National Institute for Health and Care Excellence

Sepsis: the recognition, diagnosis and management of Severe Sepsis

Stakeholder Comments – Draft Scope

NOTE:

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Please fill in both the 'stakeholder organisation' and 'name of commentator' fields below in order for your comments to be considered.

Stakeholder organisation:	**[Faculty of Intensive Care Medicine]**
Name of commentator:	**[Prof Mervyn Singer]**

Comment No.	Section number Indicate **number** or **'general'** if your comment relates to the whole document	Comments Please insert each new comment in a new row. Please do not paste other tables into this table, as your comments could get lost – type directly into this table
Example	3.4.6	Our comments are as follows …….
Proformas that are not correctly submitted as detailed in the line above may be returned to you.		
1	3.1 (a)	These definitions are either outdated or plain wrong!
2	3.2 (a)	It is well recognized by the critical care community that the current definitions developed in 1992 are no longer fit for purpose. There are major issues with over-diagnosis and concerns over excess/inappropriate use of antibiotics. I am Co-Chair of a North American/European Sepsis Redefinitions Task Force organized by the (US) Society of Critical Care Medicine and the European Society of Critical Care Medicine that is due to report by early 2015. We are hoping, as part of the redefinitions, to generate an improved organ dysfunction scoring system that will improve the sensitivity and specificity of diagnosis. This will involve interrogation of very large patient databases (emergency dept/general ward/ICU) with subsequent validation against other databases (including UK populations).
3	3.2 (a)	Again, errors here- eg sepsis does not necessarily have to involve two or more organ systems in terms of obvious dysfunction

4	3.2 (b)	Again, errors and omissions. Young children are not particularly susceptible (unless they have other risk factors), except with certain types of infection eg influenza or meningococcus. The elderly are a much greater at-risk population – 13-fold increased risk in over 65s compared to under 65s (Martin GS et al. The effect of age on the development and outcome of adult sepsis. Crit Care Med. 2006;34:15–21). Women following childbirth also does not represent a high-risk group – the RCOG (2011 Confidential Enquiry) reported that between 2006-8 there were 29 deaths from sepsis yet approx. 2m births would have occurred in this timespan. Clearly, maternal sepsis should be identified and treated promptly, though this is a relatively rare problem.
5	4.1.1.(b)	See point 4 above re: pregnant women.
6	4.3.1 (a)	Though not at liberty to divulge the revised sepsis definitions (see point 2), the current definition of 'sepsis' can include someone with a bad cold. Such patients do not need hospital admission, let alone antibiotics. More emphasis needs to be placed on the early recognition of new onset organ dysfunction, and to consider whether infection is the underlying cause. Scoring systems are generally flawed in that they do not take into account coexisting morbidities that affect many (? the majority) of patients with severe sepsis, nor age (e.g. normal blood pressure varies ++). Alas, none have been properly validated and specificity, in particular, is not great for any of them.
7	4.3.1(b)	None of the list given are specific for sepsis. All current clinical and laboratory markers of 'sepsis' are not particularly specific. Any cause of an exaggerated inflammatory response (e.g. reaction to blood transfusion, drug reaction, response to surgery) can generate a virtually identical clinical and biochemical picture as sepsis.
8	4.3.1 (b)	It's lactate – not lactic acid. Why haemoglobin? Liver function tests are more valid (for cholecystitis/cholangitis)
9	4.3.1 (b)	Again, factually incorrect statement – a normal white count does not mean an overwhelmed immune response.
10	4.3.1 c (iii)	Clearly, delay is generally not a good idea for treating severe sepsis but not so valid for 'sepsis' (see point 6 above) There is no good evidence to support the rationale that treatment within a 'golden hour' makes any difference to outcomes. Oxygen is only appropriate for correcting hypoxaemia and acid-base balance does not need to be corrected per se. Treating the underlying cause (e.g. hypovolaemia will often improve lactate etc… . Specifically targetting acid-base balance (e.g. with bicarbonate) is voodoo.
11	4.3.1 c	'Early treatment with vasopressors in people with sepsis' – I hope not! I think you mean vasopressor treatment for patients with shock who have not first responded to appropriate fluid resuscitation. Vasopressor agents are harmful in themselves.
12	4.3.1. c	The treatment of sepsis is not a medical emergency. See point 6 above re: a bad cold. I think you mean what is currently called 'severe sepsis'. Inotropes should not be considered as soon as severe sepsis is suspected. Same applies as for Point 11 above.
13	4.3.1.f	Lactate not lactic acid. Urine output, conscious level, pain, pulse oximetry need to be added

14	4.4b	Do you mean ‘progression to severe sepsis’? Problem with outcomes is that comorbidities often dictate outcomes, length of stay etc.. and not the infection/sepsis/severe sepsis. For instance, a patient with bad COPD who gets a pneumonia may end up on a ventilator and not be weaned off the machine despite recovering from the pneumonia/sepsis. Also, sepsis is often the final nail in the coffin of a terminally ill patient, eg. with advanced cancer or severe end-stage liver disease.
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Please add extra rows as needed.

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