoglycaemia at an early stage, but clinicians can emphasise the success of an appropriate dose in preventing SH without ensuing hyperglycaemia. Such fears, resulting in absolute hyperglycaemia avoidance, can also be a contributing psychological factor and have been recognised elsewhere.

Finally, adaptation of treatment recommendations to personal preference is valued by many individuals and will further enhance willingness to treat mild hypoglycaemia and increase likelihood of SH prevention. For example, accommodating a dislike of glucose tablets or drinks, and substituting these with a suitable alternative, such as jelly beans, can eliminate potential reluctance to treat early symptoms.

As these personal accounts illustrate, hypoglycaemic symptoms are idiosyncratic and also differ intra-individually between episodes. Individual cognitive, behavioural and psychological barriers to the prevention of SH exist...
(independent of treatment risks), and it seems likely that these specific factors need to be addressed by the individual and his or her medical team if recurrent SH is to be avoided. Psycho-educational interventions for people at risk of SH may likewise offer the greatest benefit if they reflect a multifactorial approach, cognisant of all the potential underlying barriers identified in this study. Recent qualitative research with people who have IAH suggests that cognitive biases may impair individuals’ ability to instigate behaviours that facilitate hypoglycaemia avoidance and/or regained awareness of hypoglycaemia. However, SH may be experienced by many people who have retained hypoglycaemia awareness as well as those with impaired or absent awareness, and our data support that the route to hypoglycaemia prevention may need to be tailored accordingly. Informed by the insights gained from this study, a brief psycho-educational intervention has been developed with the specific goal of preventing SH in those with reduced awareness of hypoglycaemia and/or previous severe events. It has been named the 'Hypo COMPaSS' educational tool, and as part of its initial validation, it has been used by all participants in an ongoing interventional trial (ISRCTN52164803).

Conclusion

In summary, our qualitative data highlight the multifaceted and idiosyncratic nature of SH and provide rich personal accounts. They support clinical observations and previous quantitative findings that individuals with a history of recurrent SH may have specific thought and behaviour risk profiles. Individualised prevention plans for these people should emphasise both the need to attend actively to individual symptoms of mild hypoglycaemia and to intervene promptly (at personal cues) with an appropriate and acceptable glucose source in order to prevent progression to SH. For some, further work to cognitively reframe SH may be needed, particularly among those with extreme hyperglycaemia avoidance.

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The authors declare that they have no conflicts of interest.

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