Reasons of the heart: Neuroscience and the ethics of consent

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The French mathematician and philosopher Blaise Pascal (1623-1662) is famous for his pronouncement that ‘the heart has its reasons, of which reason knows nothing.’ Recent developments in the neuroscience of emotion support Pascal’s conception, with important and possibly dramatic implications for the traditional understanding of informed consent in contemporary bioethics and medical practice.

Informed consent is required for medical treatment and for participation in clinical research. Typically, an individual’s consent is considered to be valid if it is: (1) properly informed; (2) freely given; and (3) mentally competent. To say that an individual is mentally competent is to make a decision tantamount to saying that he/she has the decisional capacity to make that decision.

But how do we determine whether an individual has the requisite decisional capacity?

There are now well established clinical tools to assess decisional capacity. These generally analyze decisional capacity into several sub-capacities. Thus an individual must be able to understand the relevant information; he/she must also be able to appreciate the fact that the information in question bears on his/her condition. This additionally requires that a person has the ability to evaluate the risks and benefits of the decision he/she is faced with, and of course the ability to express a choice.

The problem with this conception of decisional capacity is that all the sub-capacities that define capacity are exclusively cognitive. This is a purely cognitive approach to decisional capacity. No provision is made for the influence of emotion and the ‘reasons of the heart.’ The result is a theoretically biased and empirically inadequate approach to the ethics of informed consent. This is where modern neuroscience can help.

Recent advances in the neuroscience of emotion provide compelling evidence of how emotions and their associated feelings contribute to the decision-making process. Research shows that emotions are often excellent guides to rational action and both the definition and determination of what counts as our best interests.

Of course, excessive and inappropriate emotions can detract from, and impede, rational action. But without the capacity for appropriately emotive, rational action is impossible. There is simply no way to evaluate and decide what is best for us and what to count as our interests.

Contemporary clinical tests to assess decisional capacity are exclusively cognitive. A good example is the MacArthur Competence Assessment Tool (MacCAT-T).

In the present case, the reverse is true. There is simply no way to evaluate and decide what is best for us and what to count as our interests. The result is a theoretically biased and empirically inadequate approach to the ethics of informed consent. This is where modern neuroscience can help.

Practical Tips: When an antidepressant stops working

A Your patient appears to be suffering from recurrent major depressive disorder, a condition in which the return of symptoms is common, even with continuation or maintenance treatment. It is estimated that the rate of relapse or recurrence in patients with depression receiving reasonable doses of antidepressant medication may be as high as 33 per cent and possibly even higher (1). The reason for loss of response in treatment-adherent patients is usually unclear.

Your patient’s adherence to treatment rules out one of the most common causes of “non-response.” There are other areas that require attention before considering a change in pharmacotherapy.

First, the diagnosis should be re-evaluated. For example, does the patient suffer from a bipolar disorder, significant co-morbidity, substance abuse, or an occult medical illness like hypothyroidism? If the answer is “yes,” an appropriate therapeutic approach will be required.

Second, one should ask whether stresses in the patient’s life may be adversely affecting him/her, and whether psychotherapy might be helpful. For many patients with depression, a combination of pharmacotherapy and psychotherapy is optimal. From your question, it appears that the life situation of your patient is not problematic, and that the return of her symptoms is biologically based.

If the patient is experiencing what is referred to as “tachyphylaxis” or, more colloquially, the “poop-out” phenomenon, there are several options. These include adjusting the dose (usually upwards), switching to another antidepressant, adding a second antidepressant with a different mechanism of action, or adding augmentation therapy. The simplest of these is to change the dose. In a fairly recent survey of strategies to deal with relapse during treatment with selective serotonin reuptake inhibitors (SSRIs), the most common first choice of psychiatrists was to increase the dose (2). Since then, a clinical trial has supported this strategy in the case of fluoxetine (3). A further increase in dosage would thus be an option for your patient, who tolerated a dose of 200 mg/day of sertraline without difficulty. Plasma drug level testing can also be helpful in guiding treatment, where available. I would be less inclined to increase the dose of the bupropion, because of the increased risk of seizures with higher doses.

Sertaline and bupropion have different mechanisms of action, and thus combining these two drugs is another good therapeutic strategy for your patient, especially if she tolerated the bupropion well. Because the rate of tachyphylaxis is lower with lower triyclic antidepressants and venlafaxine (which are “dual” reuptake inhibitors, affecting both serotonin and norepinephrine) than with SSRIs (4), a switch to venlafaxine would also be a rational choice for your patient.

Another option is augmentation with agents such as triiodothyronine, lithium, buspironate, or a psychostimulant. Given the somewhat bothersome requirements of lithium treatment (baseline tests and subsequent monitoring) and the risk of lithium toxicity, I generally keep lithium in reserve for use when the other augmenting strategies fail to work.

As you can see, there are many potential “next step” treatments for your patient. In general, the smaller the number of drugs prescribed, the better, and at this point I would be inclined to replace the bupropion with venlafaxine.

References