Synthesis of endogenous catecholamines

First, you 3-hydroxylate tyrosine to yield L-dopa. This first step is the rate-limiting step of catecholamine synthesis. It happens in the cytoplasm of the body of the neuron. When the adrenal medulla is stimulated, this tyrosine hydroxylase is the enzyme that gets phosphorylated by protein kinase C and calmodulin. Thus, to get systemic release of adrenaline in a fight-or-flight situation, tyrosine must be hydroxylated at a sufficient rate.

Then, you decarboxylate your L-dopa with a pinch of pyridoxine, which results in dopamine. This also happens in the neuronal body cytoplasm. The resulting dopamine is then sucked up into storage vesicles.

The vesicles are then slowly transported towards the synapses by axoplasmic flow.

The dopamine is beta-hydroxylated to form noradrenaline inside the storage vesicles. The noradrenaline then sits and waits patiently to be released into the synaptic cleft.

Not only is noradrenaline synthesised de novo, it is also replenished by reuptake from the synaptic cleft.

Noradrenaline is N-methylated to form adrenaline in the chromaffin cells of the adrenal medulla. This doesn’t happen in the vesicles – noradrenaline has to leave the vesicles to be converted into adrenaline; and then it is taken up into another bunch of different vesicles.

Weirdly, the size of the adrenaline store is controlled by glucocorticoids. Glucocorticoids travel via the intra-adrenal portal venous network into the medulla, to induce the synthesis of phenethanolamine N-methyltransferase.

From Peck and Hill “Pharmacology for Anaesthesia and Intensive care” as well as the mighty “Handbook of Pharmacology and Physiology in Anaesthetic Practice” by Stoelting and Hillier. Overall, Goodman And Gilman (12th edition) were the most useful in getting straight answers.