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Kathy Wilkes, Teleology, and the Explanation of Behaviour

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Kathy Wilkes contributed to two books on Goal-directed Behaviour and Modelling the Mind based on interdisciplinary graduate classes at Oxford during the 1980s. In this article, I assess her contributions to those discussions. She championed the school of philosophers who prefer problem dissolution to problem-solution. She also addressed the problem of realism in psychology. But the contribution that has turned out to be most relevant to subsequent work was her idea that in modelling the mind, we might need to "use as structural elements synthetic cells, or things that behaved very like neurones." I show how this idea has been developed in my own recent work with zoologist and neuroscientist, Raymond Noble, to become a possible physiological basis for the ability of organisms to choose between alternative actions, and so become active agents. I consider that this insight became her seminal contribution in this field.

Keywords: *Teleology; goal-directed behaviour; modelling the mind; agency*.

1. *Introduction*

It was a great privilege for me to give the opening lecture at the Dubrovnik Inter-University Centre symposium honouring Kathy Wilkes.[1]

* I thank Anthony Kenny, Alan Montefiore, Andrew Packard and Raymond Noble for many discussions that have contributed to my thinking about this subject. Andrew Packard was particularly helpful in drawing my attention to multiple aspects of the work of JZ Young.

[1] This Conference was held at the Inter-University Centre, Dubrovnik, 29th April 2022.

This article is closely based on that lecture. My main credentials for doing so arise from seminars held in Balliol College Oxford during the 1980s on the explanation of animal and human behaviour. They arose from a long-standing collaboration between Alan Montefiore, a philosopher, and me, a biological and medical scientist.

We both edited the book *Goals, No Goals and Own Goals* (Montefiore and Noble 1989) that resulted from the seminars with Kathy Wilkes, and David McFarland, an ethologist, as co-organisers. Alan's description[2] of the way the debate developed is correct when he says that there was mostly an axis between Alan and me on the one hand and one between Kathy and David on the other. This outcome is itself significant. The divide was not really one between scientists and philosophers, and it shows also that scientists themselves are not neutral with respect to philosophical concepts concerning animal and human behaviour. There were two other contributors: Shawn Lockery, now a Professor of Neuroscience in the USA, and Dan Dennett, who contributed an article but did not take part in the seminars.

A further professional link with Kathy arose from the book she edited with Bill Newton-Smith, *Modelling the Mind* (Said, Newton-Smith et al. 1990). We both contributed chapters to that book. Kathy herself wrote the chapter (Wilkes 1990) that gave the book its title, while I followed some of the arguments in the *Goals* book, with a chapter on Biological Explanation and Intentional Behaviour (Noble 1990).

2. *The philosophical and scientific background*

My own interest and involvement in these seminars arose from a published interaction in 1967 with the Canadian philosopher Charles Taylor, following his book *The Explanation of Behaviour* (Taylor 1964), based on his doctoral thesis at the University of Oxford. I was introduced to the book by Anthony Kenny, who was working on related problems (Kenny 1969), and with whom I have interacted ever since on issues to do with mind, will and action. Arising out of our discussions he encouraged me to write a critique of Taylor's book, which was published in *Analysis* (Noble 1967), where I argued that Taylor's defence of teleological explanation was incorrect since it seemed to require that a difference in state at one (higher) level should not necessarily have a correlate at another (lower) level. On this view, there would be a gap in the mapping. As a physiologist I found the idea of such a gap difficult to accept.

Taylor did however reply with a very interesting argument (Taylor 1967). This was that, while there could not be a physical gap it might nevertheless be the case that, after studying a whole series of correlations between, say, behaviour and neural states, only the higher level of behaviour might show a pattern that could count as an explanation.

[2] Personal communication.

Specifically, if the behaviour states are B1, B2, B3, … and the neural event states E1, E2, E3…. the E states might be disordered with respect to explaining the behaviour whereas the B states might offer a ready explanation. I found this a very interesting reply and countered that the consequence was that the issue of the validity of teleological explanations became a conceptual issue, not an empirical one (Noble 1967). I believe that was an important clarification, and that it is still valid. The clarification will reappear later in this article. But I also think the debates have moved on very significantly since 1967.

The seminars in Balliol in which Kathy was such a major contributor formed an important stage in that development. During those seminars I was still developing the ideas on goal-directed behaviour that eventually became expressed in my more recent books *The Music of Life* (Noble 2006) and *Dance to the Tune of Life* (Noble 2016) and even more recent articles (Noble 2017, Noble and Noble 2017, Noble and Noble 2018, Noble, Tasaki et al. 2019, Noble and Noble 2021). Those publications describe the ways in which teleological behaviour naturally occurs and develops during the evolutionary process. They also show how such behaviour itself contributes to evolution and so gives evolution itself a kind of directionality. Most recently, these include a paper on purpose in physiology appearing in the *Biological Journal of the Linnean Society* (Noble and Noble 2022). I will return to what led to those books and articles at the end of this article, by showing how one of Kathy's contributions formed a key element in those developments.

However, I was far from ready during the Balliol seminars in the 1980s to give expression to those ideas at that time. It is only in retrospect that I can see the roots of my development. That is unfortunate from one point of view. If I had been able to express the ideas and marshal the biological experimental evidence more forcefully in the 1980s perhaps the debates in which Kathy was involved would have taken a different turn. But the flip side of this coin is that I remain deeply grateful to Kathy herself, and to the other participants for a sustained and deeply stimulating series of seminars that did much to clarify my own thinking. I would have loved to try the more recent ideas out on Kathy, particularly because, as I will show, I believe they answer one of the key questions she contributed to the debates of the *Goals* book.

3. *Reactions to the book*

Soon after publication of the *Goals* book in 1989, I sent a copy to the distinguished zoologist and expert on the intelligent behaviour of the cephalopods, JZ Young. I had been taught medical sciences in UCL where he was the professor of Anatomy and a world-renowned expert on the learning and behavioural repertoires of the octopus. I suspect I learnt more philosophy from him than anatomy! So, it seemed a good idea to get his reactions. He wrote to me afterwards to say that he had enjoyed reading it, several times in fact. But he wasn't exactly com-

plimentary as far as my own contributions were concerned (still the critical professor of his former student!) and he didn't seem to go much for Alan's contributions either. So much for Alan's and my side of the debate! But JZ Young was *much* more complimentary about Kathy's chapters which he thought were clear and, in his view, largely correct.

Why was JZ Young sympathetic to our debate at all, and to Kathy's contributions in particular, even though critical of some of what Alan and I wrote? To understand that we need to recall that JZ Young was the discoverer of the giant nerve axon in the squid (Young 1936, Young 1938, Keynes 2005) that enables it to trigger a form of jet propulsion (Packard 1969), in turn enabling it to successfully flee predators. This was the giant nerve on which Alan Hodgkin and Andrew Huxley worked to obtain the experimental data on which they constructed their famous mathematical model (Hodgkin and Huxley 1952) of the nerve impulse and its dependence on sodium and potassium ion channels in the axon membrane. It was an important prediction of their model that large nerve axons would conduct faster than small ones, as they were known to do (Pumphrey and Young 1938), though it should be added that this was not the reason for their choice of nerve to work on. The squid axon was simply large enough for them to insert their recording and controlling electrodes. When Hodgkin and Huxley were awarded the Nobel Prize for this work in 1963 Young was known to have commented that this was a bit like awarding a prize to the typewriter rather than to the book author. I don't think he meant to denigrate Hodgkin and Huxley's achievement. Rather he was pointing out that the reason for the existence of the giant axon, its purpose, was the evolutionary imperative to generate a rapid response to predators. Furthermore, the giant axon was not an evolutionary development found in all cephalopods. It is not found in octopods. The efficiency of the jet propulsion mechanism depends therefore more on the functional anatomy of the whole system ensuring simultaneous contraction, not just the speed of nerve conduction. He saw that this was the emergence, during evolution, of a goal directed mechanism. Every aspect of the anatomy and physiology of the cephalopods was fine-tuned in ways that endowed the organisms with a rapid escape mechanism.

He therefore regarded the mathematical analysis of the mechanism of the nerve impulse to be too low a level to explain the goal-directedness of the behaviour, with which I am sure Hodgkin and Huxley would have agreed. So, he was certainly sympathetic to the general purpose of the *Goals* book. Low-level explanations don't work, and for precisely the reason that emerged from my interaction with Charles Taylor. Incidentally, there is a very useful "Celebration of JZ Young" by Andrew Packard and Fabio DeSio published in *Physiology News* in 2010 (Packard and DeSio 2010). I see JZ Young as the embodiment of the tension between purposive and reductive accounts of biology, a view that is reinforced by this quotation from one of his collaborators, Brian Boycott:

> there is, in most of JZ's scientific design and output, a tension between his desire to investigate integrative functions of organs and systems as a whole and the practical constraint that to do this requires the reduction of a system to an experimentally manageable and interpretable entity. (cited by Packard and DeSio 2010)

So, why did JZ Young think more of what Kathy wrote than what Alan and I wrote? I suspect that he was nevertheless suspicious of teleological ways of speaking about animal behaviour. Most biological scientists were sceptical of that approach in the mid-twentieth century: "Teleology is like a mistress to a biologist: he cannot live without her but he's unwilling to be seen with her in public."[3] Some biologists even invented the word teleonomy (Pittendrigh 1958) to refer to the biological processes involved without committing to whether or not an organism is an active agent.

I now think that there was no need to invent a separate word. Organisms are definitively purposeful agents (Noble and Noble 2022). But this is not the place to justify that point. Here it suffices to say that it is a tribute to Kathy's work that such a noted expert on animal behaviour as JZ Young thought highly of it. So, what were the main points of her contributions to the *Goals* book?

4. *Kathy's contributions*

She wrote two chapters in the book, and she explains her philosophical position most clearly at the end of the second (210). She wrote:

> Our discussions of these issues over several years have left me more confused at the end than I was at the beginning.

(Surprise, surprise!) …. And then continues

> I have suggested that many of the problems might be pseudo-problems—to be dissolved rather than solved; certainly I align myself with the 'theft over honest toil' school of philosophers who prefer problem dissolution to problem-solution.

Nevertheless, she identified

> one question [that] has emerged as indissoluble, crucial and critical: what counts as 'realism' in psychology? This needs serious thought, which would and should enrich and deepen the ongoing examination of realism in the physical sciences.

On this, she was surely right. There is a veritable flood of books now on *What is Real* (Becker 2018), *The Matter with Things* (McGilchrist 2021) and similar titles, to which I would add Hilary Lawson's groundbreaking analysis of "reality" in his book *Closure: A Story of Everything* (Lawson 2001). As I will show at the end of the paper, there are good reasons for this explosion: there is a groundswell of opinion in opposition to the confidently-expressed materialist (realist) certainties of the mid-20th century.

[3] Attributed to J. B. S. Haldane.

Kathy herself was more concerned with what the common "man in the street" might want as explanations. She wrote:

> not all explanations are causal explanations…if one job of explanation is to remove puzzlement, then evidently people can be puzzled by well-nigh anything.

Here she is talking very much in the tradition of philosophy paying attention to the language of the man in the street. I found it helpful that she kept bringing us back to the pragmatic uses of philosophy. This aspect of her work was, I suspect, at one with her engagement with the problems of the world, notably here in the immense contributions she made to the cultural life of Dubrovnik, and of course the amazing contribution she made to intellectual life in Prague. There are others at this symposium who know far more than me about that aspect of Kathy. My knowledge is second-hand, largely through two other Oxford philosophers, Bill Newton-Smith and Anthony Kenny, who both lectured to the under-cover seminars held in Prague. In a recent email to me, Kenny writes:

> When the dissident Czech philosophers first made contact with Western Universities, only Oxford made a positive response, and that was due to Kathy who was then secretary to the Philosophy sub-faculty. I think that she, Bill Newton Smith and I were the only people to be arrested for talking to the Tomin group—but it was she who went on lecturing after being arrested. Nancy and I were just taken off to the police station and extradited early the following day (to the surprise of the German frontier police who assumed we were drug smugglers).

Time and again, Kathy was concerned more with pragmatics than with grand theory, of which it seems to me she was highly sceptical. By contrast, Alan and I must have seemed to her to be too strongly concerned with conceptual theory.

In this vein, here is what she thought about whether science could find correlates of intentions:

> [Common sense psychology] needn't bother about whether these intentions are explicit and real, or tacit and hence not really 'there' in any strong sense. In other words, when we ascribe intentions to an agent, we are not usually …committing ourselves to the existence of a physical correlate *to that very intention*.

I suspect that this is why she and David McFarland often joined forces. David, as an ethologist, was very sceptical of whether intentions matter at all! If I understood him correctly, these were feelings we experience but which need not have any influence on how we actually behave.

Kathy herself was not, of course, a Watsonian. She writes:

> Extreme (Watsonian) behaviourism failed because there is so much that it just cannot explain. This is scarcely surprising; it always was a priori implausible that so simple-minded a theory could account for the most complex system we know. But it rejected all 'mental' terms; here I am only examining the possibility that a scientific theory might do without one of them: intentions.

A strong feature of Kathy's contributions to the debate was her continual insistence on clarifying what we mean by an explanation:

> What sort of 'accounting for' [do] we want 'the traditional goal concept' to provide… In this book we find free use of 'causes', 'is responsible for', 'explains' 'continually guides' and more besides. This leads into the rather more specific question of whether explanation via intentions, or goal representations, is a species of causal explanation. And that forces one to ask just what is needed if A is to be 'the cause' of B: 'being a cause', 'serving to explain', and 'being responsible for' are not synonymous expressions. (195)

On this issue, Kathy and I were in agreement. We both thought that, whatever intentions might be, they could not be the cause of behaviour in the same kind of way in which nerve action potentials cause muscle movement. I think she was on exactly the right lines in insisting that, at the least, different concepts of cause need to be invoked. She wrote:

> Thus, although endorsing Noble's claim (97) that 'within an intentional context a "machine" description of what happens fails to make reference to the most significant facts' I would want to explain why this must be so by linking 'significance' to the precise characterisation of the explanandum—to the puzzlement of the inquirer. I find it increasingly difficult to find any real-life cases where there is genuine, honest-to-goodness' 'rivalry' at all between intensional and non-intensional explanations of what is indeed the one and the same explanandum.

These arguments all form part of her "attempt to underline the differences between common sense, and scientific, explanation" (198).

I now find myself in total agreement with her arguments on this issue, even to the extent that my own recent publications not only elaborate on why intentions cannot be causes in the same way as nerve impulses can be, but also that, even within purely biological levels of organisation, the forms of cause between different levels can be quite different. As an example, causation from the genetic level is mediated by templates (gene sequences) not by specific molecular interactions (Noble, Tasaki et al. 2019).

These direct quotes from her work for the *Goals* book will, I hope, give readers a flavour, at least, of what Kathy contributed to the seminars and the book. Fortunately, the book itself has been republished as an e-book by the publisher (Montefiore and Noble 2021), so interested readers can readily explore further if they wish.

Now I turn to her contributions to *Modelling the Mind*. I am not surprised that it became the overall title for that book. For, by contrast with the *Goals* book, where she says herself that she was left more confused, her chapter in the 1990 book represents Kathy in full flow as the insightful philosopher she clearly was.

She begins by clearly stating that we should never talk about *the* model. Even in physics, we need multiple models, even incompatible models, for models, like metaphors, illuminate different aspects of reality, and they can be useful even when incompatible. As Lakoff and Johnson famously said in their 1980 book, *Metaphors We Live By* (La-

koff and Johnson 1980), metaphors can have good and bad ranges of applicability. What works for the micro-level in physics, i.e. quantum mechanics, does not cover what the theory of General Relativity covers and vice versa. She writes:

> The danger, as far as psychology is concerned, comes when we switch from indefinite to definite article. (63)

Yet, particular models do become dominant:

> Hume's metaphor of the mind as an inner theatre was never more than that, a metaphor (as he was the first to insist), even though it became deeply compelling to treat it *as if* the mind were indeed really like that. (64)

So, if we "cannot think of minds as inner theatres, inspected by an unblinking inner eye, any longer" just what do we think the mind might be, or what is it to be mental?

There is then a careful analysis of the computer model of mental processes. She points to the danger that

> there is a real possibility that psychological explanations might 'bottom out' in hardware structure and function long before we have learned anything from the computer metaphor; in fact, that the really interesting work may come rather from one or other of the neurosciences than from simulation exercises. (73)

It is at this point that I encountered a fascinating speculation:

> It may be that if we were to construct a computer with capacities close to those of the human brain, we would have to use as structural elements synthetic cells, or things that behaved very like neurones—with, say, action potentials, graded potentials, 'synaptic' modifiability, 'dendritic' growth, etc. (73–4)

This paragraph is tantalisingly close to where my own thinking has gone recently. Specifically, I have speculated that, in order to access the kinds of molecular stochasticity in real brains, we might have to make "computers" using water rather than silicon. The argument is simply that novelty, creativity, in organisms may depend on precisely what kind of stochasticity is harnessed by living organisms.

My overall conclusion from re-reading Kathy's work after about 30 years have passed, is that her contribution to *Modelling the Mind* is the better example of her thinking. She was in full control of what she was writing, instead of being "more confused at the end than I was at the beginning".

I suspect that one of the reasons for that conclusion on her part is a fault of my own as the biologist in the debates. Perhaps something was missing from what I, as the physiologist, should have contributed.

5. *What was missing in the 1980s?*

I will therefore explain what I believe was missing on my part, at least, during those debates in the 1980s. So, this article now becomes a kind of *mea culpa*. The problem is actually very easy to explain. Like most bio-

logical scientists I was still under the sway of a seminal book, written in 1944 by the great quantum mechanics pioneer, Erwin Schrödinger, called *What is Life?* (Schrödinger 1944). I call it a seminal book because it led to the central Dogma of Molecular Biology in the work of Watson and Crick when they unraveled the double helical structure of DNA. Both acknowledge Schrödinger because he made two predictions in his book that were, apparently, to find their confirmations in the work of Watson and Crick. The first was that the genetic material, when it was discovered, would be found to be what he called an aperiodic crystal. If you think of a linear polymer as a kind of crystal—a bit of stretch, I agree—the description aperiodic is a very good one. It is precisely that characteristic that enables the molecular thread to encode so much information that enables a vast range of different proteins to be constructed by the living cell.

So far so good. But the second prediction of Schrödinger to be taken up by the molecular biologists simply cannot be true. He argued that, if one sees the genetic material as an information dense sequence, how is it read to enable the characteristics of an organism to be transmitted from one generation to another? A one-dimensional sequence cannot simply map a three-dimensional structure. It is not a miniature organism in the way in which some nineteenth century microscopists imagined when they looked at sperm and egg cells. Could that three-dimensional template come from somewhere else, perhaps in the three dimensional structure of the cell itself? Whichever way that is done, Schrödinger reasoned that the sequence must be read in a determinate manner if it was faithfully to transmit information. Stochasticity in a communication line is intolerable. From this he concluded that there must be an absolutely fundamental difference between physics and biology.

Physics can be characterised as order from disorder. At the micro level, there is the essential stochasticity of quantum mechanics. Even if, one day, an alternative view of "reality" is produced, as people like Albert Einstein and David Bohm believed, we can't escape the fact that the equations of quantum mechanics are precisely predictive as probabilistic descriptions. Any underlying determinism would have to reproduce this. That is not difficult to imagine since we already have an example of stochasticity at the molecular level that was discovered well before quantum mechanics. In 1827 Robert Brown observed that fine particles derived from pollen grains showed stochastic movement in water observed under the microscope. We call it Brownian motion and it was shown by Einstein (Einstein 1905) to arise from the random bombardment of the particles by the random motion of water molecules: the first demonstration of the existence of individual molecules with separate motions.

Yet, the equations of thermodynamics, which describe large numbers of particles to generate the gas laws, are determinate. The an-

swer to this apparent paradox is that, if motion at the particle level is genuinely random, then large numbers of particles will cancel their individual movements out to produce a constant pressure when hitting an object, like the wall of a pressure vessel. Order at large scales therefore arises from disorder at lower scales. In a living cell, the high-level properties of volume, pressure, temperature, acidity, and many other global parameters will display constant or smooth transitions.

But this interpretation is inconsistent with a Schrödinger view of biology in which the genetic material at the molecular level is supposed to be read in a determinate manner, rather as an X-ray beam can generate an accurate and determinate "picture" of a crystal by the diffraction of the rays by the regular structure of the crystal. Biology, he reasoned, was therefore the generation of order at large scale from order at the micro scale.

Schrödinger wrote:

> We seem to arrive at the ridiculous conclusion that the clue to understanding of life is that it is based on a pure mechanism, a 'clock-work'…The conclusion is not ridiculous and is, in my opinion, not entirely wrong, but it has to be taken "with a very big grain of salt" (1944: 101).

He then explains the "big grain of salt" by showing that even clockwork is, "after all statistical" (103). My reading of these last pages of Schrödinger's book is that he realises that something is not quite right but is struggling to identify what it might be. This confusion has muddied the waters for 80 years now.

We would now say that the molecules involved (DNA) *are* subject to statistical variation (copying errors, chemical and radiation damage, etc.), which are then corrected by the protein machinery that enables DNA to be a highly reproducible molecule. This is a three-stage process that reduces the error rate from 1 in 104 to around 1 in 1010, which is an astonishing degree of accuracy. The order at the molecular scale is therefore actually imposed *by the system as a whole.* This requires energy of course, which Schrödinger called negative entropy. Perhaps therefore this is what Schrödinger was struggling towards, but we can only see this more clearly in retrospect. He could not have known how much the genetic molecular material experiences stochasticity and is constrained to be highly reproducible by the organism itself.

So Schrödinger's idea that led to the Central Dogma can't be correct. It also led to the incorrect "read only" view of DNA.

Now, why is this important to the debates on teleology? The answer is that the Central Dogma should no longer be used to justify a closed determinate nature to biological processes. Just like everything else that depends on the motion of molecules, there is massive stochasticity at the lowest levels. Only at higher levels can there be the order that a genuine explanation of behaviour requires. Furthermore, it is precisely through the constraints that the higher order imposes on the lower level stochasticity that we can develop a multi-level theory that privileges the

higher level. That is the purpose of two of my most recent articles (Noble 2022a,b) and of my book, *Dance to the Tune of Life* (Noble 2016). Those constraints ensure that there is an asymmetry between the causal force of explanations at higher and lower levels. The higher level is genuinely causative because it is only from that level that one can understand the constraints and how they arise. This is the sense in which I think that Charles Taylor's conceptualist view of teleology is correct, and how I think it can now be given a firm biological science basis.

Furthermore, it is possible to show that this *necessarily* excludes the one-way reductionist causal explanation of organism behaviour. The complete argument is technical, but the overall conclusions are straightforward:

1. When we examine the mathematics of multi-level causation, which is encapsulated in the principle of biological relativity (Noble 2012), it is impossible to dispense with the influences of higher levels on lower-level behaviour. That is a mathematical necessity in any living system in which the molecular level is controlled by higher levels (Noble 2022, Noble 2022).
2. Organisms use lower-level stochasticity to generate their characteristic innovative activity in finding solutions to the challenges of survival. Our immune systems are doing that all the time, and they do so by changing the organism's DNA sequences in a highly targeted way (Odegard and Schatz 2006). That kind of selective targeting was supposed to be forbidden by the Central Dogma. It is not.
3. Similar harnessing of stochasticity occurs in the functioning of the nervous system, so that it becomes possible to explain the physiological processes that might underly innovative behavior (Noble and Noble 2020). It is at one and the same time, both stochastic (we can't necessarily predict a Beethoven or an Einstein), yet understandable in retrospect (we can judge the reasons and values that must have guided what was done).

I therefore think that one aspect of the debate is now closed. Higher level explanations *must* have validity because we cannot dispense with the influences of higher levels on lower-level behaviour. That is a mathematical necessity in any living system in which the molecular level is controlled by higher levels.

I want to conclude by noting that the issues on which Kathy contributed so much 30 years ago are still very much live issues today. If I have succeeded in moving the debate on somewhat I owe a lot to her insights and great contributions. Her insight that we may need to use "things that behave very like neurones" now seems prophetic.

6. *Coda*

Nearly 20 years ago, in August 2003, I was contacted by Alan Montefiore in London to ask whether I could possibly go to the John Radcliffe

hospital in Oxford to visit Kathy Wilkes, who was unwell. I did so. Kathy was indeed unwell. I was trained as a medical student, though I never treated patients, but I was saddened to see all the signs of a hopeless clinical situation. Kathy, though, immediately recognised me and we briefly discussed her work. Her mind was clearly focussed on Croatia and what happened in Dubrovnik. Sharp as a knife, she reacted immediately to my mistake in referring to Yugoslavia (which is what your country was when I first visited it in 1965). I immediately tried to correct what I said, but she was very firm and insistent: what I believe may have been her last words were "I am a fighter, I never give up."

She was!

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