Supplemental Information

Startle magnitude: Raw scores

In general, the statistical results were more significant with the raw scores than with the T scores.

Fear-potentiated startle

The Stimulus Type (cue, ITI) x Condition (N, P) x Drug (3) ANOVA revealed no significant 3way interaction. A more specific analysis restricted to the high dose of hydrocortisone in the P condition reveal no significant Drug x Stimulus Type interaction (F(1,21) = .3, p = .59). Hence, startle potentiation to the threat cue (relative to ITI) was not affected by hydrocortisone.

Anxiety-potentiated startle

The Drug (3) x Condition (N, P, U) ANOVA revealed a Condition x Drug linear trend (F(1,21) = 8.0, p = .01). Follow up tests revealed that the high hydrocortisone treatment increased ITI startle in U compared to N (Condition x Drug: F(1,21) = 8.0, p = .01) and in N compared to P (Condition x Drug: F(1,21) = 6.5, p = .018).

Subjective anxiety, state anxiety, and pain

The subjective anxiety ratings were not affected by drug treatment (Table S1). They were analyzed in an analogous manner as the startle data.

Fear: As expected, based on prior data, subjective anxiety to the cues (relative to ITI) was greater in the P condition, where cues reliably signaled threats, as compared to the N condition (Stimulus Type x Condition: F(1,21) = 65.0, p < .0001, $GG-\varepsilon = .92$). This effect was not affected by hydrocortisone, as reflected by non significant Stimulus Type x Condition x Drug (F(2,42) = 1.9, ns). An analysis restricted to the high hydrocortisone in the P condition also did not reveal any effect of drug treatment (F(1,21) = 1.6, ns).

Anxiety: Like ITI startle amplitude in the current experiment and like prior results with this paradigm in other experiments, subjective anxiety increased linearly from the N to the P to the U

condition (Condition linear trend: F(1,21) = 155.5, p < .0001). This effect was not affected by drug treatment (Drug x Condition linear trend: F(1,21) = .2, ns).

State anxiety: State anxiety scores (Table S2) were analyzed using a Drug (3) x Time (4) ANOVA. There was a main Time effect (F(3,63) = 8.4, p < .001, $GG-\epsilon = .48$) due to increased anxiety from post-drug to after the first threat block (F(1,21) = 19.9, p < .0009). The Drug x Time interaction was not significant (F(1,21) = .9, ns), indicating that hydrocortisone did not affect baseline state anxiety.

Pain: Pain rating of shock did not differ significantly among treatments (F(2,40) = .3, ns). The ratings were 6.5 (SEM = .34), 6.4 (SEM = .30), 6.6 (SEM = .31) in the placebo, low hydrocortisone, and high hydrocortisone treatments, respectively.

Table S1. Mean (SEM) retrospective rating of anxiety during the cue and ITI across treatments

 and conditions

	Neutral		Predictable		Unpredictable	
	ITI	Cue	ITI	Cue	ITI	Cue
Placebo	1.6 (.2)	1.5 (.2)	3.4 (.4)	5.8 (.5)	6.0 (.5)	6.2 (.5)
Hydrocortisone (20 mg)	1.4 (.2)	1.6 (.2)	3.6 (.4)	5.7 (.5)	6.1 (.4)	6.1 (.5)
Hydrocortisone (60 mg)	1.8 (.2)	1.8 (.2)	3.6 (.4)	6.4 (.4)	6.5 (.5)	6.7 (.5)

Table S2. Mean (SEM) state anxiety at three time points during testing

	Pre-drug	Post-drug	After 1st threat series	After 2 nd threat series
Placebo	30.3 (2.2)	29.5 (1.8)	35.1 (2.9)	33.8 (3.0)
Hydrocortisone (20 mg)	28.5 (1.6)	28.9 (1.0)	32.2 (1.5)	32.4 (1.5)
Hydrocortisone (60 mg)	32.7 (1.5)	29.7 (1.2)	36.2 (1.8)	34.1 (1.8)