Placebos Effects and Informed Consent

Abstract

The concepts of placebos and placebo effects refer to extremely diverse phenomena. I recommend dissolving the concepts of placebos and placebo effects into loosely-related groups of specific mechanisms, including (potentially among others) expectation-fulfillment, classical conditioning, and attentional-somatic feedback loops. If this approach is on the right track, it has three main implications for the ethics of informed consent. First, because of the expectation-fulfillment mechanism, the process of informing cannot be considered independently from the potential effects of treatment. Obtaining informed consent influences the effects of treatment. This provides support for the authorized concealment and authorized deception paradigms, and perhaps even for outright deceptive placebo use. Second, doctors sometimes fail to consider the potential benefits of conditioning, leading them to misjudge the trade-off between beneficence and autonomy. Third, how attentional-somatic feedback loops play out depends not only on the content of the informing process but also on its framing. This suggests a role for libertarian paternalism in clinical practice.
To calm the imagination of the invalid, so that at least he should not, as hitherto, have to suffer more from thinking about his illness than from the illness itself – that, I think, would be something!

~ Friedrich Nietzsche, *Daybreak* 54

1. Introduction

In a paradigmatic case of informed consent to medical intervention, there is a strict temporal sequence. First, the patient presents at the clinic, signaling a desire or need for medical help. Second, the physician examines the patient and forms a diagnosis. Third, the physician recommends an intervention. She doesn’t just hook the patient up to an IV drip, force-feed him pills, or anaesthetize him and start cutting. She informs him of what she’d like to do, explains the potential benefits, and identifies the risks. Fourth, the informed patient deliberates about these benefits and risks, deciding whether to consent to the doctor’s recommendation. Finally, if consent has been provided, the doctor goes ahead with the recommended treatment.

As you might suspect, I don’t think things are always so simple.

I want to argue that the phenomena of placebo and nocebo effects complicate the ethics of the informed consent process. To see why this is so, I first need to show why the current conceptualization of placebos and placebo effects is misguided. The concepts of placebos and placebo effects refer to phenomena that are incredibly diverse. They have been classed together only because they have been defined in a negative and theory-relative way. I recommend dissolving the concepts of placebos and placebo effects into loosely-related groups of specific mechanisms, including expectation-fulfillment, classical conditioning, and attentional-somatic feedback loops, then investigating these mechanisms both independently and in their interactions.
If this approach is on the right track, it has implications for the ethics of informed consent. Three values governing doctor-patient interactions are autonomy, beneficence, and non-maleficence. These values often point in the same direction. If autonomy is a component of wellbeing, then promoting the patient’s autonomy always benefits him and undermining his autonomy always harms him. Enhancing someone’s wellbeing can help him to make better decisions, thus promoting autonomy. But these values can also come into conflict. Reasonable, informed patients may make choices that the doctor knows to be sub-optimal, even harmful.

Epistemic and emotional imperatives may pull in opposite directions. For instance, being told that you have a significant chance of developing a deadly cancer – something you obviously need to know if you’re to make an informed decision – may cause anxiety or depression. A single value, such as autonomy, can even conflict with itself. We need to resist making autonomy a sacred value and think hard about the trade-offs physicians often face during the informed consent process. Additionally, we need a nuanced, empirically-informed conception of autonomy. More information doesn’t always lead to better decision-making, and can even introduce bias. Promoting someone’s autonomy can therefore involve concealing information or even providing misinformation.

In the final section of the paper, I’ll argue for three normative claims about informed consent and placebo effects. First, because of the expectation-fulfillment mechanism, the process of informing moderates (in the statistical sense) the effectiveness of treatment. The same treatment will have different effects depending on how the patient has been informed. This provides support for the authorized concealment and authorized deception paradigms, and perhaps even for unauthorized deception. Second, physicians often fail to consider the potential benefits of conditioning, leading them to misjudge the trade-offs among beneficence, non-
maleficence, and autonomy. Third, how attentional-somatic feedback loops play out depends not only on the content of the informing process but also on its framing. This suggests a role for libertarian paternalism in clinical practice.¹

2. Critique of the AMA and Grünbaum

Negative predicates are typically pretty useless in science. When we can, we should avoid formulating scientific theories negatively. Unfortunately, all of the prominent theories of the placebo effect are framed negatively.

A prominent, early attempt to conceptualize placebos and placebo effects is due to Shapiro & Morris (1978), who define a placebo as a therapy that is prescribed for its “nonspecific” effect, or is “without specific activity for the condition being treated,” and a placebo effect as the effect characteristically produced by a placebo. The term ‘specific’ is vague and ambiguous. ‘Nonspecific’ is even worse. The class of nonspecific effects on health and illness is mind-bogglingly diverse. How, one might wonder, will we ever have a scientific theory of placebo effects if we define them negatively?

Following Shapiro & Morris, the American Medical Association in 2007 defined a placebo as “a substance provided to a patient that the physician believes has no specific pharmacological effect upon the condition being treated.” There are so many things wrong with

¹ Libertarian paternalism is a view in political philosophy developed by Thaler & Sunstein (2003) according to which it is sometimes permissible for a state, authority, or institution to select a policy “with the goal of influencing the choices of affected parties in a way that will make those parties better off,” but which does not involve coercion. Such interventions instead involve “nudges,” which shape decision-making without taking any options off the table.
this that I hesitate to start criticizing it. Just for starters, because ‘pharmacological’ is included in the definition, the AMA is committed a priori to the claim that surgery, vertebroplasty, and cognitive-behavioral therapy are all placebos. But even if we tidy up the AMA definition by dropping ‘pharmacological’, we’re back at Shapiro & Morris’s 36-year-old definition.

The AMA can be forgiven its confusion, though, because even Adolf Grünbaum’s (1981, 1994) model, arguably the most sophisticated model in the literature, is also negative and theory-relative. According to Grünbaum, a placebo effect is any effect on the target disorder caused by factors of the treatment that are not identified by the dominant therapeutic theory as efficacious.²

This leads to theoretical, empirical, and programmatic difficulties. Theoretically, defining placebo effects negatively makes them difficult to understand, explain, and unify. It would be roundabout at best to define rabbits as non-hare, non-pika lagomorphs. There might turn out to be other, quite diverse, animals that fit this description. In the same way, it’s roundabout at best to define placebo effects as effects on the target treatment produced by nonspecific factors. There’s no reason to suppose that what isn’t specific forms a unified class. Hence, there’s no reason, if placebos and placebo effects are defined negatively, to talk about the placebo effect as if it were a unified phenomenon.

Empirically, a negative definition in terms of effects that are “nonspecific” is at odds with our knowledge of the neurobiological and psychological causes of some placebo effects, which are quite specific indeed (Miller & Brody 2011; Kong et al. 2013; Benedetti 2008). We now

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² Grünbaum does not say what makes a therapeutic theory dominant. Presumably, this is a matter of sociological facts, including both the proportion of physicians who accept the theory, the expertise hierarchy in which they find themselves, testimonial and justificatory links among them, and so on.
know which neurobiological interventions produce certain placebo effects; for instance, some instances of placebogenic analgesia are a product of expectation-induced activation of endogenous opioids and cholecystokinin (Levin, Gordon, & Fields 1978; Benedetti 2008).

Programmatically, defining placebo effects negatively sets investigators of the causes of placebo effects adrift. Scientific research programs thrive on refining and testing ever more precise and audacious hypotheses (Lakatos 1995). The essential imprecision of “nonspecific” makes it refine theories of the placebo effect. Furthermore, negative definitions have a pernicious institutional effect by discouraging research on the mechanisms that underlie placebo effects. Imagine designing a research program aimed at explaining placebo effects. If you subscribed to a negative definition, it would be necessarily self-defeating: once you explain a placebo effect, it’s no longer a placebo effect because now it’s specific rather than nonspecific. By analogy, think about a phenomenon called the “mysterious effect,” which contemporary researchers don’t understand. If someone claimed to have explained the mysterious effect, it would cease to be mysterious, or she might be accused of explaining some other thing—since of course the mysterious effect is mysterious.3

As I mentioned above, Grünbaum’s definitions of placebos and placebo effects are arguably the best in the literature, but I think they are the best of a bad lot.4 One reason for this

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3 I am here indebted to [insert reference]. This problem is related to what Brody (1985) calls “the disappearance problem.”

4 Benedetti (2008) should be an exception. In his recent monograph, he explores at great length the diverse mechanisms that explain various placebo effects. However, he defines a placebo effect simply as “the effect that follows from the administration of a placebo, that is, of an inert
is that his definitions are problematically negative in exactly the same way that the AMA’s is. He defines a placebo effect as any change in the target disorder produced by non-characteristic (incidental) factors of the treatment (1994, p. 295). To clarify this definition, it’s helpful to reproduce a figure from his paper (p. 291):

![Figure 1: Grünbaum’s Placebo Model](image)

In this model, the characteristic factors $F$ are any and all aspects of the treatment that are explicitly or implicitly picked out by the dominant therapeutic theory as therapeutically efficacious. The incidental treatment factors $C$ are everything else, so placebo effects are defined as whatever effects are produced in the target disorder by everything that is not characteristic. Just as the class of nonspecific effects is so broad and diverse that it’s hard to say anything interesting and informative about it, so the class of incidental factors is so broad and diverse that it’s hard to say anything interesting and informative about it.

Another drawback of Grünbaum’s definition is that it is problematically theory-relative. In this context, a definition is theory-relative to the extent that what counts as a placebo or a placebo effect depends on what the prescribing physician (or the community of medical experts, treatment)” (p. 37). Of course, an inert treatment is just a treatment that is not effective on its own.
or the patient, or anyone else) thinks or espouses. For instance, the following definition is theory-relative: a treatment is a placebo just in case the way it produces its effect is contrary to what the patient thinks. This definition is also theory-relative: a treatment is a placebo just in case the way it produces its effect is not described in the dominant therapeutic theory of the target disorder.

Furthermore, Grübaum’s notion of a dominant therapeutic theory assumes that the theory in question concerns mechanisms of standard medical treatments and is not a theory of how a given family of placebo effects is produced. The characteristic factors specified or assumed by therapeutic theories generally are the chemical composition of the drug and the physical manipulation of the surgical intervention. The theory might also identify a physiological pathway by virtue of which the characteristic treatment factors produce therapeutic benefit. This is turned on its head when the focus of intervention is promoting and elucidating placebo effects. In the latter situation, we might talk about “placebo therapeutic theories,” which understand the physical properties of treatments (and their physiological effects) as incidental. Characteristic features on this approach include a neutral stimulus in a classical conditioning paradigm, aspects of the clinician-patient relationship, verbal suggestions, the ritual of treatment, and so on. If we plug a “placebo therapeutic theory” into this model, it now looks like what are ordinarily called “placebo effects” turn into non-placebo effects, since they are the product of characteristic factors as specified by the placebo therapeutic theory.5

Finally, Grübaum takes the notion of a dominant therapeutic theory for granted. At any given time, though, there will often be multiple therapeutic theories in play. Different medical “tribes” of researchers and physicians endorse different theories of the same disorder. If this is

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5 I am here indebted to Frank Miller.
right, there will often be multiple dominant theories, and medical practitioners unaligned with any particular tribe may pick and choose somewhat eclectically from them. A prescribing physician might be aiming to maximize patient benefit, and so would want to harness the benefits of two or more dominant theories. She would then be drawing simultaneously on multiple theories, each of which treats at least some of the others’ characteristic factors as incidental. What are we to say about such a case? If the theories are kept distinct, then the same intervention would be both a placebo and a non-placebo, and the same effect would be both a placebo effect and a non-placebo effect.

Grünbaum and his allies might have responses to these problems. I’m not claiming to have decisively undermined his conceptual analysis, but these problems seem sufficiently thorny that I’m inclined to seek an alternative approach to placebos and placebo effects.

3. The family resemblance of placebogenic mechanisms

To drive these points home, let me briefly canvas three psychological mechanisms that seem to be responsible for some cases of placebo effects. They have very little to do with each other, but all three are placebogenic.

3.1. Expectation-confirmation

Probably the best-attested psychological theory of the mechanism of placebo effects is the expectation-confirmation theory. Humphrey (2002, p. 256; see also Miller 2005) is a recent exemplar. He defines a placebo as:

a treatment which, while not being effective through its direct action on the body, works when and because:
• the patient is aware that the treatment is being given;
• the patient has a certain belief in the treatment, based, for example, on prior experience or on the treatment’s reputation;
• the patient’s belief leads her to expect that, following the treatment, she is likely to get better;
• the expectation influences her capacity for self-cure, so as to hasten the very results that she expects.

The difference between the effects of standard treatment and a placebo effect, on this view, is that the patient’s expectations are causally implicated in their own satisfaction. An appendectomy will end your appendicitis regardless of whether you think it will, but placebo analgesia works only because you expect that it will.

The expectation-confirmation mechanism has been investigated for decades and is now well-substantiated. People report more pain relief from branded aspirin than from unbranded aspirin, more relief from unbranded aspirin than from branded placebo, and more relief from branded placebo than from unbranded placebo (Branthwaite & Cooper 1981). Patients who expect pain-relief from their saline drip because they’ve been told that it’s infused with a painkiller request acute analgesia less frequently than patients who are less inclined to expect continuous analgesia (Pollo et al. 2001). Positron emission tomography (PET) reveals that raising expectations of symptomatic relief leads to a large release of dopamine in the brains of patients with Parkinson’s disease (de la Fuente-Fernandez et al. 2001). In a functional magnetic resonance imaging (fMRI) experiment on both placebo analgesia and nocebo hyperalgesia, Wager et al. (2004) found that when expectations of pain relief are induced, there is a corresponding decrease in activity in brain regions associated with pain processing, such as the
thalamus, insula, and anterior cingulate cortex, but when an increase in pain is anticipated, there is a corresponding increase in activity in the prefrontal cortex.

Thus the expectation-confirmation theory has been corroborated as an explanation of not only some placebo effects but also some nocebo effects. There are many further examples, which I can only touch on here. For instance, the incidence of the listed side-effects of a given drug spikes even in the placebo arm of trials studying that drug (Amanzio et al. 2009). The same treatment (influenza vaccine) leads to worse outcomes on both subjective measures (reported side effects) and objective measures (absenteeism) when framed negatively (mentioning that side effects occur in N% of cases) than when framed positively (mentioning that side effects are not observed in (100 − N)% of cases), even though the facts expressed by the different framings are identical (O’Connor, Pennie, & Dales 1996). Even the mere verbal suggestion of potential negative outcomes can generate nocebo effects (Benedetti et al. 2007). For instance, when male patients treated with finasteride are told that it may cause erectile dysfunction, decreased libido, and problems with ejaculation, but that these are uncommon, their reports of these symptoms, as compared with uninformed patients, jump from 9.6% to 30.9% for erectile dysfunction, 7.7% to 23.6% for decreased libido, and 5.7% to 16.3% for problems with ejaculation (Mondaini et al. 2007).

3.2. Classical conditioning

While the expectation-confirmation theory is well corroborated, it does not cover all known cases of placebo effects. Some placebo effects are explained instead by the classical conditioning theory: the body learns to react to the conditioned stimulus, such as a pill of a
certain shape and size, in the same way it responds to the unconditioned stimulus, such as the
drug hitherto contained in pills of that shape and size.

Ader (2000), for instance, showed that starting a patient off on a course of treatment for
psoriasis with pills that contain 100% of the normal dosage and then weaning them to otherwise
inert pills that look and taste exactly like the real thing leads to outcomes indistinguishable from
normal treatment. Using pills with 50% of the normal dosage throughout treatment is not as
effective. Perhaps even more telling is the fact that inducing expectations of increase or decrease
in growth hormone and cortisol has no effect on their secretion, but preconditioning with
sumatriptan, which inhibits cortisol secretion while stimulating growth hormone secretion,
enables placebogenic enhancement of growth hormone production and placebogenic reductions
in cortisol production (Benedetti et al. 2003). Preconditioning in this way is effective even when
patients are led to expect otherwise. Moreover, the expectations and conditioning mechanisms
can sometimes be harnessed in tandem to strengthen the effect that either would produce on its

3.3. Somatic attention and feedback

Yet another potential mechanism for the production of placebo and nocebo effects, which
has, unfortunately, received less scientific investigation, is somatic attention and feedback. Daily
experience is a continuous deluge of stimuli. We notice only some of them. We actively attend
to even fewer. When we attend, we interpret or construe in particular ways. How we construe
depends both on what the stimuli are and our current state of mind – including our current
beliefs, desires, emotions, and moods. Construal then affects our state of mind, our attentional
focus, our behavior, and through these channels the stimuli that we experience later. If your
doctor primes the concept of pain in your left big toe, you will pay more attention to the toe and be more disposed to interpret ambiguous sensations there as pain. Thus, you will be more disposed to notice slight pains that would otherwise have slipped under the radar of attention, as well as to interpret ambiguous stimuli as pain (Allen & Siegel 2002). If instead your doctor primes the concept of relief of your headache, you will pay more attention to sensations in and around your head and be more disposed to interpret ambiguous stimuli as the alleviation of pain. Thus, you will be more disposed to notice slight lessening of the intensity of your headache, as well as to interpret ambiguous stimuli as pain relief. One way in which placebogenic analgesia might occur, then, is simply by raising the threshold for both noticing pains and for counting a sensation as pain rather than heat or pressure.

One distinguishing characteristic of this mechanism is that it is essentially dynamic. When you attend to your head – ready, as it were, for pain relief – and you end up construing a sensation as alleviation, you may become less anxious, which would produce bodily changes such as decreased blood pressure and heart rate. These bodily changes might then cause further pain relief. In this way, a feedback loop connects your sensations, somatic attention, construal, and bodily states.

When such a feedback loop repeats over and over again, it can generate a feedback cascade. The slight or ambiguous pain relief that you notice only because your doctor led you to look for pain relief leads to slight bodily changes. These slight changes cause changes in sensations, in somatic attention, and in dispositions to construe, which in turn lead to noticing further pain relief, which leads to further bodily changes, and so on.

The same story can be told about certain nocebo effects, such as nocebogenic erectile dysfunction. If, during the informed consent process, a man is led by his physician to worry
about erectile dysfunction, he will be more disposed to notice even slight or potential problems, and to construe ambiguous phenomena as evidence of dysfunction. Noticing and construing in this way will lead to bodily changes and loss of confidence, which in turn may affect experienced stimuli, somatic attention, and processes of construal. If this process cascades – that is, if the loop repeats several times and builds on itself – it may lead to outright erectile dysfunction.

Few empirical investigations of the mechanisms of placebogenic and nocebogenic effects have theorized that somatic focus plays this role, and, to my knowledge, none has been formulated in terms of feedback loops and cascades. There are a couple of suggestive results, however. In one recent study, Geers et al. (2006) found that participants who were instructed to attend to their bodily states for potential negative side effects of an otherwise inert pill reported feeling more anxious and nauseated than participants who were not so instructed. Another recent study (Walker et al. 2006) found that children felt and reported most discomfort when their parents were instructed to attend to their symptoms, less discomfort when their parents were uninstructed, and least discomfort when their parents were instructed to distract them from their symptoms.

3.4. Summing up: Family resemblance

Diverse psychological mechanisms seem to be implicated in the production of placebo effects. Expectation-induced release of dopamine in the striatum is a placebogenic treatment for Parkinson’s disease; preconditioning with immunosuppressive drugs such as cyclophosphamide and cyclosporine A modulates immune mediators such as IL-2, IFN-γ, and lymphocytes
(Benedetti 2008). It seems incredible to interpret these phenomena as falling under a single natural, biological, or psychological kind.

I contend that the best response to this issue is to dissolve the heterogeneous placebo phenomena into their coherent parts, which should be theorized and investigated both independently and in their interactions. It may remain useful to retain the concept of a placebo effect for some purposes, just as it remains useful to retain the concept of congestive heart failure. However, in research contexts and in many clinical contexts, clarity and effectiveness will be promoted by specifying more clearly the precise mechanisms at work.

4. Implications for informed consent

If my arguments so far are on the right track, they have several implications for informed consent. Before considering these implications, it’s important to clarify two points.

First, these placebogenic mechanisms do not require deception. Naturally, much research on the placebo effect has used deception, and it may be part of folk medical lore that deception is an essential ingredient in producing placebo effects. Nevertheless, deception is not required to alter a patient’s expectations, to classically condition them, or to modulate their somatic attention. Expectations can be managed without lying to, misleading, or otherwise deceiving someone. Classical conditioning works even if you know you’re being conditioned. Directing, focusing, and dilating attention is a venerable part of meditative practices that have nothing to do with deception.

Second, these placebogenic mechanisms are not alternatives to traditional treatments: they can be used fine-tune and enhance traditional treatments. In the same way that a “drug cocktail” can be more effective than the sum of its parts, ordinary treatments combined with
placebogenic mechanisms may sometimes produce better results than simply summing the benefits of each would suggest.

How might this work?

4.1. Implications based on expectation-confirmation

For one thing, expectation-setting occurs as early as the informing stage of the informed consent process (and perhaps even earlier, under the influence of the appearance of the clinic and physician, the staff’s demeanor, the reputation of the treatment and physician, and so on). This means that a physician’s recognition of the psychological, somatic, and attentional implications of the ways in which informing can take place should impact her decisions about how to inform. Raising expectations of benefit and lowering expectations of harmful side-effects is a treatment pathway that gets up and running during the informing stage and before consent.

Given this, we need to weigh concerns for autonomy against concerns for beneficence and non-maleficence during the informing stage (Miller & Colloca 2011; Colloca & Miller 2011). It’s comforting to think that informing means telling the patient all and only the things he needs to know about his condition and the potential benefits and risks associated with each treatment option. Spelling out what someone needs to know, though, turns out to be pretty tricky. If the patient were a dispassionate statistician, one could give him a list of all potential benefits and harms, paired with their probabilities, for each treatment option. Since so few patients fit that description, the doctor inevitably says less – usually much, much less. Suppose, as seems reasonable, that some probability- and value-weighted threshold is used, if only intuitively, to decide when to mention a benefit and when to mention a risk. A benefit or risk might not be mentioned because it is deemed too trivial or too unlikely (or some combination of
the two). But, as I pointed out earlier, the probability of some benefits and harms is conditional on whether someone is informed about them. If research suggests that the probability of a benefit’s occurring given that it’s mentioned is greater than the probability of the same benefit’s occurring given that it’s not mentioned, a beneficent physician should mention it. Likewise, if research suggests that the probability of a harm’s occurring given that it’s mentioned is greater than the probability of the same harm’s occurring given that it’s not mentioned, a non-maleficent physician should think twice before mentioning it. Of course, if the probability is high enough either way, or if the harm is serious enough, autonomy may outweigh non-maleficence, but the tradeoffs need to be borne in mind.

Another way to manage expectations in the interest of non-maleficence is to employ authorized concealment or authorized deception rather than informed consent. Miller, Kaptchuk, Colloca, and others have proposed using authorized deception or concealment instead of both informed consent and outright deception in some contexts (Colloca & Miller 2011; Miller, Wendler, & Swartzman 2005; Miller, Colloca, & Kaptchuk 2009). It’s worthwhile to explore whether authorized deception or concealment might also be appropriate in some clinical contexts.

The basic idea with authorized concealment is straightforward and attractive. Suppose a doctor is consulting with a man who seeks treatment for pattern baldness. She recommends finasteride. As I mentioned earlier, one side effect of finasteride is erectile dysfunction, which is 300% more likely when the patient has been led to expect it. Instead of telling the patient about this side effect, the doctor might say, “As with any hormone therapy, this treatment carries the risk of side effects. Research suggests that if I tell you what those side effects are, you’ll be much more likely to experience them. So, what I’d like to do is ask your permission not to
mention these side effects. I’ll debrief you about them after the treatment is over.” Such a strategy carries its own risks. If the patient is led to expect an ominous but diffuse set of risks, he may end up even more anxious than he would have been with the precise details, which could lead to nocebo effects even worse than those already documented. He might also interpret any negative health outcome as probably just the side-effect of the finasteride treatment – even if it’s a quite serious and unrelated problem, such as a minor stroke or heart attack. If he shrugs such problems off because his physician told him to expect some indeterminate negative symptoms, he will have been seriously harmed by authorized concealment.⁶

There are a couple of potential solutions to this problem. First, the physician should only use authorized concealment when the risks are merely symptomatic. She could then instruct the patient not to ignore anything that was more-than-symptomatic. However, even in that case, the patient may not know what’s merely symptomatic and what is serious or even life-threatening. Given this, the physician could provide the patient with a “bad fortune cookie” – essentially, just a list of the potential side-effects that she concealed, contained in a sealed envelope. The patient would be instructed to open the bad fortune cookie if he experienced an unexpected adverse health outcome during the course of treatment, but not otherwise. This would enable him to avoid mistakenly judging non-side-effects as side-effects, and it would constitute a kind of informing. Of course, one might worry that patients will always (or never) open their bad fortune cookies. In the former case, no additional harm would be done, since they would have exactly the same expectations as someone who was directly informed about potential negative side-effects. In the latter case, serious harm might result. This is an empirical question to which we currently lack an answer.

⁶ I am here indebted to Roberta Millstein.
Authorized deception goes a bit further than authorized concealment. In this paradigm, the physician doesn’t just say, “There are certain things I’d like to keep from you. Is that OK?” Instead, she says something along the lines of, “Research suggests that you will have better symptomatic outcomes if I lead you to believe some things that are, in fact, false. Naturally, I understand that you want to know everything that’s relevant to your prospects, but some of that knowledge might lead you to suffer unnecessarily. What I’d like to do is ask your permission to deceive you about some things in order to promote your welfare. I’ll debrief you about them after the treatment is over.” Again, it’s an empirical question how this would play out, but it does seem to be worth exploring. And, again, the physician could provide the patient with a bad fortune cookie that he could consult in case of unexpected adverse health outcomes, which would disabuse him of the deception. In addition to the bad fortune cookie, an “agony aunt” could be elected – basically, someone who knew the patient well, had somewhat matched values, and could be expected to keep his information confidential. The agony aunt would be told the truth about the patient’s prospects, asked to keep an eye on him, but instructed not to reveal the deception unless it seemed necessary. (This solution might also work well in the authorized concealment paradigm.)

Beyond authorized concealment and authorized deception, there are various more paternalistic policies to consider. It may be that the associated benefits and harms associated with them would never outweigh concerns about autonomy, but it’s worth considering them if only to see how different they are from my earlier proposals. There is a spectrum of practices to consider. At one extreme, there’s outright assertion of a falsehood to the patient – directly lying with the aim of improving or protecting his wellbeing. My impression is that this is actually quite prevalent, but don’t go clutching your pearls just yet. Lying to someone during the
informing stage surely undermines their autonomy, so it already has one strike against it. A further point against it, which I don’t think has been mentioned in the literature, is that it violates the principle of non-maleficence, since it involves epistemic injustice. As Miranda Fricker (2007) defines this term, epistemic injustice is any injustice that harms someone *qua* potential knower. The benefit to the patient would have to be large indeed to justify both undermining his autonomy and committing epistemic injustice against him.\(^7\)

Various actions fall further and further short of outright lying. For instance, the physician could implicate or suggest a falsehood without outright asserting it. “This might help” doesn’t specify how likely it is that the intervention will help, and so should be used when the probability of help is low. When the probability is high, say 90%, the physician should mention it. When it is low, perhaps the best thing to do is not to mention it and allow the patient to infer that it is high.

As I’ve already discussed, instead of simply lying, the physician could seek authorization for deception. Especially if the patient were provided with a bad fortune cookie and/or an agony aunt, this is even less problematic than lying or implicating a falsehood.

Next, consider the fact that patients often present with wild misconceptions about what treatment might be able to do for them. When research suggests that a particular misconception (for instance, the misconception that prayer is an effective healing pathway) might be beneficial and is otherwise harmless, the physician should consider whether it is worthwhile to debunk the

\(^7\) Foddy (2009) argues that deceptive placebo, when it is the best available and most effective treatment with no potential risks, should be prescribed in the clinical context. Indeed, he argues that “[r]esponsible placebo prescription cannot limit a patient’s capacity for self-government” (p. 9). It’s worth noting how different my suggestions here are from his position.
misconception. Perhaps this seems objectionably paternalistic. I think not. After all, there are many potential beneficial misconceptions. Should the physician seek to disabuse her patient of all of them, just in case? Presumably not. If that’s the case, though, then the question is not whether to allow beneficial illusions to remain in place but which ones not to oppose (after all, the physician could try but fail to disabuse her patient of an illusion). This decision should be made, as I already argued, by thinking through the potential benefits and risks, with special attention to whether the risks are merely symptomatic and the probability and clinical importance of the potential benefits. It might seem that more information is always better, but if information shatters the patient’s worldview and confidence, learning the truth might actually undermine his ability to make decisions and thus harm his autonomy.

4.2. Implications based on conditioning

As I explained above, there are placebogenic pathways beyond expectation-confirmation, such as classical conditioning. When research suggests that the body can learn to produce the desired response without pharmacological intervention, the physician should consider doing so. Pharmacological interventions often produce unwanted side effects. If the same benefits can be had without those effects, conditioning better aligns with the value of non-maleficence.

If adherence were not an issue, this recommendation would be completely uncontroversial. However, the physician might worry that her patient will not take his medicine consistently enough for conditioning to work, especially if he knows that it no longer contains a pharmacologically active ingredient. One solution to this is to continue with 100% dosage through the course of the treatment. Another is to explain classical conditioning and insist on
adherence. Yet another is to have the patient install an adherence app such as MyMedSchedule, MyMeds, or RxmindMe.

This method, if pursued aggressively and consistently, might have beneficial side-effects on adherence to other treatment regimens. If the patient learns that his prospects depend on adherence in one instance, he may be more inclined to adhere to prescribed treatments in other cases well. After all, if I know that classical conditional will help me in the course of treatment X, it’s natural to think that it might help me in the course of treatment Y. Paradoxically, informing patients about the placebo-genic causes of some of their treatments may – when suitably paired with education and encouragement – induce them to ensure that their other, non-placebogenic treatments are more successful.

Beyond this, the physician could consider employing authorized concealment, authorized deception, and outright deception to encourage adherence. In the case of authorized deception, she could say, “It’s very important that you take your meds exactly as prescribed. As it turns out, unless you insist otherwise, some of them won’t be exactly what you expect, but we do this only to reduce the incidence of side-effects. I’ll inform you of the details after the course of treatment is over.” In this case, assuming that there are no issues associated with adverse side-effects from using the placebo rather than the actual drug, there is almost no reason to worry. To be sure, the physician could give the patient a bad fortune cookie or ask him to elect an agony aunt.

4.3. Implications based on attentional-somatic feedback loops

One final implication for informed consent, based on attentional-somatic feedback loops, is at the other end of the paternalism scale. How attentional-somatic feedback loops play out
depends not only on the content of the informing process but also on its mode of delivery. This suggests a role for libertarian paternalism in clinical practice. As I mentioned earlier, priming can influence the phenomenology of pain. Arguably, it’s impossible to deceive with priming, since it’s a non-propositional form of influence that does not lead to the formation of beliefs – not directly at any rate. This means that placebogenic priming is always consistent with the value of autonomy.

One might worry that this overstates the case. Granted, priming someone never constitutes a lie and therefore cannot undermine autonomy through deception, but might it not undermine autonomy through manipulation?

In this instance, it’s useful to bear in mind Pugh’s (forthcoming) recent argument about autonomy. Deception doesn’t, he argues, essentially undermine autonomy. Instead, it does so through one or both of two of its typical effects. The first such effect is subjugating the patient’s will to the physician’s authority. When people are deceived into treatments that are riskier than they think, as in the infamous Tuskegee syphilis experiment, this happens. The second such effect is short-circuiting the patient’s ability to act effectively in pursuit of his ends. In many instances of deceptive clinical placebo prescription, neither of these autonomy-undermining criteria is met. Pugh’s argument concerns deception, but it applies equally well – perhaps better – to benign manipulation. As he argues, deception (and benign manipulation) in such cases may be morally permissible or even obligatory. He claims that deception is obligatory when the patient is unable to act as an effective agent without the false belief engendered by deception. By parity of reasoning, we might conclude that benign manipulation is obligatory or at least permissible when the patient is unable to act as an effective agent without the framing and
nudging of the physician. If this line of argument is on the right track, autonomy and beneficence may not be so much at odds as the foregoing discussion suggests.⁸

A slightly trickier case involves not priming but framing. The same proposition can be expressed in different ways, called frames. Decades of psychological research have shown that different framings of the same information can induce divergent decision-making. For instance, people are more inclined to prefer a program that saves 200 of 600 threatened lives to a program that loses 400 of 600 threatened lives. Different framings of the same information have also been shown to induce divergent side effects. As I mentioned earlier, influenza vaccination produces worse outcomes on both reported side effects and absenteeism when framed negatively (mentioning that side effects occur in N% of cases) than when framed positively (mentioning that side effects are not observed in (100 – N)% of cases), even though the facts expressed by the different framings are identical (O’Connor, Pennie, & Dales 1996).⁹ This is an example of the libertarian paternalism (cited above) favored by the behavioral economists Cass Sunstein and Richard Thaler.

It seems fairly clear to me that non-maleficence outweighs autonomy (to the extent that autonomy is threatened at all by strategic framing) in cases like this. If one worries that I’m wrong about this, it’s worth considering recent work on the moral permissibility of nudges and other attempts to shape people’s preferences outside the clinical context (Alfano 2012, Khader 2011, Kim forthcoming). The main upshot of this work is that, since people’s preferences and

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⁸ It’s worth noting here that this line of argument supports Katz’s (1994) sustained arguments that doctors may have been right to resist extensive informed consent procedures in the name of beneficence and non-maleficence.

⁹ Colloca & Miller (2011) make a similar suggestion.
values are to some extent indeterminate, unstable, and context-sensitive, it may sometimes be that their autonomy is better supported by shaping their preferences than by helping them to get what they already want (to the extent that there even is such a thing). Khader in particular convincingly argues that such shaping of preferences is morally permissible when it is done in consultation with the very people whose preferences are to be shaped. After all, it’s impossible (or at least not morally objectionable) to manipulate yourself. In other words, Khader has already provided a stout defense the beneficient, non-maleficent, and autonomy-preserving use of framing outside the clinical context. My own argument simply imports that view to bioethics.10

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