Bioethics ISSN 0269-9702
Volume 15 Number 1 2001

A PAIN IN THE FETUS: TOWARD ENDING CONFUSION ABOUT FETAL PAIN

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ABSTRACT

Are fetuses, at any stage of their development, capable of feeling pain? In his paper, 'Locating the Beginnings of Pain', Stuart Derbyshire argues that they are not. We argue that he reaches this conclusion by way of conceptual confusion, a misreading of the available scientific data and the inclusion of irrelevant data. Despite his assertion to the contrary, the work of most scientists in the area supports the conclusion that fetuses can feel pain. At the outset we examine the concept of pain and distinguish it from the allied concept of nociception, with which it is sometimes confused. With the relevant conceptual framework in place, we elucidate the problem of determining when, in its development, a human becomes capable of feeling pain. We then examine the available data showing how, on balance, it tends more to support than undermine the claim that fetuses of around 28 to 30 weeks' gestation are capable of feeling pain.

INTRODUCTION

Are human fetuses, at any stage of their development, capable of feeling pain? One requirement for answering this question is an understanding of the available scientific evidence. For this reason, we agree with the (former) editors of this journal that if the question can only be answered by providing a technical scientific account, then there is good reason for bioethics journals to publish papers outlining the relevant data. There are dangers, however, in publishing technical scientific data in a general bioethics journal. These are well illustrated by Stuart Derbyshire's paper 'Locating the Beginnings of Pain',[1] which includes unnecessary technicality, and, therefore, is likely to

[1] S.W. Derbyshire. Locating the Beginnings of Pain. *Bioethics* 1999; 13,1: 1–31.

confuse rather than enlighten many readers. This problem is compounded by the incorrect impression his paper gives that the weight of scientific evidence speaks against the possibility of fetal pain.[2] The waters are further muddied by the confused concepts of pain and consciousness that he employs.

At the outset we shall examine the concept of pain and distinguish it from the allied concept of nociception, with which it is sometimes confused. With the relevant conceptual framework in place, we shall elucidate the problem of determining when, in its development, a human becomes capable of feeling pain. We shall then outline Derbyshire's argument for the conclusion that the fetus does not feel pain. There is an extensive literature on fetal and neonatal pain (reviewed, for example, by K. Anand and P. Hickey)[3] and we cannot hope to examine it all. We shall focus primarily on the studies to which Derbyshire refers. These studies are reasonably representative of the available data. We shall summarise the relevant data and show how, given a clear understanding of what pain is, the scientific evidence does not support the conclusion he draws.

WHAT IS PAIN?

We agree with Derbyshire that one cannot determine whether a fetus feels pain unless one has a conception of what pain is. Unfortunately his definition, although not uncommon, has serious shortcomings. He accepts a definition of pain as 'a sort

[2] He claims that unlike J. A. Burgess and S. A. Tawia [When Did You First Begin to Feel It? Locating the Beginning of Human Consciousness. *Bioethics* 1996; 10,1: 1–26], 'medical researchers largely concluded that the fetus could not feel pain, regardless of gestational age' (p. 2). The medical literature he cites in support of this claim comprises six references. The only original research article amongst these demonstrates hormonal responses to intrauterine needling of fetuses. As we show later, although the authors are cautious in interpreting their data, the thrust of this paper is, in fact, in favour of fetal pain. Two of the references are to discussion papers he has authored and the remaining three are also opinion pieces. The two opinion pieces that reject the possibility of fetal pain do so without providing or referring to original data. As such they cannot be construed as accurately representing the available scientific evidence. Moreover, it is curious that in referencing this claim, he does not cite a number of other articles (which he mentions elsewhere in his paper) which argue to a different conclusion.

[3] K.J.S. Anand and P.R. Hickey. Pain and its Effects in the Human Neonate and Fetus. *New England Journal of Medicine* 1987; 317, 21: 1321–1329.

of amalgam of cognition, sensation and affective processes.'[4] It is, he says, 'a conscious experience which may be modulated by mental, emotional and sensory mechanisms and includes both sensory and emotional components'.[5] He distinguishes this from an understanding of pain 'as merely a physical sensation of noxious stimulus and disease'.[6] He notes that his definition is congruent with the International Association for the Study of Pain's (IASP) definition of pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'.[7]

What these definitions correctly capture is the subjective experiential feature of pain. Pain is typically something that is consciously felt. Although one can be injured or diseased without being aware of it, one cannot be in pain without being conscious of it. Similarly, although one may not be injured or diseased one can still feel pain. Pain is, essentially, an unpleasant *feeling*. This is not to say that every unpleasant feeling is rightly called 'pain'. Perhaps some feelings, although unpleasant, are not sufficiently severe to merit the designation 'pain'. Thus, we might say that pain is an unpleasant feeling of a sufficient degree of severity. Of course, this leaves open the problem of distinguishing a non-painful unpleasant feeling from a painful one. While pain is by definition worse than non-painful unpleasant feelings, it is also the case that pains can be of varying degrees of severity. Thus, the semantic distinction between pain and other unpleasant feelings should not be invested with too much moral significance. Varying degrees of unpleasant feeling, whether termed pain or not, must be given moral weight commensurate with the degree of unpleasantness. Although we shall focus on pain, much of what we shall say will also have a bearing on less severe unpleasant mental states.

Derbyshire, like the IASP, thinks that emotion is an essential feature of pain. As noted earlier, the IASP claims that pain is 'an unpleasant sensory *and emotional* experience'(our emphasis).[8] Derbyshire's definition is more ambiguous. He says that pain is a 'conscious experience which *may* be modulated by mental,

[4] *Op. cit.*, Note 1, p. 4.
[5] *Ibid.*
[6] *Ibid.*
[7] H. Merskey, D. Albe-Fessard, J. Bonica, A. Carmen, R. Dubner. Pain Terms: A List with Definitions and Notes on Usage. Recommended by the IASP Subcommittee on Taxonomy.' *Pain* 1979; 6, 3: 249–252.
[8] *Ibid.*

emotional and sensory mechanisms' (our emphasis).[9] The use of 'may' suggests that emotions are not necessary for pain. However, he continues his definition of pain by saying that it 'includes both sensory and emotional components', which implies that both the sensory and emotional components are essential. Although his definition is ambiguous, the structure of his argument – or, at least one of his arguments – clearly reveals that he thinks emotions are essential to the experiencing of pain. One argument he offers is that because fetuses are incapable of emotions, they are incapable of experiencing pain. The conclusion only follows from the premise if there is an additional premise that emotions are essential for pain.

There are various ways in which one can understand what is meant by 'emotion' and how it relates to pain. Understood very broadly, 'emotion' could include the unpleasant feel of pain.[10] On this view, pain is itself a kind of emotion. But this cannot be the sense in which Derbyshire uses the term 'emotion'. If it were, then arguing that fetuses cannot feel pain because they cannot have emotions would be circular. To say that fetuses are incapable of (the sort of) emotions (involved) in pain would just be to say that fetuses are incapable of having pain. An alternative view of emotion would be that it is logically distinct from the feel of pain, even if it can modulate pain in specific circumstances. That is to say, although pain can be enhanced, or dulled or otherwise affected by the emotions, the experience of pain is possible without this emotional overlay. Having the raw unpleasant feel of pain would be possible in the absence of emotions, where emotions are understood as something more than the unpleasant feel of pain. This is the sense in which we shall use the term 'emotion'.

Pain also needs to be distinguished from 'nociception'. Nociception refers to the neural activity in those peripheral receptors and centripetal (that is, afferent) pathways via which noxious stimuli are transmitted to the brain. Put more simply, it is the process whereby noxious stimuli are sensed and transmitted to the brain. Thus, while nociception is neural activity, pain is an unpleasant feeling. It follows that while pain requires some level of consciousness, nociception does not. Although nociception often causes pain, it is possible for nociception to occur without there being pain, when, for

[9] *Op. cit.*, Note 1, p. 4.

[10] The IASP seems to hold this view. According to them, pain is 'always unpleasant and therefore … an emotional experience'.

example, a noxious stimulus is administered below the level at which a spinal cord has been transected (severed). Similarly, pain may occur in the absence of nociception, as happens when a person reports pain in the absence of tissue damage or another peripheral stimulus. Thus, while nociception and pain are distinct concepts, the former often causes the latter.

FETAL AND OTHER MINDS

The problem of determining whether fetuses are capable of feeling pain is an instance, albeit a particularly troubling one, of a more general philosophical problem – the problem of 'other minds'. The problem of other minds is the problem of whether one can know whether anybody else has a mind and, by extension, whether they have thoughts, perceptual experiences and pains. Now it may seem to many that the problem of other minds, quite unlike the problem of fetal pain, is the absurd sort of question, which only philosophers could entertain or take seriously. Those tempted to say this, should note that our claim is not that the problem of other minds and the subsidiary problem of fetal pain are equally troubling in practice. Rather, we are claiming that, as a species of the other minds problem, the question of fetal pain arises in the same way: One has direct access to one's own mind and one's own thoughts, experiences and pains. One feels one's own pains, for example. One has no such access, however, to the pains of others. How does one know whether other humans and animals have minds and feel pain? Those wanting to accept no evidence short of subjective experience of another's mind or pain, will have no actual way of determining whether others have minds or whether fetuses feel pain.

Clearly, any judgment that another being has a mind or is in pain will have to be *inferred* from evidence other than subjective experience of their minds. Typically the evidence we use is a combination of behaviour and the presence of the neurological anatomy and physiology required for bringing about pain. That is to say, we notice that others behave as if they are in pain in the sorts of circumstances in which we would feel pain and behave in those ways. This, coupled with evidence that others have the neurological mechanisms capable of causing or feeling pain, leads us to infer that others are capable of feeling pain (unless we have reason to think that they are feigning).

Now the sort of pain behaviour we witness in those most like us – other humans with whom we can speak – includes their reports

of being in pain. When dealing with beings with whom we cannot speak – animals, pre-lingual children, as well as those adult humans who do not speak our language – we have no (accessible) self report to which we can appeal. But this does not mean that attributing pain to them is never justifiable. In such cases we have to rely on pain behaviour that is less explicit than the linguistic self-report. Few people doubt that dogs, one-year old humans and those adult humans who do not speak our language are capable of feeling pain. The less a being resembles us, both behaviourally and physiologically, the harder it is to infer what it feels like to be like such a being and therefore whether they are capable of thoughts or pains. It is at least in part for this reason that it is so difficult to determine whether a fetus is capable of feeling pain.

Derbyshire notes that the 'inability to derive any subjective report from the fetus means that all comments about the psychological experience of the fetus are drawn from inference'.[11] Later[12] he seems to criticise those who wish to infer the presence of fetal pain from behavioural and biological data. His concern may, in part, be that the particular behavioural and biological data used are insufficient to infer fetal pain – a matter that we shall pursue in the next section. But Derbyshire is claiming something more than this. He seems to bemoan the use of any standard other than self-report.[13] Although subjective report is often (although not always) more reliable evidence of pain than other behaviour, it would be unreasonable to accept it as the sole grounds for deciding whether to attribute pain, especially (although again not only) when considering beings incapable of subjective linguistic report.

DOES THE AVAILABLE DATA PROVIDE GOOD GROUNDS FOR INFERRING FETAL PAIN?

Derbyshire discusses evidence for the differences between the nervous system of the adult and the fetus. He draws on data from *anatomical* and *functional* studies.[14] He also alludes to *behavioural* evidence, albeit only briefly. He shows that there is maturation of the nervous system that accompanies the progression from the

[11] *Op. cit.*, Note 1, p. 5.
[12] *Op. cit.*, Note 1, pp. 21–22.
[13] *Op. cit.*, Note 1, p. 22.
[14] Such as electro-encephalography (EEG), evoked potentials (EP), positron emission tomography (PET) and biochemistry.

fetus to the adult. His conclusion is that 'while there are similarities between the fetal nervous system and the adult, it is important to remember that the real explosion of events in the cortex occurs postnatally between the third and sixth months of life', and that although 'the basic connections may develop early on in the fetus ... the subsequent neuronal development needed to create a sophisticated nervous system comes much later'.[15] In reviewing the relevant data, we shall demonstrate that although it bears out the claim that there is significant neurological development after birth, it fails to show that the late fetal and the postnatal development is the sort that is required for feeling pain. Consider the following analogy. Sexual maturation continues beyond the stage at which an adolescent becomes capable of reproduction. Pointing to ongoing sexual maturation – such as the further development of secondary sexual characteristics – would not demonstrate that reproductive capacities have yet to be attained. Similarly, humans become capable of feeling pain before full neurological maturity is reached. To point to neurological development in the late fetus or young child, by itself fails to show that such beings cannot feel pain. The balance of evidence, as we shall show, suggests that they can. We do not suggest that the evidence is unequivocal or conclusive – only that, considered as a whole, it tends to support rather than undermine the case for pain in fetuses from around 28 to 30 weeks gestational age.

Anatomy

Consider first the anatomical evidence. Derbyshire writes at great length about neuroanatomy. The evidence he cites, however, is intended only to establish the (now) uncontroversial claim that there is no 'pain centre'[16] but that it is rather a complex neural network that gives rise to pain. We certainly agree. However, in his subsequent discussion he does not say anything about at which stage in human development this neural network is sufficiently complex to support the experience of pain.

Providing a detailed account of neurodevelopmental milestones would be unhelpful given that the relevance of many of these to the question of pain is unclear. What can be said with

[15] *Op. cit.*, Note 1, p. 21.

[16] He says that his (lengthy) section entitled 'What is the underlying neurology of pain processing' is not 'entirely necessary for the flow of the argument' (*Op. cit.*, Note 1, p. 6).

some degree of confidence is that pain seems impossible prior to the formation, at 18 weeks gestation, of synaptic connections in the cortical plate (the precursor of the cortex). This is because at least some cortical function is a necessary condition for pain and there can be no cortical function in the absence of any cortical synaptic connections. Although the presence of such connections is a necessary condition for pain, it is not sufficient. It is certainly the case that the perception of pain as a result of external noxious stimuli would not be possible until a complete neuronal connection is established from peripheral nociceptors to cerebral cortex (via spinal cord, brain stem and thalamus). This occurs by about 28 to 30 weeks gestation. By itself, the presence of this connection between periphery and cortex does not prove the capacity for pain. However, in conjunction with other evidence that we shall discuss later, we shall argue that the presence of these anatomical pathways constitutes contributory evidence.

One possible objection to the importance of an intact connection between periphery and cortex is that all of these pathways do not become fully myelinated until later in development. Myelination is the process whereby an insulating layer of fatty tissue, called myelin, wraps around the neuronal structures. Lack of myelination should not, however, be taken as evidence of immaturity of the nociceptive nervous system. Even in adults, *nociceptive* (in contrast to other sensory) impulses are transmitted via *unmyelinated* C-fibres and only thinly myelinated Aδ-fibres. Furthermore, incomplete myelination simply implies a slower conduction velocity, and this may be offset by the shorter interneuronal distances found in the nervous system of fetuses and infants compared to those found in older children and adults. As a result, lack of myelination cannot be used as an argument to undermine the significance of the previously mentioned neuronal connections from periphery to cortex.

Function

So far we have considered the anatomical evidence. The presence of anatomical structures alone, however, is insufficient to demonstrate a capacity for pain. Anatomical structures, after all, are present in the dead. This would explain why Derbyshire wants to move beyond anatomy to consider the functioning of neuronal structures. There are a variety of measures of such functioning. These include electroencephelography (EEG),

recording of somatosensory evoked potentials (SEPs) and measures of cerebral metabolism with positron emission tomography (PET). Regrettably, these measures provide no direct evidence of the capacity for feeling pain. They provide only very general information about cerebral and other neurological functioning. Their relevance to the question of fetal pain is far from clear. Given this, one would be ill-advised to make inferences about fetal pain from this data in isolation from evidence that is more direct and more clearly relevant. We shall outline and evaluate the functional evidence to which Derbyshire refers, showing where it is irrelevant and where its relevance is questionable.

EEGs provide a record of the electrical activity of the brain and reflect the function of both cortical and subcortical structures. The EEG may be used to determine the presence of focal or widespread cerebral dysfunction, to establish the presence or absence of the electrical correlates of seizure activity and, relevant to our inquiry, to distinguish the waking from the sleeping state. EEGs do not provide information about what people are thinking or feeling. As such the EEG does not provide direct information about the feeling of pain. By this we mean that one cannot determine whether somebody is in pain merely by looking at the EEG. There are no 'pain patterns' discernable on an EEG. However, the EEG can provide data about a functional capacity that is required for feeling pain. This functional capacity is wakefulness.

Wakefulness, it must be stressed, is not to be confused with consciousness, at least in neurological parlance. Instead, it is a state of arousal that is to be contrasted with (the various stages of) sleep. Arousal is a state of the ascending arousal system in the brain stem and thalamus. It is not a state of the cerebral cortex. Where the ascending arousal system is connected to an intact functional cortex, its activities bring about changes in the cortex that are discernable clinically and electroencephalographically. While consciousness is supervenient on the function of the cortex, it is only possible in the wakeful state. In this sense, the brainstem and thalamus only support consciousness indirectly. Since arousal states – wakefulness and sleep – are states of the brain stem and thalamus (even though they usually have cortical consequences), and consciousness is a function of the cortex, wakefulness and consciousness are separable. One can be awake but not conscious, as where the ascending arousal system is in the awake mode, but the cortex is impaired in particular ways. For

instance, some patients in persistent vegetative states exhibit wakeful EEG patterns but are unconscious.[17]

Given (1) the distinction between wakefulness and consciousness and (2) that it is consciousness rather than wakefulness to which pain is most directly linked, what is the relevance of EEG as a measure of wakefulness to the question of pain? Presumably it is this. Whereas wakefulness is not sufficient for the presence of consciousness, it seems reasonable to assume that consciousness is not possible in the absence of wakefulness. Although sleeping people are sometimes responsive to their environment – that is to say, they can react to stimuli – they are not aware or conscious. If this assumption is correct, then a being that lacks the capacity for wakefulness will also lack the capacity for consciousness. Thus EEGs provide evidence for a condition – wakefulness – without which consciousness (and hence pain) is not possible, even though they do not provide evidence of consciousness itself.

Although there are intermittent bursts of (sleep pattern) electroencephalographic activity in fetuses as young as 20 weeks gestation, it is only around 30 weeks that EEGs reveal sleep-wake cycles. In other words, it is only around 30 weeks that the first wakeful states are discernable. At this early stage, it must be emphasized, the EEG patterns for wakefulness and sleep are quite different from those of the adult. In the first few months of post-natal life the fetal EEG pattern gradually gives way to one that much more closely resembles the adult pattern, even though maturation of the EEG continues throughout the first year of life and, to a lesser extent throughout childhood and adolescence.

There are at least two explanations for the relatively large difference between fetal and adult EEGs. One, implicit in Derbyshire's argument, is that the sort of wakefulness needed for consciousness (and hence pain) has not yet developed. The other is that the electroencephalographic differences are a result of the general immaturity (and thus difference) of the fetal nervous system, but suggest nothing about the absence of the neurological function necessary for pain. In this view, fetal wakefulness may produce a different EEG, but may still facilitate consciousness. How does one choose between these possible explanations? In support of the latter, one might note that fetal and infant *sleep* EEGs are as different from adult sleep EEGs, as fetal and infant *wakeful* EEGs are different from the adult variety.

[17] Multi-Society Task Force on PVS. Medical Aspects of the Persistent Vegetative State. *New England Journal of Medicine* 1994; 330, 21: 1499–1508.

If it would be odd to suggest that premature infants of around 30 weeks gestation are not sleeping when clinically and electroencephalographically they appear to be, would it not also be odd to suggest that when such infants are both clinically and electroencephelographically awake (and conscious) that they are not? Now it might be suggested that since fetal sleep is clinically more similar to adult sleep than fetal wakefulness is to adult wakefulness, we can be more sure of fetal sleep than we can be of fetal wakefulness. To the extent that this is so, all it shows is that we have less reason *for* thinking that fetuses have adult-like wakeful states. It does not show that they do *not* have the sorts of wakeful states necessary for consciousness and pain. Were there no other evidence for fetal pain, we agree that the EEG data would provide no evidence for it. However, there is other evidence – most especially the anatomical, which we have discussed and, more compellingly, the behavioural, to which we shall still give attention.

Before we turn to that, however, we shall first consider additional functional data to which Stuart Derbyshire refers. In his discussion of EEGs he makes reference to evoked potentials. An evoked potential is the component of the EEG that can be measured in response to a significant sensory stimulus. Thus the evoked potential recorded following a strong visual stimulus is known as the visual evoked potential and the evoked potential following a significant somatic sensory stimulus is known as the somatosensory evoked potential (SEP). Stuart Derbyshire states that 'although it is true that the early negative evoked potentials begin to develop from 25 weeks, the differences between the patterns of the adult evoked response and the developing fetus and newborn are striking'.[18]

The appeal to SEP data is mystifying, as these data bear no relevance to the question of nociception (and resulting pain). First, the SEP does not provide any information about those structures involved in nociception. It is the slow (unmyelinated) C- and (thinly myelinated) Aδ-fibres that transmit nociceptive impulses. The SEP, by contrast, is a measure of conduction in the *fastest* conducting (thickly myelinated) fibres. Second, the SEP is reported in terms of its latency (that is, the time delay between the sensory stimulus and the recording of the evoked potential) and not its amplitude (size) or morphology (shape), as Derbyshire seems to think, given the diagrams he uses to illustrate his case. While it is true that the SEP latency is

[18] *Op. cit.*, Note 1, p. 18.

prolonged in the fetus and neonate relative to the adult and that this is attributable to immature myelination, this is not an immaturity of the *nociceptive* system.

Consider next, positron emission tomography (PET), a non-invasive technique that may be used to investigate cerebral function. PET scanning with radioactive glucose provides a measure of cerebral glucose utilization, which indicates cerebral metabolism as a marker for cerebral activity. Derbyshire makes brief reference to the study by Harry Chugani and Michael Phelps of developmental changes in cerebral function as determined by radioactive glucose PET.[19] According to him, this study shows that 'the neuronal function of the cerebral cortex, especially the somatosensory cortex, the prefrontal cortex and the anterior cingulate cortex, increased by a third from birth to 18 months'. A closer look at the findings of this study suggests a more refined interpretation. It does indeed demonstrate an increase in glucose utilization in the somatosensory cortex over the first 18 months of age. However, the exponential increase takes place over the first 3–4 months with only an incremental increase over the ensuing 14 months. Like the EEG evidence, this suggests a massive increase in cerebral function in the first few months of post-term life. However, it is again unclear what conclusions about fetal pain can be drawn from this. The PET data tells us nothing about whether the sorts of developments are ones necessary for feeling pain or whether the functional capacities for pain are already present (to a significant degree) in late gestation or by birth. One reason to think that there is sufficient cerebral function for at least some feeling of pain is that the PET evidence of activity in the anterior cingulate cortex, an area relevant to the feeling of pain, is comparable to the changes in the primary and association visual cortex. Although there is maturation of the visual capacities, they are present in some form even at birth, before PET shows the largest post-natal increase in activities of the visual cortex. If we think that neonates are capable of having visual experiences (even if only of a rudimentary form) then we should think that they are capable of at least some kind of painful experience.

So far, we have discussed Derbyshire's treatment of neuro-anatomical evidence as well as a variety of functional measures of the nervous system. Some investigators have suggested an

[19] H.T. Chugani, M.E. Phelps. Maturational Changes in Cerebral Function in Infants Determined by ^{18}FDG Positron Emission Tomography. *Science* 1986; 231, 4740: 840–843.

alternative indirect approach to assessing the presence of nociception and pain in beings incapable of reporting their mental states. This approach involves the measurement of hormonal changes that are associated with pain in those beings who are capable of reporting such experiences. Cortisol and β-endorphin are two hormones that are released in response to noxious stimuli with the effect of minimizing the pain. Cortisol acts as an anti-inflammatory agent to counteract the noxious stimulus and β-endorphin (an endogenous opiate-like substance) acts to dampen the painful feeling. To understand the significance of changes in the concentration of these hormones, it is necessary to appreciate their temporal relationship to pain. Following the administration of a noxious stimulus, the concentrations of these hormones rise and the intensity of the painful feeling is reduced. The result is that a correlation emerges between elevated levels of these hormones and the relief from pain. The important point, therefore, is that these hormones *rise* in response to a painful stimulus and that *elevated* levels then correlate with the alleviation of pain.

Xenophon Giannakoulopoulos and colleagues have investigated whether the fetus mounts a hormonal stress response in reaction to a stimulus that would be painful in an adult. They measured fetal cortisol and β-endorphin production in response to invasive intrahepatic vein transfusion and, as a control, to non-invasive umbilical vein transfusion.

The intrahepatic vein transfusion was accompanied by vigorous body and breathing movements, the sort of behaviour that would be expected to accompany pain. They demonstrated a rise in cortisol and β-endorphin levels following the invasive transfusion. These biochemical and behavioural changes were not observed following the non-invasive transfusion.[20]

As the authors of this study caution, 'a hormonal response cannot be equated with the perception of pain'[21] and therefore, their findings do not provide *definitive* evidence of pain in the fetus. However, their study, they correctly conclude, 'shows that as with neonates, the fetus mounts a similar hormonal response to that which would be mounted by older children and adults to stimuli that they would find painful'.[22] Coupled with the

[20] X. Giannakoulopoulos, W. Sepulveda, P. Kourtis, V. Glover, N. M. Fisk. Fetal Plasma Cortisol and Beta-endorphin Response to Intrauterine Needling. *Lancet* 1994; 344, 8915: 77–81.

[21] *Ibid.*

[22] *Ibid.*

observed behavioural response, this biochemical data speaks more in favour of fetal pain than against it.

Derbyshire dismisses this important evidence altogether too glibly. He claims that 'the increases in cortisol and β-endorphin responses recorded were much smaller than those usually seen in adult populations'.[23] In defence of this claim he cites a study of fear conditioning in rats.[24] Given that this study provides no measurements of cortisol or β-endorphin, it is hard to understand how it can support his claim. Let us assume, however, that his claim is nonetheless true (although unsatisfactorily referenced). It would not follow that fetuses lack the capacity for pain. At the most, it would be suggestive of an underdeveloped, but not absent, capacity for feeling pain.

Another response he has to the hormonal evidence provided by Giannakoulopoulos and colleagues is to claim that 'high cortisol concentrations in adults following surgery can be associated with *lower* pain ratings'.[25] This finding is not unexpected, but to interpret it as counter-evidence, as Derbyshire does, involves ignoring the dynamic relationship between these hormones and the feeling of pain. As we have previously explained, the presence of pain stimulates a *rise* in the concentration of cortisol and β-endorphin. The resulting *elevated* concentration of these hormones is associated with *relief* from pain.

Behaviour

So far, we have considered evidence from anatomy and from the function of individual systems. Next we wish to consider behavioural evidence. Behaviour integrates many individual functional systems. At least in its crudest forms, it has the added advantage of not requiring any specialized scientific knowledge and as such, is accessible to any intelligent observer. It is, in fact, usually behaviour that leads to the attribution of mental states to others. There are, of course, problems with behavioural evidence and we shall consider these in due course.

Derbyshire makes only brief reference to behavioural data. He acknowledges that there are premature infants that show

[23] *Op. cit.*, Note 1, p. 20.

[24] J.W. Rudy, P. Morledge. Ontogeny of Contextual Fear Conditioning in Rats: Implications for Consolidation, Infantile Amnesia and Hippocampal System Function. *Behavioural Neuroscience* 1994; 108, 2: 227–234.

[25] *Op. cit.*, Note 1, pp. 20–21.

coordinated facial actions in response to noxious stimuli that are not manifest in infants of a younger gestational age.[26] However, his cursory reference to this issue grossly understates the importance of this data and the insights it provides into the question of fetal and neonatal pain. For example, Kenneth Craig *et al*[27] used the Neonatal Facial Coding System (NFCS) to evaluate the response of *preterm* neonates to noxious and non-noxious stimuli. Neonates of varying ages were videotaped before, during and after a heel swab and lancing procedure. The heel swab provides a non-noxious stimulus, whereas the heel lance is a noxious stimulus that would be painful in beings with a mature nervous system. In response to the lance but not the swab, infants older than 28 weeks gestation were found to exhibit a distinct set of facial movements that are also characteristic of term infants and adults subject to painful stimuli. These facial movements include brow lowering, eyes squeezed shut, deepening of the nasolabial furrow, open lips and mouth and a taut, cupped tongue.[28] The authors of this study also observed that these facial movements varied depending on whether the premature infant was asleep or awake at the time of the lancing. Given that wakefulness facilitates the experience of pain, this observation is noteworthy. In contrast to these striking observations about humans of 28 weeks gestational age, infants of 25–27 weeks gestation did not display a response sufficiently different from baseline.[29]

It is, of course, possible, and skeptics of fetal pain might well rush to say, that the facial movements observed in the older preterm neonates are mere reflexes and do not reflect any unpleasant mental state. There is no way decisively to lay such doubts to rest. Nevertheless, the complex and coordinated nature of this behaviour makes it harder to dismiss as a mere reflex. Given that, at that young age, the behavioural repertoire is restricted, not least by limited energy resources, and that facial movements may be the best behavioural insight we have into the fetal mind, it would be rash not to give due weight to the

[26] *Op. cit.*, Note 1, p. 18.

[27] K.D. Craig, M.F. Whitfield, R.V. Grunau, J. Linton, H.D. Hadjistavropoulos. Pain in the Preterm Neonate: Behavioural and Physiological Indices. *Pain* 1993; 52, 3: 287–299.

[28] *Ibid.*

[29] The authors of this study caution that the failure to detect these behavioural changes in the younger age group might be an artifact of the small number of infants studied.

evidence of distinctive facial movements. To expect greater behavioural responses would be like denying that a one-year old is in pain because it does not say so or because it does not get up and walk away from the painful stimulus. Given that one-year olds are not capable of such behaviour we cannot expect it of them, but it does not follow that they lack the underlying unpleasant mental state which we call pain.

Reflexive behaviour is that which does not result from a conscious mind. Thus withdrawing from a noxious stimulus is reflexive if it is not a result of a painful feeling. It is not a reflex if it does result from such a feeling. From this, it should not be concluded that the presence of a reflex and the presence of pain are mutually incompatible. Spinal reflexes, for example, can result in the withdrawal of a limb from the source of a noxious stimulus even before the pain-causing impulse has reached the cortex. The withdrawal movement is itself reflex. It does not follow that there is not an accompanying painful sensation, even if that sensation is not the cause of the reflex but rather occurs milliseconds after it. Distinguishing between those behaviours that are both reflexive *and* unaccompanied by painful experiences from behaviour, whether reflexive or not, that *is* accompanied by pain, can be attempted only by inference. As we noted earlier, one can never gain direct access to the consciousness of another. It can only be inferred from (or denied on the basis of) publicly observable phenomena – such as behaviour, as well as anatomical and functional evidence which we have considered.

Consciousness

Derbyshire claims that '[d]espite the importance of providing evidence for the conscious appreciation of pain, the fetal and neonatal literature largely ignores this issue'.[30] Were it true that this literature ignored such evidence, it would be a damning criticism of a literature that sought to determine whether fetuses and neonates are capable of feeling pain. The problem, however, is that Derbyshire seems to confuse the distinction between 'indirect evidence for the conscious experience of pain' and 'direct evidence or conclusive proof of the conscious experience of pain'. The latter, we have indicated is impossible, yet it is exactly this that he seems to want in some places. Thus he says that 'too often … neurological and behavioural measures [are]

[30] *Op. cit.*, Note 1, p. 22.

suggested as a proxy for psychological experience'.[31] Where authors appropriately caution that the evidence they provide for pain is not to be equated with pain, he criticizes them.[32] Yet, he seems content with a standard of evidence that falls short of the psychological experience of pain. Thus he seems to hold up a subjective report of pain as an adequate standard.[33] As we have indicated before, this is simply another form of indirect evidence, and although usually (but not always) very compelling, cannot be the only standard deemed acceptable, especially when dealing with beings incapable of self report or whose self report we are unable to understand.

It is curious that when Derbyshire himself considers the question of consciousness, he is content with evidence that is as indirect but also less robust than much of the evidence for fetal pain that has been considered until now. We agree with his uncontroversial claim that it is mistaken to think of consciousness in simple binary terms as being either present or absent. Instead it should be thought of as something that may gradually appear or may exist to varying extents. The analogy of a dimmer switch, which he borrows from Susan Greenfield, is a helpful one. Where he is mistaken, we believe, is in the way he maps the development of consciousness onto the human developmental continuum. On his view, consciousness and thus pain only begin around 12 months post-gestational age.

It is his odd view of consciousness that accounts for this mapping. First, he seems to think that consciousness is not attributable simply to biological developments, but also to the social interaction of the developing being. Now, we do not doubt that children raised in isolation from other beings would develop differently and that their minds would be affected by that unfortunate method of rearing. Derbyshire's claim is much stronger than this, however. He thinks that 'if left entirely alone, a human being might operate in an unconscious computational manner'.[34] This is an exceedingly odd view on which to pin confident conclusions about the absence of fetal pain. It is not unreasonable to think that children who develop without contact with others would not acquire any particular language and thus might lack sophisticated concepts attendant upon a developing use of language. What reason is there to think, however, that the

[31] *Op. cit.*, Note 1, p. 21.
[32] *Op. cit.*, Note 1, p. 22.
[33] *Ibid.*
[34] *Op. cit.*, Note 1, p. 23.

most primitive contents of consciousness, such as pain, would be absent in a child that grew up without any interactions with others? If Derbyshire is correct, then any child reared in this way could never be capable of suffering from the experience. Accordingly, the moral case against such rearing would be reduced and, for hedonists, eliminated. Yet Derbyshire himself is 'thankful' that there are 'no documented cases of children being reared in such *obscene* circumstances' (our emphasis).[35]

A second, and related, problem with Derbyshire's view of consciousness is that it is far too narrow. He thinks that 'conceptual and symbolic abilities, along with purposive, directed awareness are ... core components of consciousness'.[36] This understanding of consciousness is not anything like the common-sense one. Most people would accept that some beings – such as four- or eight-month old children, or some animals – are conscious, aware, capable of *feeling*, even though they may lack conceptual and symbolic abilities and even if they are incapable of adult-like intentionality. Although common views should not be accepted unreflectively, Derbyshire has provided no arguments for rejecting the common-sense view about consciousness.

He may be aware of this. This would explain why he is so willing to concede that his judgment about the timing of the 'dimmer switch' of consciousness 'has been made entirely by the current author and no doubt others will feel that the switch should move more or less quickly'.[37] He goes so far as to acknowledge that his conclusion about consciousness 'does not rule out the possibility of a rudimentary, or impoverished, awareness at an earlier stage'.[38] It is exactly this sort of awareness or consciousness that leads many people to think that late fetuses and neonates are capable of feeling (at least rudimentary) pain. But Derbyshire wants to deny that we can use the term 'pain' to refer to anything fetuses experience. This is because of how he understands pain. As we indicated earlier, he thinks that emotions are an essential component of pain. We showed then, why that interpretation of 'pain' is problematic. If one abandons that misguided understanding of 'pain', it is quite possible to concede – and Derbyshire would then seem required to concede – that late fetuses are capable of pain. The definition of pain is, as

[35] *Ibid.*
[36] *Op. cit.*, Note 1, p. 24.
[37] *Op. cit.*, Note 1, p. 26.
[38] *Op. cit.*, Note 1, p. 27.

he says, crucial to any inquiry into whether fetuses can experience pain. Ironically, it is his faulty view of pain that leads him to the conclusion that they cannot have such an experience.

CONCLUSION

Common sense, derived in part from observing neonates, suggests that humans of late gestational and early post-term age are capable of painful experiences. Derbyshire gives the impression that the weight of scientific evidence speaks against the possibility of fetal pain. Most authors writing on the subject disagree with him. The dominant scientific opinion reinforces common sense. There is non-negligible evidence to support the claim that from around 28 to 30 weeks gestational age, fetuses are capable of feeling pain. By that stage, for instance, the neuronal connections from the periphery to cerebral cortex are in place, electroencelphalographically distinctive periods of wakefulness are evident, hormonal responses that alleviate pain are mounted to noxious stimuli, and behaviours associated with pain are consistently elicited in response to such stimuli. None of this makes it unequivocally clear that fetuses *do* feel pain. Nevertheless, this evidence does lend more support to the view that they do than to the view that they do not.

In the face of this, the burden of proof is surely on those who would contest the possibility of fetal pain. Derbyshire fails to meet this burden. His SEP data, we have shown, is irrelevant, and the relevance of the PET data is far from clear. He regularly refers to the extensive neurological maturation that takes place in the first year of (post-natal) life, but fails to show that the developments that take place are ones that make pain possible. He assumes highly unusual interpretations of 'pain' and 'consciousness' in order to generate his conclusions.

It seems to us that, at least on the available evidence, fetuses are capable of feeling pain. Given the problem of fetal minds (as an instance of the other minds problem), it is impossible to determine what it feels like to be a fetus that is having an unpleasant experience as a result of a noxious stimulus. Perhaps, because of the immaturity of the nervous system it does not feel *as bad* as it would for us. On the other hand, perhaps it feels worse. Children, after all, sometimes react to pain more vigorously than adults. Although it is hard to say whether it is simply the reaction that is greater or also the qualitative feel of the experience, the latter possibility cannot be discounted. If so, then late fetuses might be more easily pained than adults.

No moral conclusions follow automatically from assenting to the possibility of pain in late fetuses. Many people, ourselves included, think that pain is morally significant in the sense that, all things being equal, it ought to be avoided. But clearly not all things are equal in most situations in which one is considering, for example, a therapeutic procedure that will cause a fetus pain but also benefit it. Administering analgesia might have damaging side-effects on the developing fetus. It has not been our purpose to say what should be done in such circumstances. All we have sought to do is show that those who would eliminate or alleviate the problem by denying the possibility of fetal pain have failed to make their case.[39]

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[39] We should like to thank Frank Drislane for his comments.