GUIDELINE FOR NEUROLOGICAL PROGNOSTICATION AFTER HYPOXIC ENCEPHALOPATHY

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Key Amendments

Date	Amendment	Approved by
8th October 2019	Document extended with no changes as part of Disease Management section in critical care	Dr Nick Cowley/Dr Andy Burtenshaw

INTRODUCTION

Over recent years we have seen a steady increase in the numbers of patients admitted to intensive care with a return of spontaneous circulation after resuscitation for cardiorespiratory arrest. However, mortality in this group of patients remains high at approximately 50%, with over 70% of deaths attributable to hypoxic encephalopathy [1].

Therapeutic hypothermia had been shown to improve neurological outcome and was widely practiced over the last decade. However more recent evidence has shown that prevention of pyrexia in the 24 hours following return of spontaneous circulation (ROSC) is equally efficacious and we have recently changed our practice to target a temperature of 36°c.

Neurological prognostication is essential as a foundation for informing relatives, prioritising intensive care resources, avoiding futile care in cases where a vegetative state or death can be anticipated, and ensuring that treatment withdrawal decisions are appropriate. Poor neurological outcome is widely regarded as requiring full nursing care, persistent vegetative state or death.

Prediction of poor neurological outcome has been based on guidance published in 2006 from the American Academy of Neurology [5]. In particular, the early presence of myoclonus status epilepticus, the absence of pupillary and corneal reflexes and a motor response no better than extension at 72 hours were considered reliable predictors of poor outcome.

However, there is mounting evidence that patients who have undergone therapeutic hypothermia / targeted temperature management and who have the potential to make a good recovery are not reliably identified based on neurological examination at 72 hours alone [6-8]. In a recent case series, 16% of patients with a poor initial motor score progressed to make a good recovery [7]. The reasons for this discrepancy are not established, although there is evidence that the metabolism and clearance of sedative drugs is delayed in patients who have undergone hypothermia [9]. Patients who improve their level of consciousness after withdrawal of sedative medication usually have a good outcome [10]. For those that remain in a coma, prognosis becomes gradually worse with increasing time from the insult [11].

Data from a recent trial of over 900 patients admitted to intensive care following successful resuscitation from cardiorespiratory arrest showed approximately 15% of patients died, predominantly from multiple organ failure within the first 24-48 hours. Approximately 50% of patients woke up with a good Glasgow Coma Score once sedation was turned off. The remaining 35% of patients who did not regain consciousness required a structured

neurological prognostication to support withdrawal of life sustaining support in those whom a vegetative state or death could be anticipated [4].

In Worcestershire, we admit approximately 60 patients annually with anoxic encephalopathy. The majority of these are as a result of resuscitation from cardiac arrest. It is estimated that 10-20 patients annually will remain comatose following cardiorespiratory arrest and will require structured neurological prognostication.

The European Resuscitation Council and the European Society of Intensive Care Medicine have recently acknowledged the uncertainty surrounding prognostication and have published an advisory statement addressing this.[12] This guideline incorporates this statement along with the latest evidence base to provide a structured approach to neurological prognostication in patients with hypoxic encephalopathy Worcestershire Critical Care Units.

DETAILS OF GUIDELINE

0-24 hours post arrest:
Where appropriate, patients who regain spontaneous circulation after cardiorespiratory arrest and who remain comatose are admitted to the Intensive Care Unit. They are sedated and ventilated, may have undergone percutaneous coronary intervention and may be actively cooled to prevent pyrexia >37^{o}c for 24 hours. Any organ dysfunction is actively supported during this time.

24-72 hours post arrest:
Period of passive rewarming if patient has been actively cooled.
If clinically able, sedation is discontinued to allow assessment of neurological state.

Reasons for withdrawing active support in the first 72 hours include:

- Progressive multiple organ failure
- Evidence of a pre-morbid state that would limit Intensive Care Support
- Evidence of brainstem death.
- Status myoclonus (persistent myoclonic jerks for greater than a minimum of 30 minutes) **in combination** with absent bilateral N20 response on somatosensory evoked potential (SSEP)

72 hours post arrest: Patients who remain comatose with GMS 1-2 ONLY

If possible, perform EEG and SSEPs. This should occur no earlier than 72 hours post ROSC (unless myoclonus) and only in patients with GMS 1-2

Consider CT / MRI

Reasons for withdrawing active support at 72 hours:

- Bilateral absence of pupillary and corneal reflexes or
- Bilateral absence of SSEP N20 response

Two of more of:

- Status myoclonus >30 minutes within the first 48 hours
- Absence of EEG reactivity
- Unreactive burst suppression or status epilepticus on EEG
- Diffuse anoxic injury on brain CT or MRI

If patient remains comatose without meeting criteria for withdrawal of support then continue active management for further 48 hours.

In addition:

- EEG can often be equivocal – neurophysiology advice is to repeat EEG after 24 hours in these patients to look for changes.
- SSEPs will be reported as bilaterally present, bilaterally absent or indeterminate. If used to aid decision making about withdrawal of active support, then only bilaterally absent N20 responses should be considered to be significant.

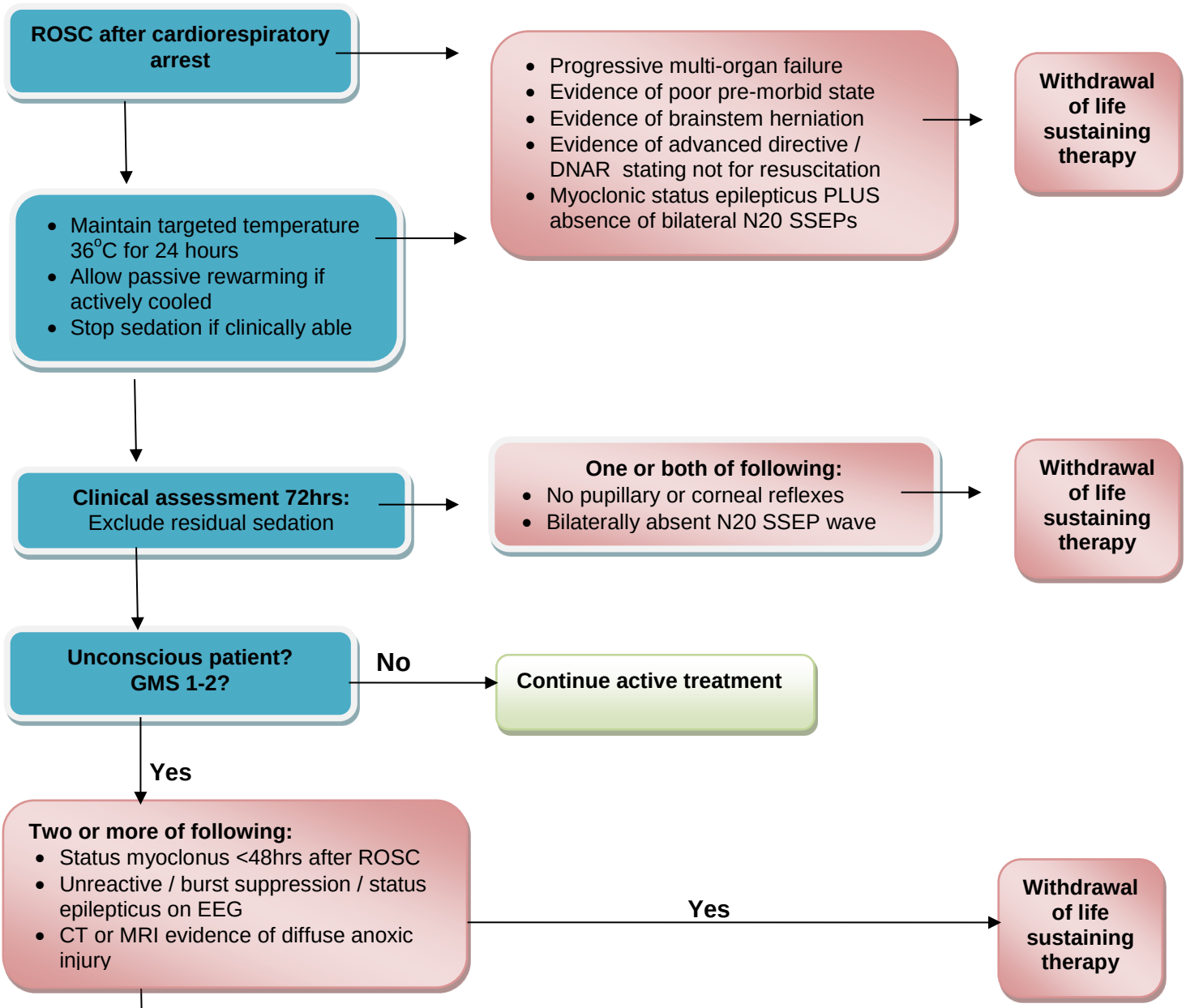
Days 4 – 5 post arrest:

- Twice daily clinical neurological assessment.
- Repeat EEG

If at day five, patient remains comatose with GMS 1-2, then poor outcome remains likely.

Withdrawal of active life sustaining therapy should be considered. This decision should ideally be made by two Intensive Care Consultants.

The above is summarised in the following flow sheet:



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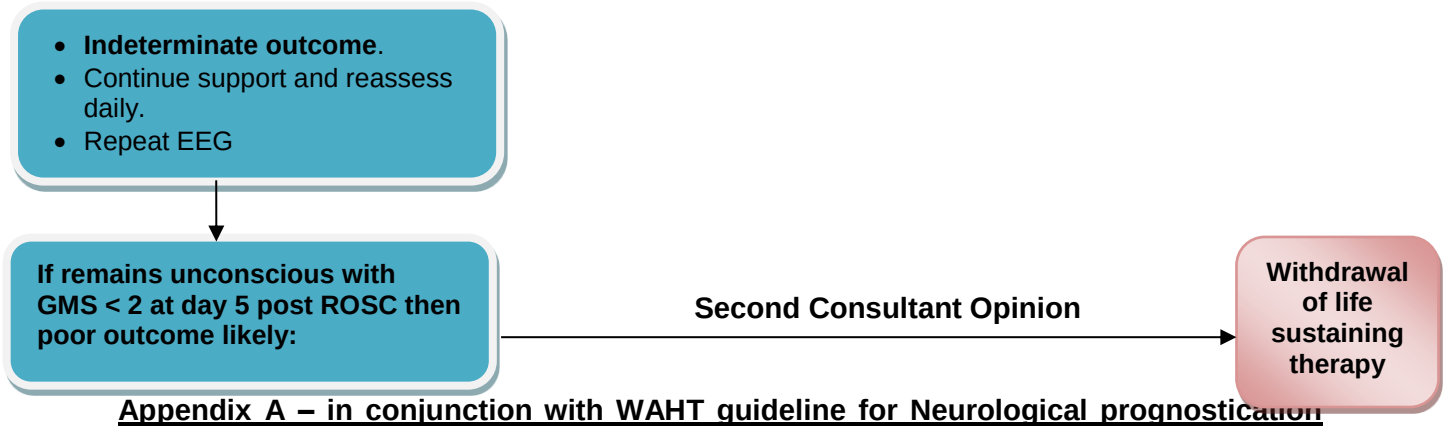


Appendix A – in conjunction with WAHT guideline for Neurological prognostication after hypoxic encephalopathy.

Guideline for median somatosensory (SEP) evoked potential ITU.

Introduction

The median SEP is used to check the integrity of the peripheral and central pathways of the median nerve, recording at the mid-clavicular position (Erb's point), dorsum of the neck (this consists of potentials generated at the dorsal roots, dorsal horns and sensory tract of the spinal cord and structures of the lower brainstem) and contra lateral hand area of the sensory cortex (consists of activity in the thalamus, the sensory radiations and sensory cortex).

Preparation

Appropriate timing of SSEP request – follow the WHAT guideline for neurological prognostication after hypoxic encephalopathy

- ITU should be informed of the investigation time
- Patient data should be entered on the computer.
- Clinical history should be collected from the notes and documented on the history sheet.
  Limitations to electrode placement of peripheral sites
  Previous co-morbidities that effect peripheral nerve conduction
  Previous neck injury
- The procedure should be explained in full to the patient and any parents/carers, ensuring all parts of the investigation are understood and that consent is enlisted.

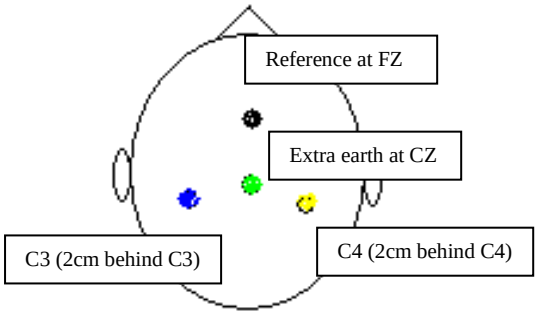
Positioning the patient

Ensure there are no external sources of interference and the patient is in a safe position.

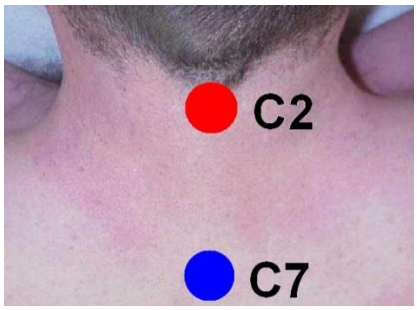
All neck braces, collars must be removed. Clear access is needed to both arms/ wrists.

Electrodes

On the head



At the neck



Electrodes for median SEP's are as follows:

Median SEP channels

	Active	Headbox	Reference (FZon headbox)
<u>Right median nerve</u>			
Channel 1	**Erb's point**	**O2**	**FZ**
Channel 2	**C7**	**OZ**	**FZ**
Channel 3	**C2**	**PZ**	**FZ**
Channel 4	**Cortical**	**C3**	**FZ**
<u>Left median nerve</u>			
Channel 1	**Erb's point**	**O1**	**FZ**
Channel 2	**C7**	**OZ**	**FZ**
Channel 3	**C2**	**PZ**	**FZ**
Channel 4	**Cortical**	**C4**	**FZ**

*** 7 cm down from CZ and 2cm back**

The reference electrode is placed at FZ on the head and plugged into FZ on the headbox.

Electrodes are placed at 7 cm down from CZ either side (towards the ear) and 2 cm back (i.e. 2 cm behind the C3/C4 placement). These are plugged into C3 and C4 on the headbox respectively.

An electrode is placed over Erbs point (on the side being tested) and plugged into O2/O1 on the headbox.

An electrode is placed at C7 (on the prominent bone) and plugged into OZ in the headbox.

An electrode is placed at C2 (just above the hairline) and plugged into PZ in the headbox.

A second earth electrode can be used and this is placed at CZ area on the head and is plugged into the green slot on the headbox.

In addition, an earth electrode must be applied to the patient's arm.

Check electrode resistances at this point and ensure they are less than 5 kOhms where possible.

Machine settings for median SEP are as follows:

LFF- 3 Hz

HFF – 1 kHz

Sensitivity - 2µV

Sweep – 50 ms

Artefact reject – 50µV or higher

Averages – 128

Repetition rate – 2 - 4 pps

(These should already be set)

Recording

Attach the stimulator over the median nerve at the wrist (negative up), ensuring the skin is clean and dry. It is necessary to ask for bandages to be removed to allow access to the stimulation point.

Encourage the patient to relax.

To acquire the data, the average button should be pressed on the stimulator box (button 2). Make sure that you are clicked in the appropriate box in order to acquire and average the data. When you are happy with the averaged data, press button 5 to move the data onto stored data on the left hand side of the screen.

Turn the stimulator up slowly until there is a visible twitch of the thumb you may need to readjust the location of the stimulator over the wrist to get a visible twitch.

Acquire and record a minimum of 128 averages. Traces should be repeated to allow for reproducibility.

Repeat on the other side.

Reporting

All waveforms should be marked appropriately. Waveforms can be marked by clicking the lower X on the top right; this will then display the markers. Make sure you widen the table on the right, so that amplitude values are displayed.

Reports should contain no waveforms – these should be printed separately (landscape view) with the sensitivity turned up for the consultant to view.

Test data should be compared to normal control comparative data.

Reports should be printed with a written factual and given to the consultant to report.

Interpretation of SSEP

a. Present – must have peripheral and cortical potential (39-46% favourable outcome)
b. Absent – must have peripheral response with absent cortical (bilateral) (false positives, <24-48hrs post cardiac arrest)
c. Indeterminable

References: Appendix A

Walsh et al. (2005) The clinical role of evoked potentials. 76 Journal of Neurology, neurosurgery and psychiatry.
Michael J.A.M Van Putten 2011 Clinical neurophysiology, science direct.
E.F.M Wijdicks et al 2006 Practice parameter: predication of outcome in comatose survivors after CPR
Cruccu et al 2008 Recommendations for the clinical use of SSEP; clinical Neurophysiology.