

## Cognitive Distortion, Behavioral Shutdown, and Recovery: A PV–PP Case-Formulation Model for CBT Practice

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### Abstract

This paper presents a bounded conceptual model for one important class of depressive shutdown: cases in which meaningful actual capacity remains, but the patient’s reading of what remains possible has deteriorated enough that the person behaves as though no viable recovery-bearing path remains. The model is drawn from the productive value, productive power (PV–PP) framework, a decision architecture that distinguishes actual capacity from perceived capacity, separates the construction of possible actions from final choice among them, and treats recovery as restoration of a viable forward path rather than temporary symptom relief alone. Within this paper, perceived productive power (PPP) refers to the patient’s interpreted estimate of what they can still carry, survive, and realistically do. The paper does not propose a total theory of depression and does not displace biological, developmental, trauma-based, or pharmacological accounts. Its narrower claim is that the PV–PP architecture may help CBT practice by locating superficially similar shutdown states on different internal failure surfaces. In this account, withdrawal often arises upstream of final choice because viable actions are screened out before comparison as too costly, too exposed, too delayed, or too unlikely to alter trajectory. The practical value of the model is differential case formulation: it separates actual capacity loss from capability underestimation, memory contamination from expectation-level learned futility, hard barriers from falsely hardened barriers, and temporary relief from genuine recovery-corridor reopening. These distinctions support more targeted use of cognitive restructuring, retrospective reappraisal, behavioral activation, and graded re-entry.

## 1. Introduction

This paper uses a limited portion of the productive value, productive power (PV–PP) framework to address a clinician-facing problem in CBT case formulation: depressed patients can look similarly shut down on the surface while differing materially in where their action field is failing.

For purposes of this paper, PV–PP can be read simply as a decision architecture. It distinguishes a person's actual remaining capacity from the person's perceived remaining capacity, distinguishes the construction of possible actions from final selection among them, and distinguishes temporary relief from reopening a path the person can actually live in. The paper does not require prior familiarity with the broader framework beyond those distinctions.

A large share of depressive suffering is not explained solely by literal absence of options or total external collapse. It is also shaped by how the person interprets what remains possible, what burdens appear survivable, what supports still count as usable, and whether any recovery-bearing path still looks live. In practice, many depressed patients do not present as people calmly choosing among intact options. They present as people for whom the live action field has already narrowed.

That clinical picture matters because shutdown often looks less like sadness alone and more like collapse of initiation, collapse of bridge tolerance, and collapse of confidence that effort can change trajectory. Existing CBT traditions describe many of these features well. What is often less explicit is where the failure is located. Is the primary problem inaccurate reading of current capability? Distorted remembered evidence? Generalized expectation that effort will not pay? Misclassification of soft barriers as hard impossibilities? Or false conclusion that no

adequate recovery path exists? Those are not the same clinical problem, and they do not imply the same intervention emphasis.

The contribution claimed here is not that PV–PP discovers cognitive distortion, self-efficacy loss, hopelessness, or learned helplessness. CBT and adjacent literatures already describe those phenomena. The narrower contribution is ordered failure-surface attribution. In this paper, perceived productive power (PPP) means the patient’s interpreted estimate of what they can still carry, survive, and realistically do. The architecture separates actual productive power (PP) from PPP, distinguishes policy construction from final selection, and distinguishes temporary relief from corridor-level recovery. That makes it possible to ask a more useful clinician-facing question: where in the viability pipeline is the patient's action field failing?

In plain terms, the paper's claim is this: some depressive shutdown states are best understood not as bad final choice, but as upstream corruption of the field from which choice is made. For CBT practice, that matters only if the model improves case formulation and intervention selection. The paper therefore stays bounded. It focuses on cases in which actual capacity remains materially above perceived capacity, and it treats the model as a conceptual practice aid rather than a complete diagnostic theory.

## **2. Scope and claim discipline**

### **2.1 What this paper claims**

- A major subset of depressive shutdown can be modeled as distorted PPP, distorted action-outcome expectation, and policy-space contraction.
- Behavioral withdrawal can arise upstream of final selection because candidate policies are screened out before explicit comparison as too costly, too risky, too exhausting, or too hopeless.

- Retrospective reappraisal and cognitive restructuring can function as evidentiary correction rather than as generic positive self-talk.
- Behavioral activation can function as both state-changing action and capability-relevant evidence generation.
- Recovery is better modeled as reopening a recovery corridor than as feeling better in the moment.

## 2.2 What this paper does not claim

- It does not provide a full theory of depression in all diagnostic forms.
- It does not replace biological, pharmacological, developmental, trauma-based, or social explanations.
- It does not claim that every depressive presentation is primarily PPP distortion.
- It does not claim theorem-level closure or full empirical validation.
- It does not claim that any positive action or improved feeling automatically constitutes recovery.

## 3. Minimal PV–PP architecture required for the argument

The relevant high-level ordering is:

PPP → Φ → H → G → R → Graph Layer / seed substrate → Π → Π completeness  
validation → Constraints → Domain Framing → Adequacy → Σ → ε

At paper level, the ordering matters because the argument is not that depressed agents simply choose badly at the end. The claim is that distortions earlier in the pipeline can prevent viable recovery-bearing policies from becoming live enough to build before any final comparison is reached.

### 3.1 PP versus PPP

Actual productive power is the person's real condition. Perceived productive power is the interpreted state from which Layer 2 operates. A person can therefore behave as though collapse is near when actual capability, support, or recoverability remain materially higher than the person currently perceives.

This distinction is clinically useful only if it is not blurred. Many depressive cases are mixed cases: real PP has deteriorated some, but PPP has deteriorated more. The model is strongest where that gap is large enough to matter. It is weaker where genuine incapacitation is so dominant that the primary explanatory burden no longer sits on interpretive distortion.

### 3.2 Pressure, horizon, and governing structure

$\Phi$  maps current pressure structure.  $H$  maps collapse-horizon compression.  $G$  identifies the governing domain set, including dependency-closed support domains where recovery necessity requires them. For the depressive cases modeled here, the governing set will often include basic maintenance together with occupational, relational, or role-continuity domains. Support domains such as usable routine, available help, medication adherence, transport, remaining health margin, or minimally functioning social access may still be present but underread.

That matters clinically because a painful domain may dominate salience without being the whole governing structure. Likewise, a local gain may not restore broader viability if the governing set remains broken elsewhere.

### 3.3 Policy construction before selection

$\Pi$  is upstream of  $\Sigma$ . The patient in shutdown is therefore not always a clean chooser from a rich option set. Often the richer option set never becomes live policy structure in the first place.

In the account developed later, this occurs partly because regime-conditioned construction posture changes which policy families clear the threshold for active construction before any final comparison is reached.

### **3.4 Constraints, framing, and adequacy**

Constraints determine feasibility. Domain framing determines what function is actually being preserved. Adequacy determines whether a policy opens a genuine recovery corridor. These are not the same failure surface. Constraint hardening can make a path look impossible before adequacy is even asked, while false adequacy failure arises downstream when an already narrowed and misframed field is treated as corridor-empty.

## **4. Depressive shutdown as structural miscalibration**

### **4.1 False crisis posture**

The pathology examined here is not generic negativity. It is a structured miscalibration in which the system's reading of capability, burden, threat, and recoverability becomes distorted enough that the decision architecture begins behaving as though collapse is much nearer, survivability margins are much smaller, and viable paths are much rarer than is actually the case.

When PPP falls sharply below PP, ordinary burdens begin to look catastrophic. Time horizons compress. Temporary instability becomes intolerable. Social exposure, effort, uncertainty, and delayed payoff are read as less survivable than they really are. The person begins operating in a false crisis posture: a survival-like decision posture not because all real maneuver room is gone, but because the internal model of maneuver room has collapsed.

## 4.2 Action-outcome mislearning

Expectation-state  $K_i(t)$  matters here. The person may cease to treat action as capable of meaningfully changing trajectory. Repeated disappointment, demoralization, or biased update patterns can teach the system that effort does not pay, bridge periods do not lead anywhere, and costly exposure is structurally pointless. That is not identical to current-state distortion, but it compounds it.

## 4.3 Policy-space contraction

Policy-space contraction should not be treated as one unanalyzed event. Distorted PPP lowers perceived survivability and inflates perceived cost. H and R then convert that picture into compressed bridge tolerance and a more defensive operating posture. Under that more defensive posture, Graph /  $\Pi$  raises the practical threshold for activating exposed, delayed-payoff, bridge-dependent, substitute-function, and staged-recovery families. In paper terms, the threshold is best understood as a higher survivability-and-corridor-relevance requirement for licensing a policy family into active construction. Families that no longer look sufficiently survivable, timely, or recovery-relevant are less likely to enter the live candidate field at all.

## 4.4 False adequacy failure

False adequacy failure is downstream of that narrowed field. Once the person is operating over an underbuilt, hardened, and often misframed candidate set, the world can begin to appear corridor-empty even where a broader and more accurate field would still contain recovery-capable structure. Hopelessness is therefore intelligible here as false absence of visible adequacy, not merely as mood language.

## 5. Worked architectural trace: localized shutdown through Layer 2

The central claim is stronger if the distortion is shown moving through the pipeline rather than only described in prose. The trace below remains a paper-level propagation account, not an operator-level derivation. Its purpose is narrower: to show which stages mainly pass through upstream distortion, which stages transform it into a narrower action field, and where distinct clinician-facing consequences emerge.

Illustrative case. A patient retains meaningful work capacity, intact basic social access, and recoverable daily structure, but has recently experienced repeated setbacks and now interprets effort as highly risky, bridge periods as intolerable, and delayed payoff as largely unavailable.

PPP. Current capability is read as lower, more fragile, and less recoverable than it is. This is the primary upstream defect.

$\Phi$ . Pressure is mapped more heavily onto effort, uncertainty, exposure, and bridge cost. In many depressive cases  $\Phi$  is mainly an expression surface here rather than an independent pathology.

H. Compressed perceived margins make bridge periods and delayed payoff look less survivable. H does real work by converting low-capability interpretation into shorter apparent tolerable horizons.

G. Emotionally painful domains dominate salience while support or dependency domains such as routine, accessible help, or still-usable role continuity are underread. The governing set may therefore be represented too narrowly or with missing support structure.

R. Compressed horizon and elevated perceived pressure push the system toward a more defensive operating posture.

Graph / Π. Exposed, delayed-payoff, substitute-function, bridge-dependent, or staged-recovery families are underactivated or not retained because the PPP-plus-regime picture no longer licenses them as sufficiently survivable or timely. Withdrawal is localized primarily as upstream construction narrowing, not merely bad final choice.

Constraints. Conditional and soft barriers are experienced as hard impossibilities. Even among the remaining candidates, the field tightens further.

Domain Framing. PPP distortion and underread support structure can narrow what is taken to be the function to preserve, while hardened barriers around substitute paths make one threatened mechanism or role look like the whole governed function.

Adequacy. The narrowed and misframed field appears corridor-empty. Hopelessness becomes structurally intelligible as false absence of visible adequate paths.

Σ and ε. Selection and execution act on an already corrupted field. The pathology is largely upstream of final choice.

The strongest clinician-facing implication of the trace is simple: the same outward withdrawal can arise from different internal failure surfaces. That is the point at which case formulation becomes intervention-relevant.

## **6. Differential case formulation: $M_i(t)$ versus $K_i(t)$**

The paper becomes more than CBT relabeling only if similar-looking cases can be assigned to different internal failure surfaces with different intervention implications. The most important split here is between contaminated remembered evidence  $M_i(t)$  and expectation-driven learned futility  $K_i(t)$ .

Case A: memory-dominant impossibility narrative. A past failure, rejection, humiliation, or betrayal episode is recalled in contaminated or overgeneralized form and now dominates

present interpretation. The patient says, in effect, this happened before, therefore this path is structurally dead now. The intervention emphasis is retrospective reappraisal, contradiction of contaminated recall, externalized reconstruction of what actually happened, and disciplined separation of one event from the broader action field.

Case B: expectation-dominant learned futility. The patient may remember prior attempts accurately enough, but  $K_i(t)$  has generalized from them into a broader rule that effort is pointless, bridge periods never pay, and action will not alter trajectory. The intervention emphasis is prediction-error-generating action, bounded behavioral activation, graded task entry, and classification of outcomes in a way that prevents one failed trial from being globalized into total impossibility.

The clinical surface may look similar in both cases: nothing will work, I cannot do it, there is no point. Architecturally, however, the repair route differs. If the primary defect is  $M_i(t)$ , memory correction is central. If the primary defect is  $K_i(t)$ , prediction-error-generating action is central.

A second distinction is equally important for practice: actual PP loss versus PPP distortion. Two patients may both present as unable to work, unable to socialize, and unable to tolerate effort. In one case, severe sleep deprivation, medication disruption, psychomotor retardation, and somatic depletion may mean actual capacity is sharply degraded. In the other, actual capacity may be reduced but still materially usable, while perceived capacity has collapsed further. Those are not the same case. The first demands heavier respect for real limitation; the second demands more aggressive correction of underread usable capacity.

A third distinction is constraint hardening versus false adequacy failure. In constraint hardening, the patient experiences soft, conditional, or staged barriers as hard impossibilities. In

false adequacy failure, the patient may no longer see any recovery-bearing corridor even after barriers are reconsidered, because the candidate field has already been narrowed and misframed upstream. The first often calls for barrier reclassification; the second often calls for rebuilding the field itself.

## **7. Retrospective reappraisal and evidentiary correction**

The architecture supports a stronger reading of cognitive correction than generic changing of thoughts. Evidentiary correction can target different internal objects for different reasons.

Present-state correction targets PPP through current evidence, reinterpretation, and clarification. Its main structural effect is reduction of false pressure and false crisis loading.

Memory correction targets  $M_i(t)$  through reappraisal of past events, contradiction of contaminated recall, and structured reconstruction. Its main structural effect is weakening distorted trust, failure, or impossibility narratives.

Expectation correction targets  $K_i(t)$  through prediction-error handling and revised success models. Its main structural effect is restoring action-outcome mapping and bridge visibility.

Constraint reclassification targets PPP plus framing by recognizing that soft or conditional barriers are not hard impossibilities. Its main structural effect is reopening candidate policy classes that were excluded too early.

Retrospective reappraisal therefore has a clear architectural role. It can correct contaminated encoded evidence, reduce unjustified confidence in false remembered structure, and weaken globalized inferences such as I always fail, that path never works, or this cost always destroys me. The point is not reassurance. The point is input repair.

Externalized methods such as journaling, structured questioning, truth-focused dialogue, written decision records, and guided review of prior attempts matter because they expose hidden assumptions, disentangle domain structure, slow affectively compressed interpretation, and make contradictory evidence harder to suppress.

## **8. Behavioral activation as recovery re-entry**

Behavioral activation is the strongest practical bridge in the paper because it is not only interpretive. It is evidentiary and state-changing. A bounded action can generate real local PV while also supplying information the shut-down system was not receiving.

### **8.1 Why small actions can matter**

A small successful action can disconfirm total incapacity, weaken a false hard constraint, restore some action-outcome mapping, and reveal that a bridge period is survivable. That is why a modest step may carry disproportionate structural value in a severely narrowed action field.

### **8.2 Execution-contingent capability revision**

People often enter an interaction under provisional sufficiency, not total certainty. They begin a task because current PPP is sufficient to enter a bounded execution corridor, not because every capability requirement is already known and satisfied. During execution they may discover that they can continue, need assistance, should stage the work, or should abort and re-enter decision.

This matters clinically because behavioral activation need not assume the patient already knows they can complete the larger task. What matters is that the patient can enter a survivable corridor in which capability may be confirmed, augmented, revised, or partially degraded. Recovery re-entry is often staged in exactly this way.

### 8.3 Downward execution revision and failed activation

The downward branch matters because it can deepen shutdown if the failure is globalized rather than correctly classified. A patient enters a bounded task, fails halfway for reasons not modeled in advance, and then relearns from the failure in the wrong way: PPP drops further,  $K_i(t)$  generalizes futility, and future Graph /  $\Pi$  activation narrows more aggressively.

This yields a practical prediction: poorly calibrated activation tasks can worsen shutdown when their failure is globalized rather than correctly classified. The point is not that behavioral activation is unsafe in general. The point is that task calibration, staging, and interpretation are part of the mechanism.

Activation therefore is not magic. A corrective action must be survivable, interpretable, function-relevant, and strong enough to alter the system's reading of what is possible. Tasks that are too large, too ambiguous, or too weakly connected to a real recovery corridor can fail because the patient cannot classify the result well enough for it to become usable evidentiary correction.

## 9. Recovery corridor reopening versus relief

The framework is useful here because it refuses a sloppy equation of feeling better with recovering. At least four structurally distinct states must be kept separate.

- Temporary relief: subjective pressure reduction without durable structural reopening.
- Local improvement: one domain improves while the governing set remains globally unresolved.
- Bridge support: a survivable transitional state that can carry the patient toward later recovery.

- Recovery corridor reopening: a policy path that preserves viability until recovery becomes reachable and then supports joint sustainment across the governing set.

For CBT practice, this distinction matters because an intervention can lower distress without rebuilding a path the patient can actually live in. The practical question is not only whether the patient feels some relief. It is whether the action field has reopened enough to support viable forward movement.

## 10. Clinical contribution and implications for CBT practice

This paper overlaps strongly with familiar clinical ideas. Beck's work on cognitive distortions maps onto systematic interpretive error (Beck, 1976). Bandura's work on self-efficacy maps onto capability-conditioned action gating (Bandura, 1977). Learned helplessness maps onto one major class of  $K_i(t)$  distortion (Abramson, Seligman, and Teasdale, 1978). Behavioral activation maps onto evidence-producing re-entry into action (Dimidjian et al., 2006). The contribution claimed here is narrower and should stay narrower.

The contribution is not that PV-PP discovers these phenomena. It is that the architecture orders them. It distinguishes current-state distortion from memory distortion, expectation mislearning from present-state underestimation, policy construction failure from final-selection failure, barrier hardening from false corridor absence, and temporary relief from corridor reopening. That ordering gives CBT case formulation a tighter way to explain why patients who sound similar may need different intervention emphases.

The practical implication is straightforward. When a patient says nothing will work, the therapist should not treat that statement as diagnostically exhausted by its surface form. The question is what kind of impossibility claim is being made. Is the patient misreading present capability? Overgeneralizing remembered failure? Modeling action as globally futile? Treating

conditional barriers as hard impossibilities? Or seeing no recovery corridor because the candidate field has already collapsed? Each points toward a different mix of reappraisal, cognitive restructuring, evidence review, graded activation, barrier reclassification, support-domain restoration, and staged corridor rebuilding.

The model also disciplines what CBT should not overclaim. If actual PP is sharply degraded, the right response is not to push confidence language or aggressive activation as though the problem were merely interpretive. If the governing set includes medication adherence, housing stability, transport, or basic sleep, then treatment planning that ignores those support domains will misread the failure surface. The architecture is useful only if it protects against that kind of conceptual sloppiness.

The paper therefore should not claim predictive triumph. Its current contribution is mechanism discrimination for practice-oriented case formulation. If later work shows that symptom-similar cases divide into different failure surfaces with different intervention responses, that would be the stronger empirical result. The present paper is the conceptual step that makes that question cleaner.

## **11. Limits**

- The model does not provide a complete diagnostic theory of depression.
- It does not encode the full biology of affective disorders.
- It does not yet distinguish all depressive subtypes or severity gradients.
- It does not establish empirical efficacy of any therapy by itself.
- It should presently be read as a structural account of one important pathway into shutdown and one important pathway out.

The model is most plausible where the person retains meaningful real capability yet behaves as though recovery is structurally absent, where bridge intolerance is pronounced, and where action-outcome expectancy appears distorted. It is less plausibly primary where biological disturbance or severe global incapacitation is so dominant that the main explanatory burden no longer sits on PPP distortion,  $K_i(t)$  distortion, or policy-space contraction.

## 12. Conclusion

The strongest defensible conclusion is narrower than a general theory of depression but stronger than a loose CBT analogy. For a bounded class of depressive shutdown cases, the PV-PP architecture provides a usable CBT case-formulation model for locating where the action field is failing.

Its main value is failure-surface discrimination. It separates distorted PPP from actual capacity loss, contaminated remembered evidence from expectation-level learned futility, hardened barriers from false corridor absence, and temporary relief from genuine corridor reopening. Those distinctions matter because they imply different correction routes. Some cases call primarily for evidentiary correction of present-state or remembered structure. Others require prediction-error-generating action, graded re-entry, support-domain restoration, or rebuilding of the candidate field itself.

The paper therefore asks CBT practice to become more explicit about mechanism. Depressive shutdown should not be treated as one undifferentiated condition in which every patient who says nothing will work has the same underlying problem. The more accurate claim is that superficially similar shutdown states may sit on different internal failure surfaces. If that claim is right, better case formulation should produce better targeted intervention.

The next step is empirical, not rhetorical. The model should be tested on whether it improves differentiation among symptom-similar cases, predicts which interventions are more likely to work given the dominant failure surface, and clarifies when apparent relief does or does not amount to real recovery-corridor reopening.

## **Declaration of Generative AI and AI-Assisted Technologies in the Manuscript Preparation**

### **Process**

During preparation of this manuscript, a generative AI tool was used for drafting support, restructuring, wording alternatives, and editorial revision. All substantive judgments, theoretical claims, interpretive decisions, and final approval of the manuscript remained entirely with the author. The author reviewed and edited the output carefully and takes full responsibility for the content of the submitted manuscript.

### **Disclosure Statement**

The author reports no conflicts of interest.

### **References**

Abramson, L. Y., Seligman, M. E. P., and Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology*, 87(1), 49-74.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, 84(2), 191-215.

Beck, A. T. (1976). *Cognitive Therapy and the Emotional Disorders*. New York: International Universities Press.

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E., Gallop, R., McGlinchey, J. B., Markley, D. K., Gollan, J. K., Atkins, D. C., Dunner, D. L., and Jacobson, N. S. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology, 74*(4), 658-670.