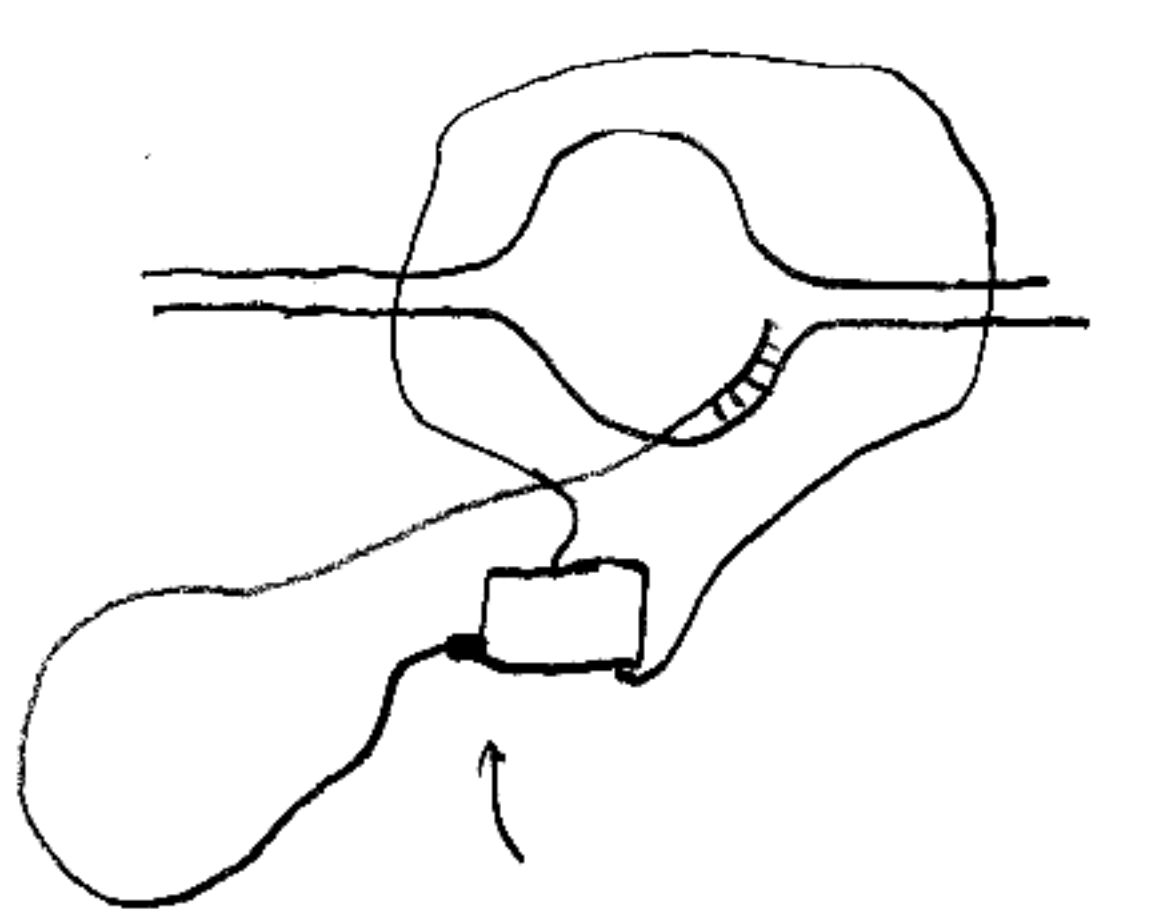
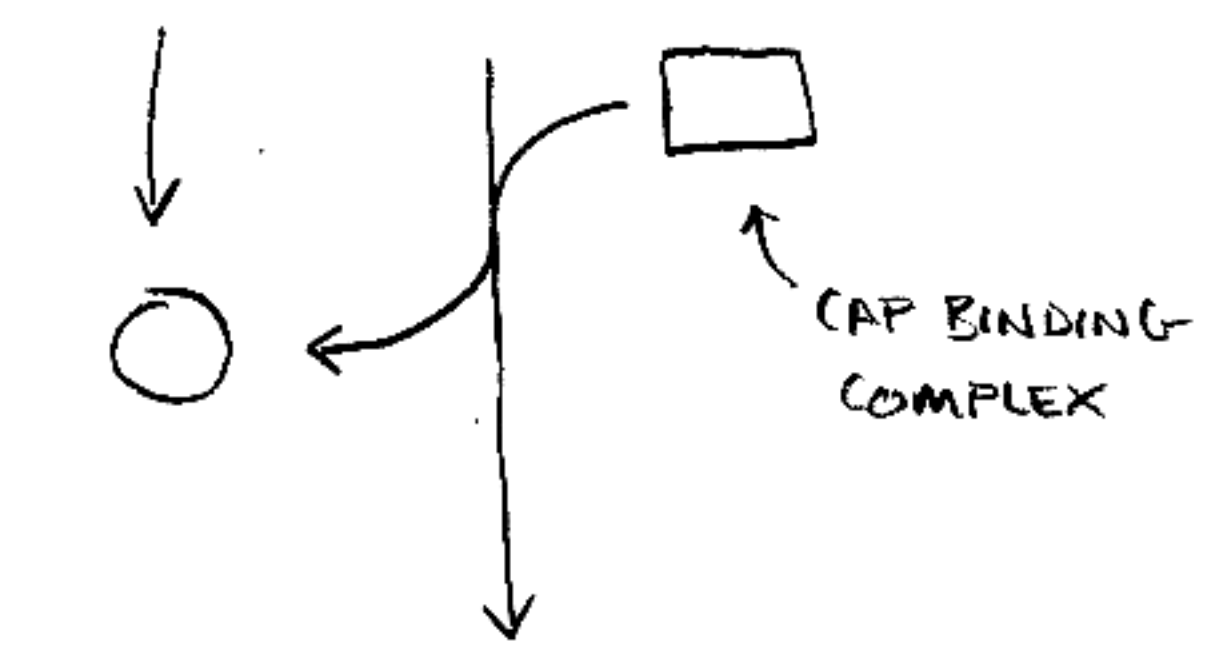
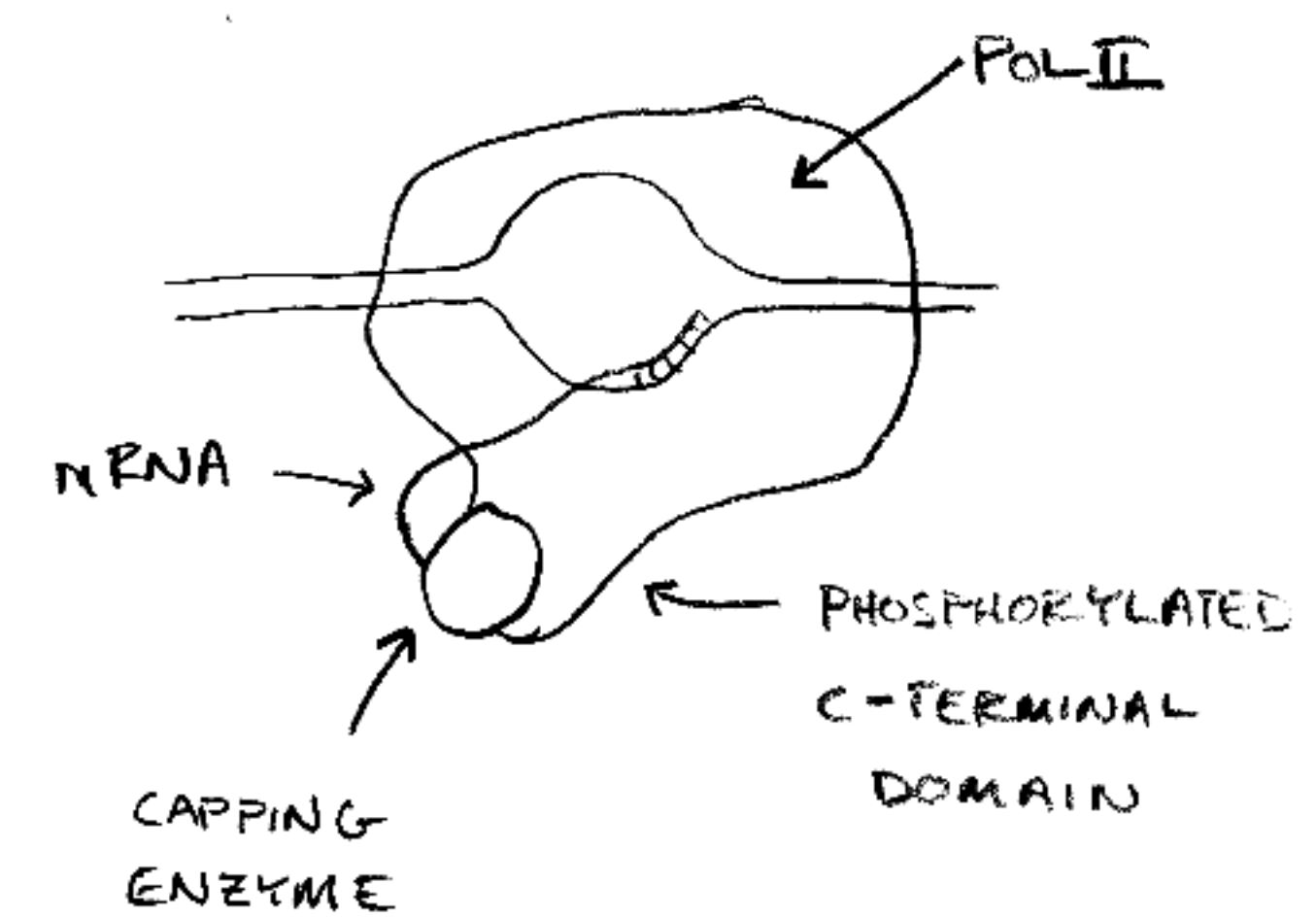


RNA PROCESSING:

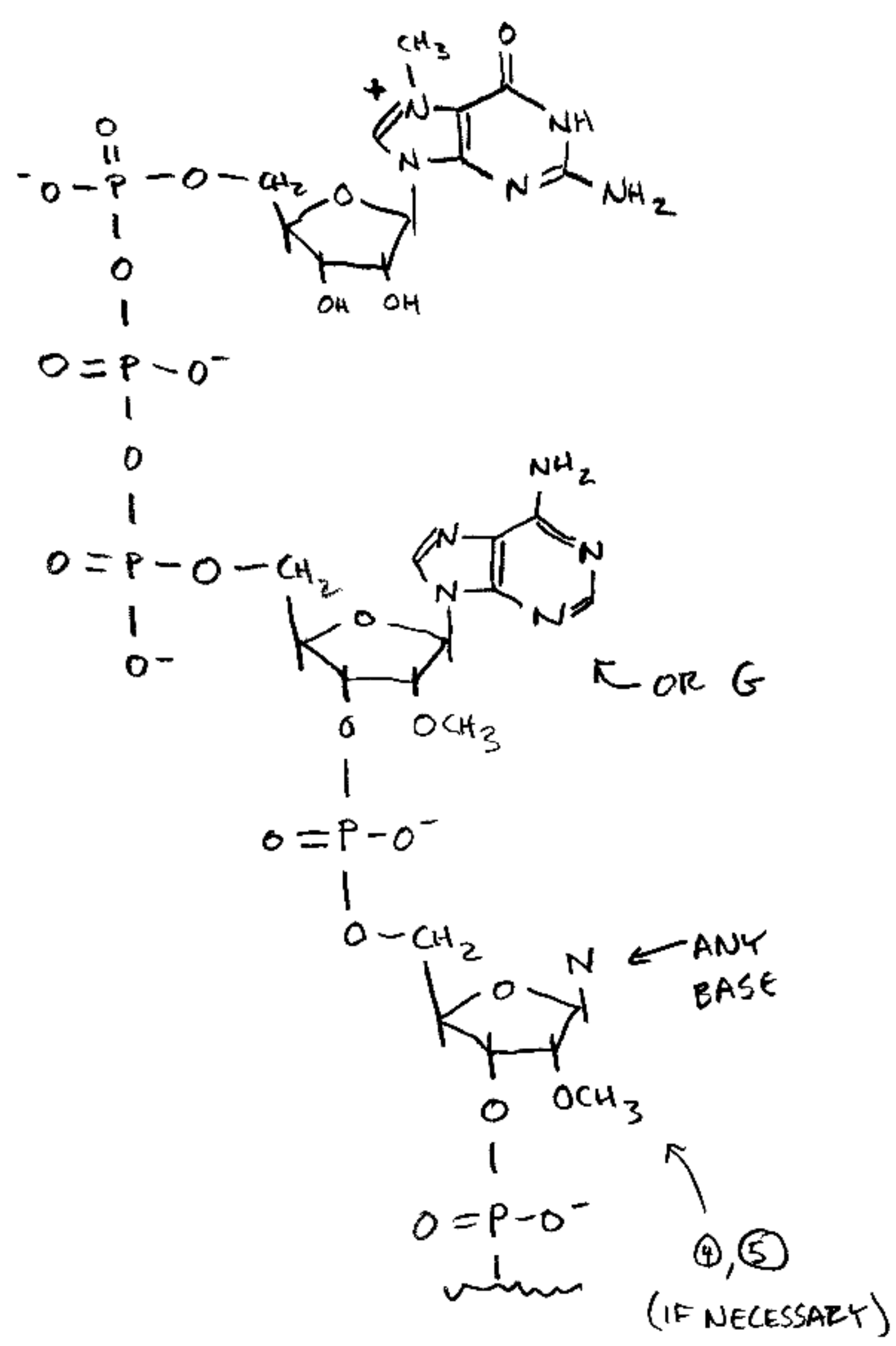
①

AS WE SAW, EUKARYOTES "CAP" MRNAs USING ENZYMES THAT ARE ASSOCIATED WITH THE PHOSPHORYLATED CARBOXY TERMINAL DOMAIN OF POL II.



5'-CAP REMAINS ASSOCIATED WITH THE POLYMERASE DURING ELONGATION

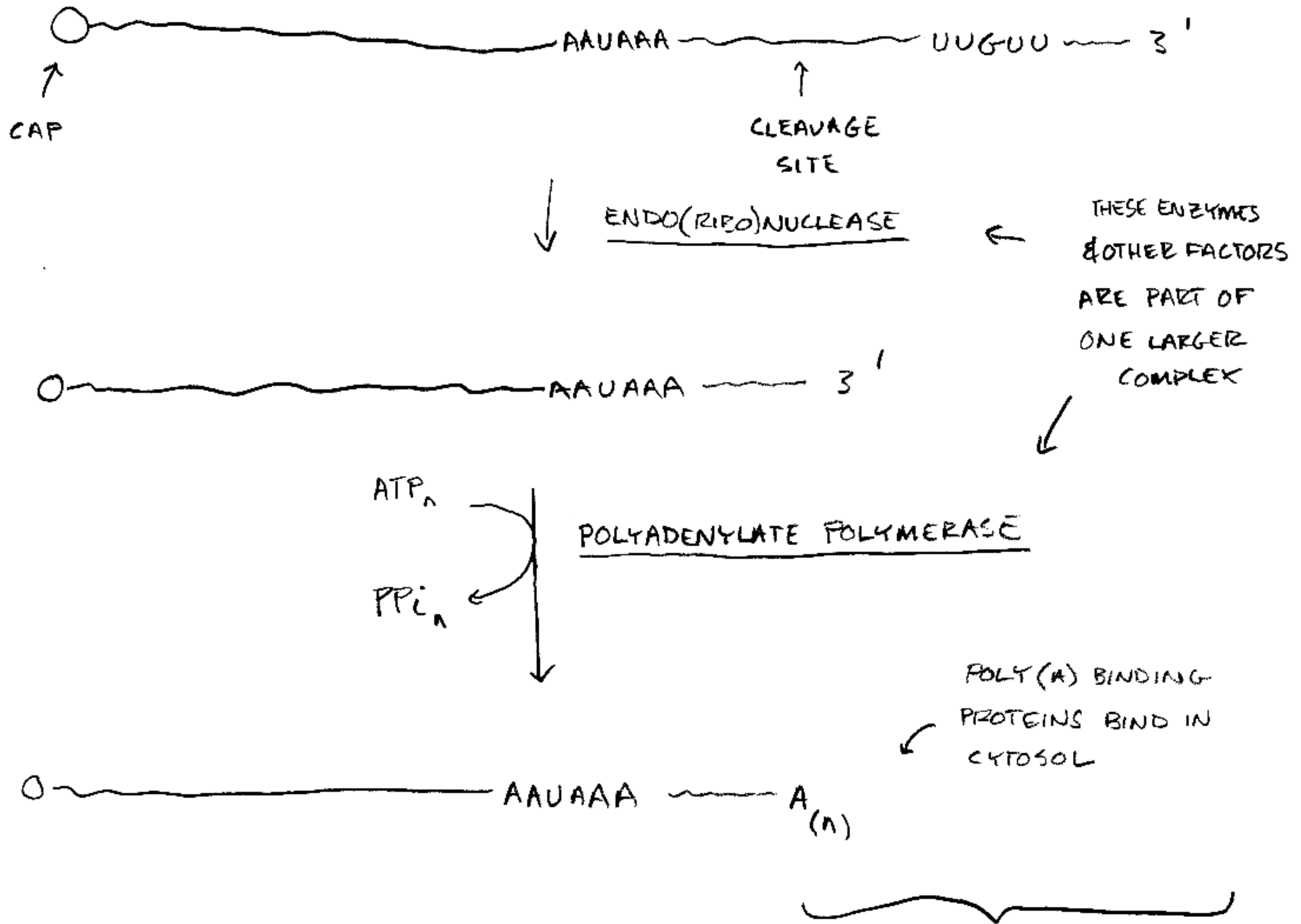
5'-CAP



- ④ RNA TRIPHOSPHATASE
- ⑤ 2'-O-METHYL TRANSFERASE
- ② CAPPING ENZYME
- ③ GUANINE-7-METHYLTRANSFERASE (S-ADENOSYLMETHIONINE)

EUKARYOTES ALSO ATTACH POLY (A) "TAILS" TO mRNA;

AFTER TERMINATION, THE NEW TRANSCRIPT IS FIRST CLEAVED AT A SPECIFIC SITE THAT IS 15 TO 25 NUCLEOTIDES PAST AN AAUAAA SEQUENCE, BUT < 50 NUCLEOTIDES BEFORE A U-RICH OR UG-RICH SEQUENCE. A POLY(A) TAIL IS THEN BUILT ON THE 3' END OF THE TRANSCRIPT.



THIS POLYMERASE CATALYZES RNA SYNTHESIS WITHOUT A TEMPLATE (NOT CODED FOR)

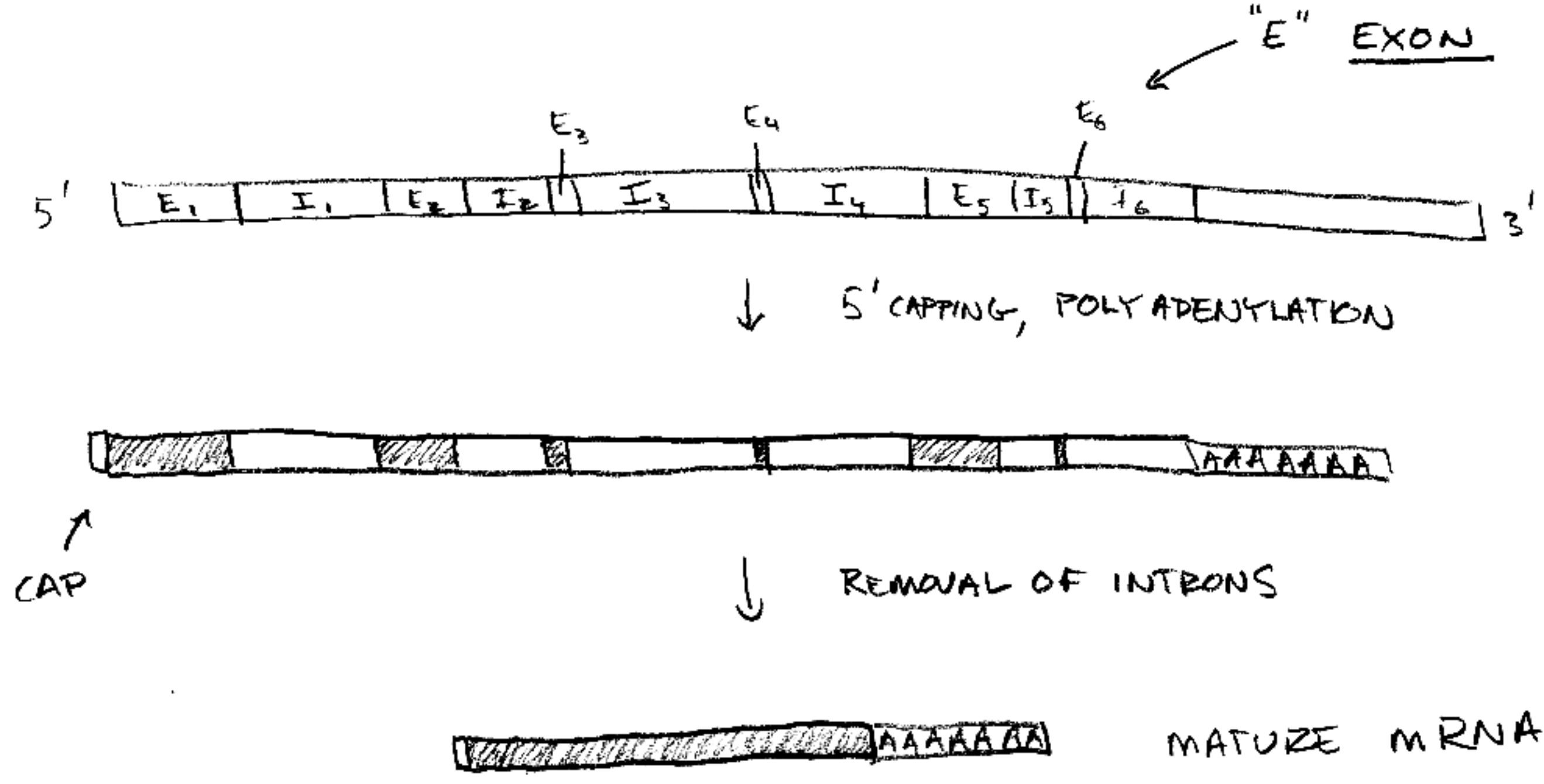
NO TEMPLATE, BUT REQUIRES PRIMER

USUALLY 80-250 A'S

MOST HISTONES LACK AAUAAA & LACK POLY(A) TAILS - SHORT LIFETIMES

REMOVAL OF INTRONS:

MOST EUKARYOTIC GENES CONTAIN STRETCHES OF SEQUENCE THAT DO NOT APPEAR IN THE MATURE mRNA. THESE INTRON SEQUENCES ARE TRANSCRIBED, BUT ARE REMOVED BEFORE THE mRNA IS TRANSLATED.



- EXONS ARE USUALLY < 300 NUCLEOTIDES LONG (~150 AVERAGE)
 (UP TO 17,106 NUCLEOTIDES IN THE 29,926 AMINO ACID TITIN PROTEIN FOUND IN MUSCLE! LARGEST KNOWN POLYPEPTIDE)
- INTRONS ARE USUALLY LONGER, ~ 3500 NUCLEOTIDES AVERAGE
 (UP TO 2.4 MILLION IN THE MUSCLE PROTEIN DYSTROPHIN!)

WHY DO INTRONS EXIST AT ALL? IT IS NOT KNOWN, MAY BE TO ENABLE ALTERNATIVE SPLICING, OR A METHOD TO FACILITATE PROTEIN EVOLUTION.

THERE ARE A NUMBER OF DIFFERENT TYPES OF INTRONS.

SELF SPLICING:

TYPE I - NUCLEAR, MT, CHLOROPLAST (MRNA, rRNA, tRNA)

TYPE II - MT, CHLOROPLAST (MRNA)

SPLICEOSOMAL (NOT SELF SPLICING):

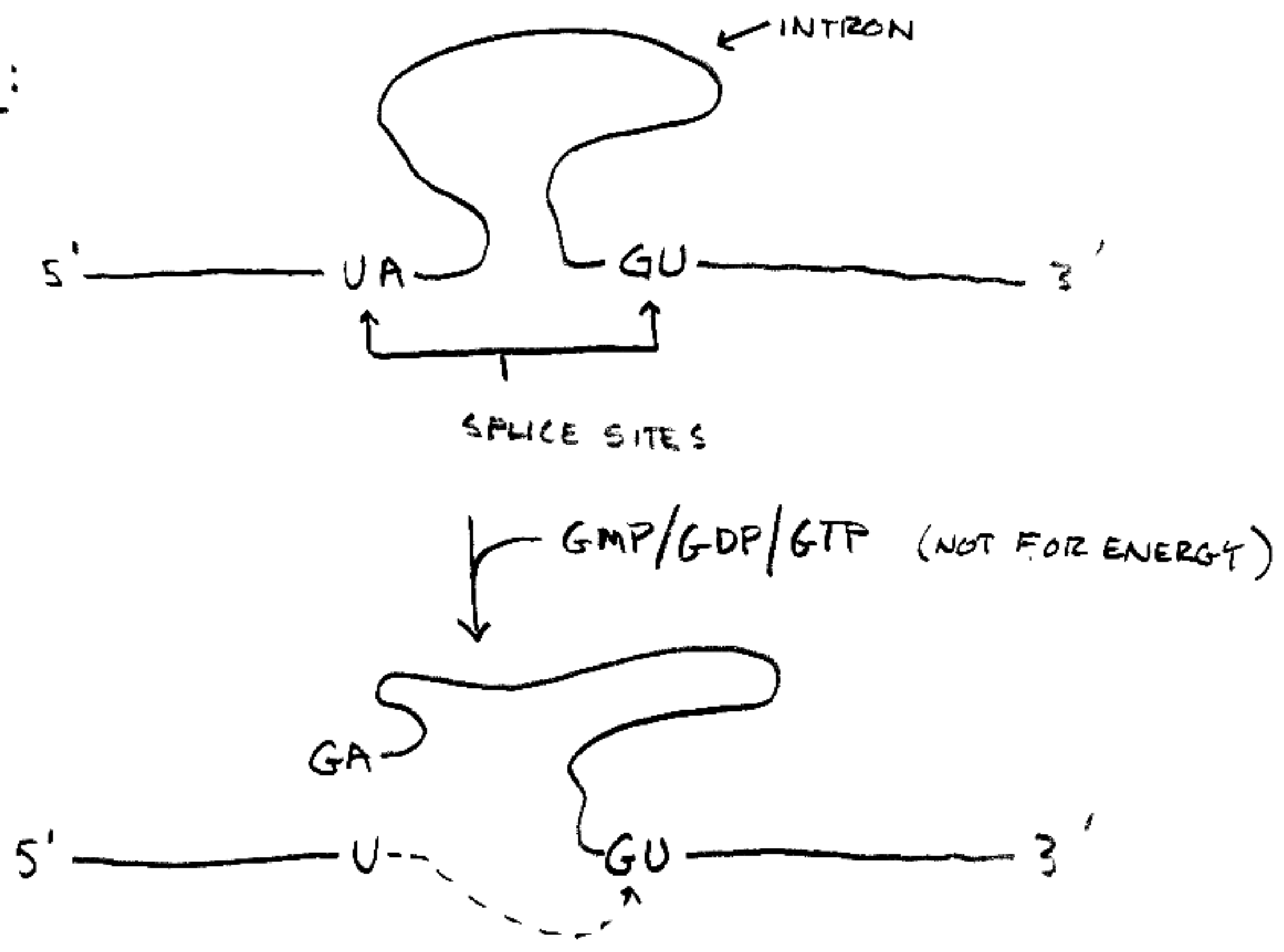
GU-AG - NUCLEAR (MRNA) - MOST COMMON

AU-AC - NUCLEAR (MRNA) - UNCOMMON

SELF SPLICING INTRONS:

BOTH TYPES ARE TRANSESTERIFICATIONS & REQUIRE NO ENERGY INPUT (NO ATP).

TYPE I:



INTRON 5' GA _____ G 3'

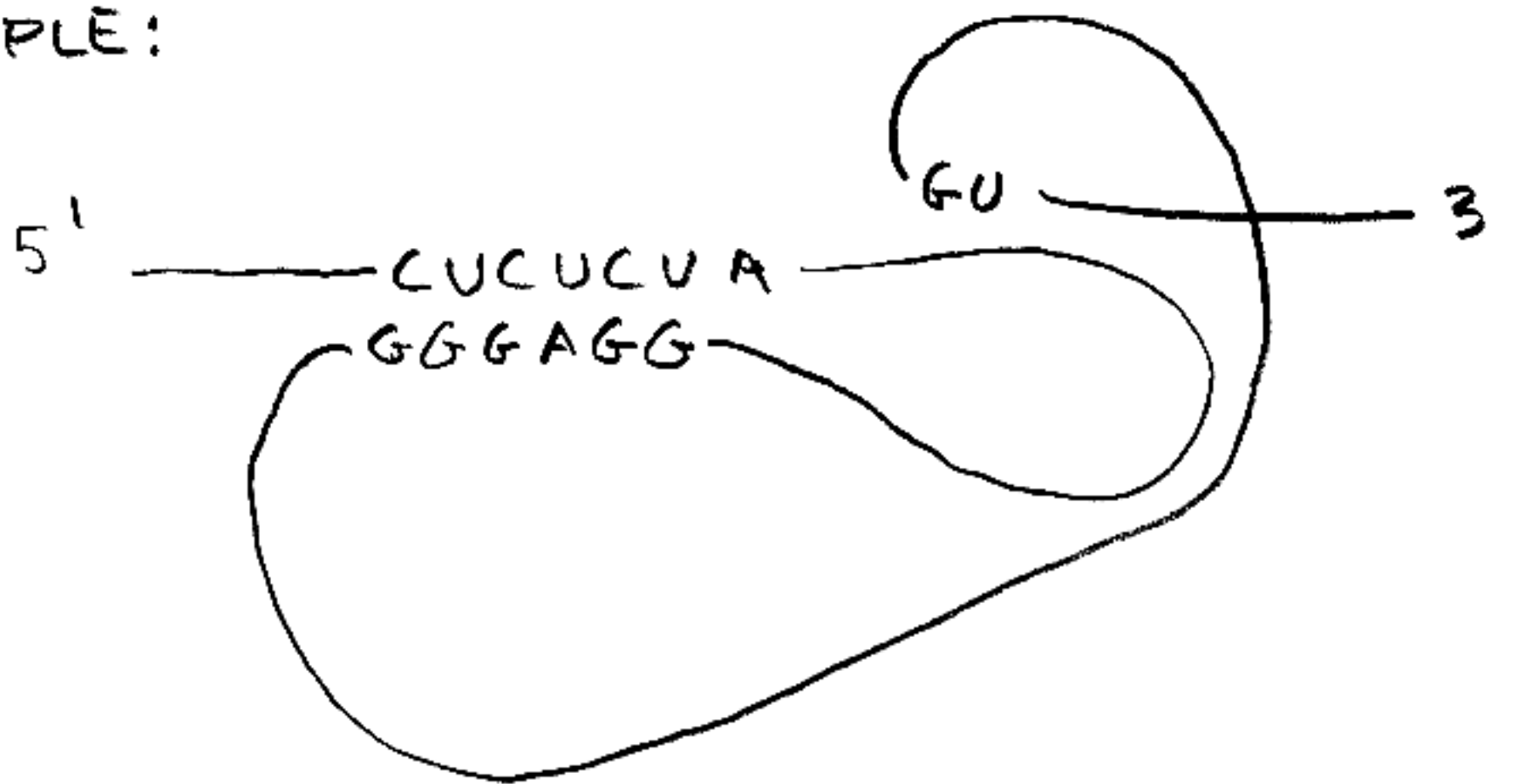
5' _____ UU _____ 3' JOINED EXONS

HOW IS THIS DONE IN THE ABSENCE OF ANY PROTEINS?

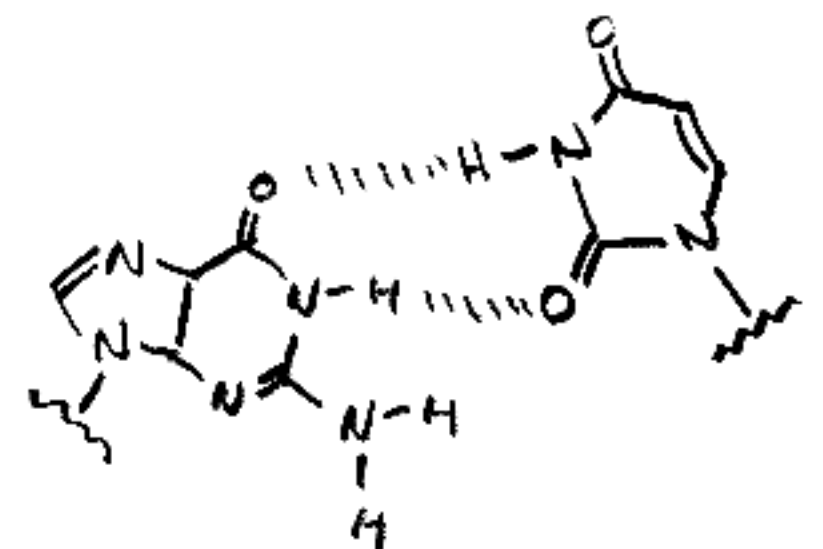
THE RNA ITSELF IS DOING ALL THE WORK

BY FOLDING A CERTAIN WAY THE RNA ACCURATELY SELF SPLICES.

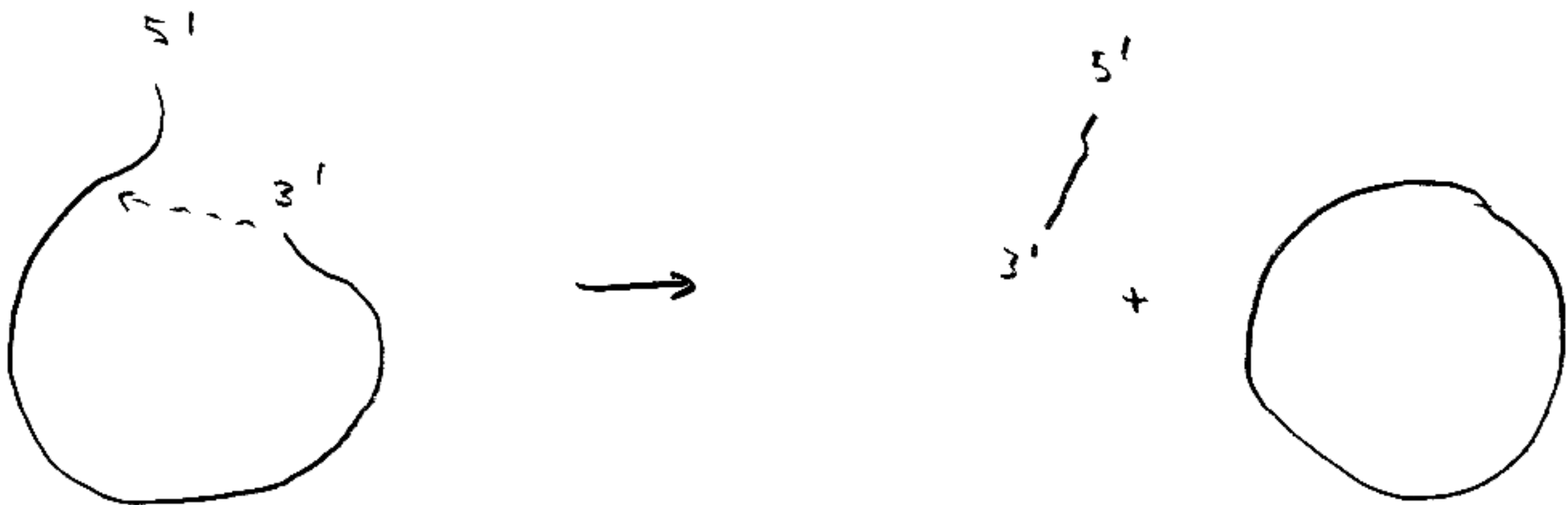
FOR EXAMPLE:



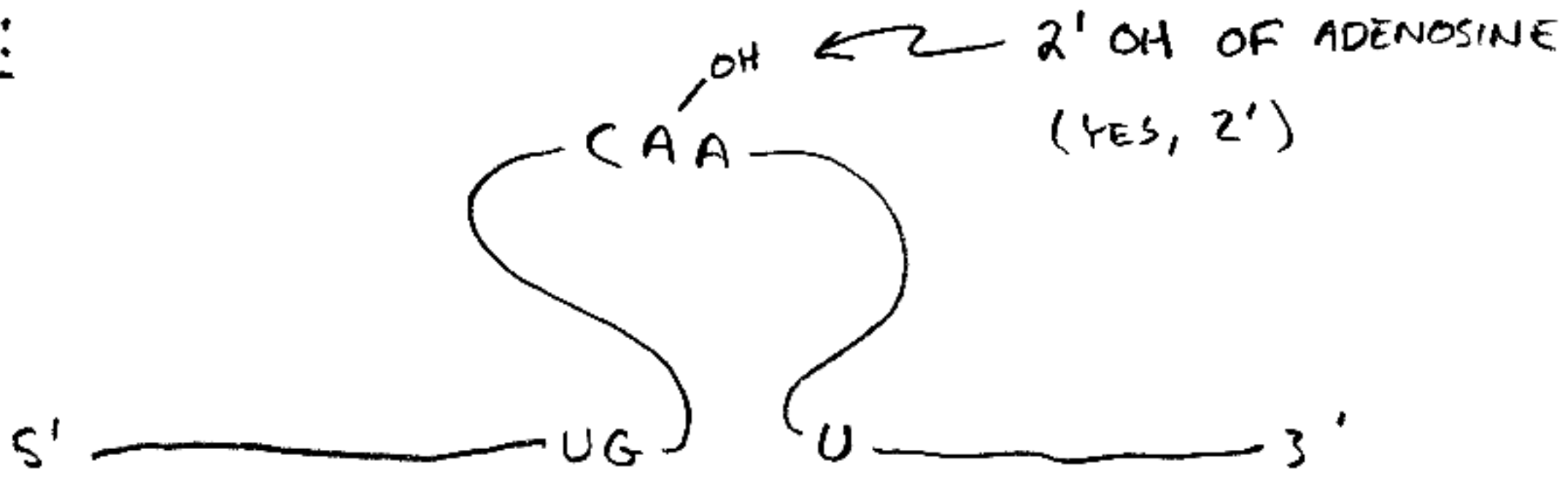
GU PAIRING:



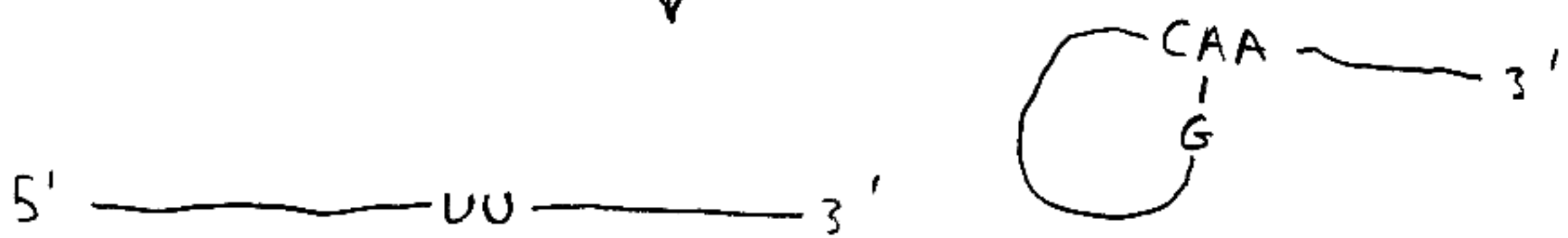
SOMETIMES THE INTRON CAN CONTINUE TO SPLICE:



TYPE II:

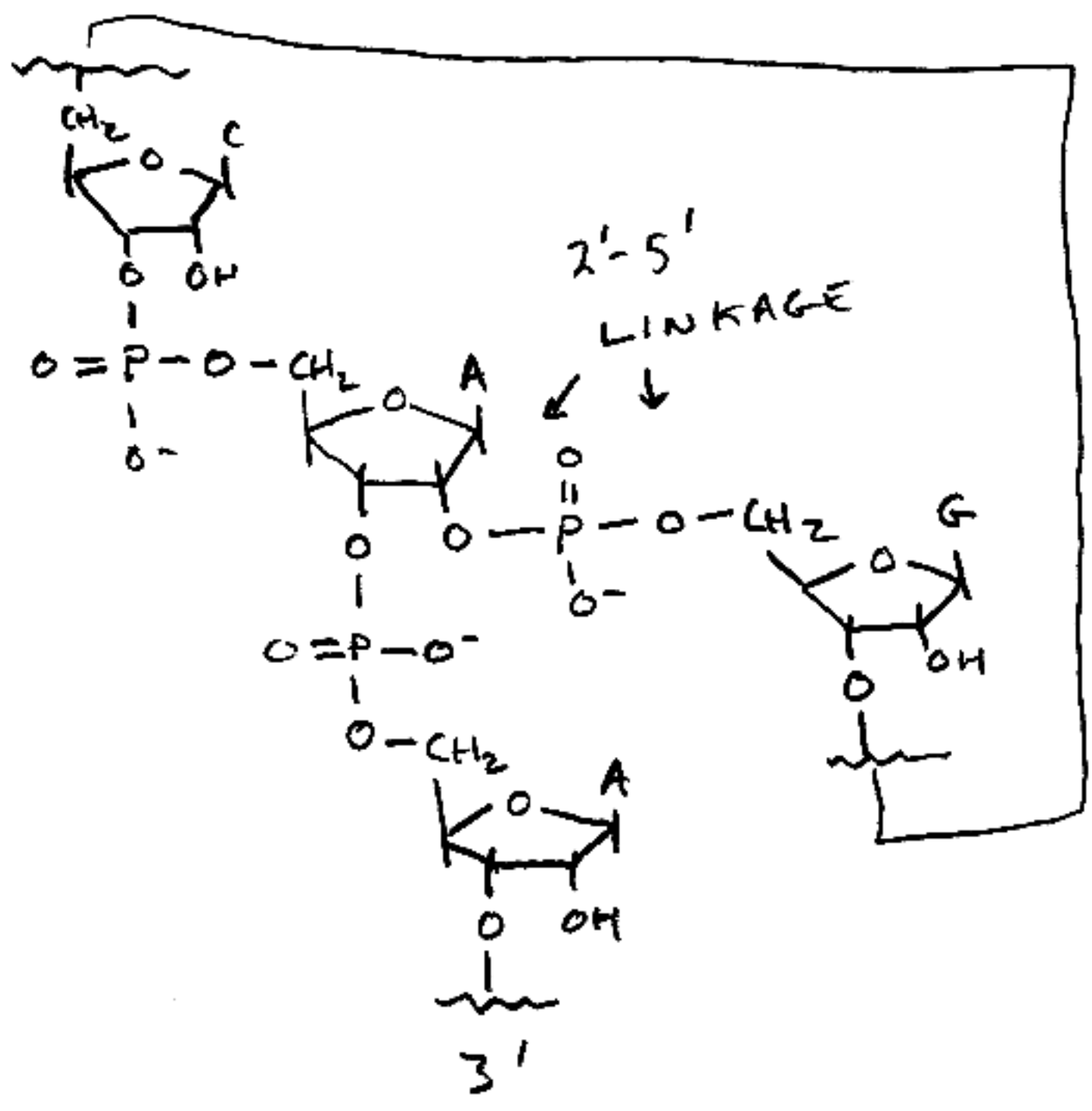
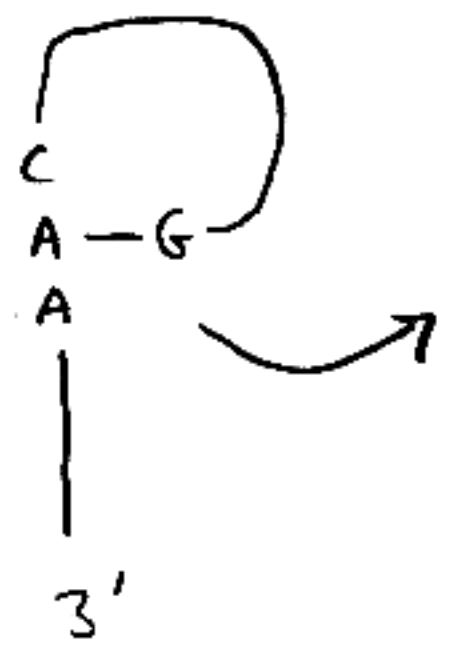


INTRAMOLECULAR REACTION



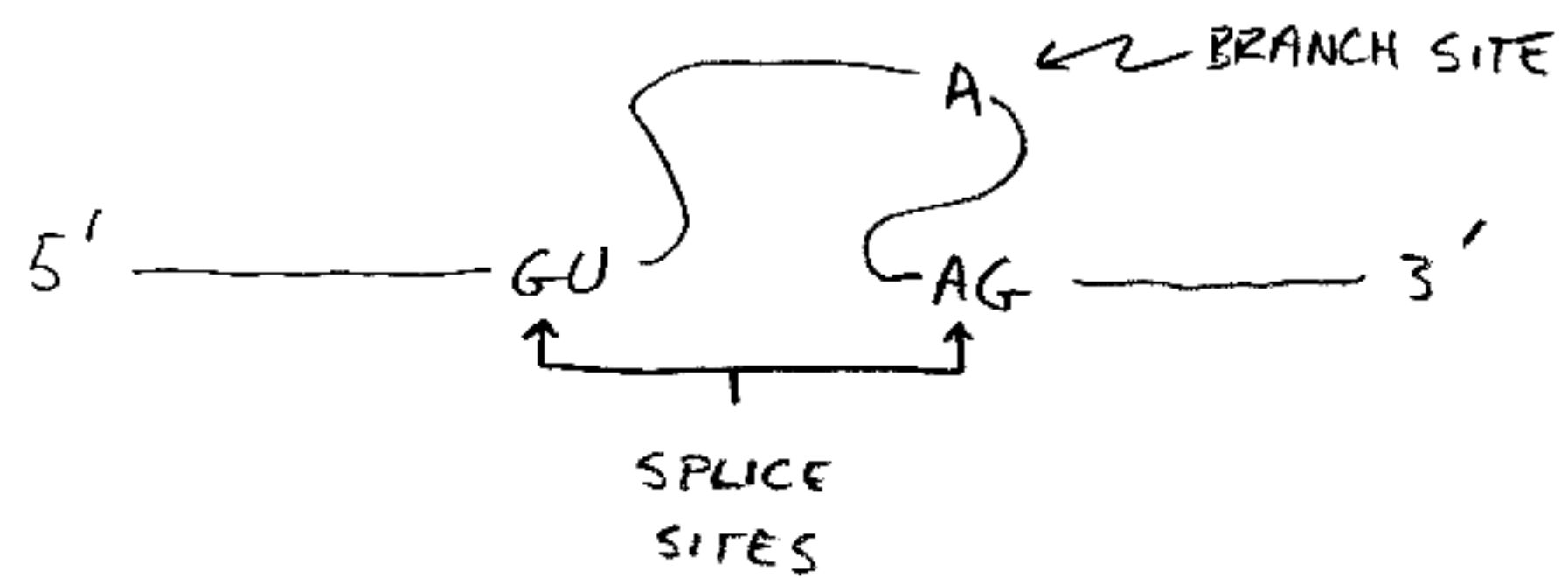
"LARIAT" STRUCTURE

WHICH HAS THE STRUCTURE:



SPLICEOSOMAL INTRONS:

MOST SPLICING OCCURS WITH THE HELP OF PROTEINS & SMALL NUCLEAR RNA'S (snRNA'S), MOST OF THESE CONTAIN GU-AG AT THE 5' & 3' SPICE SITES, RESPECTIVELY.

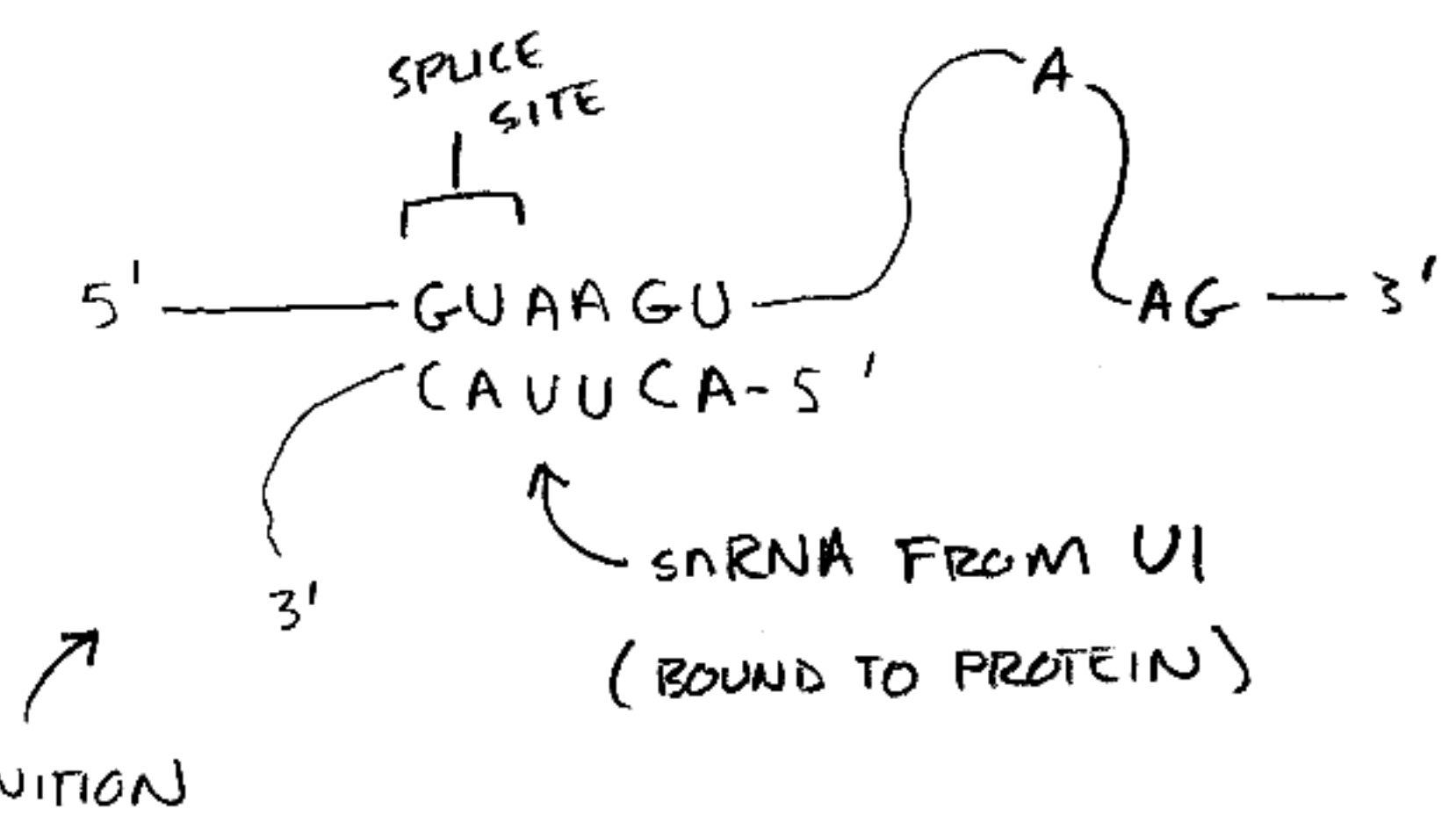


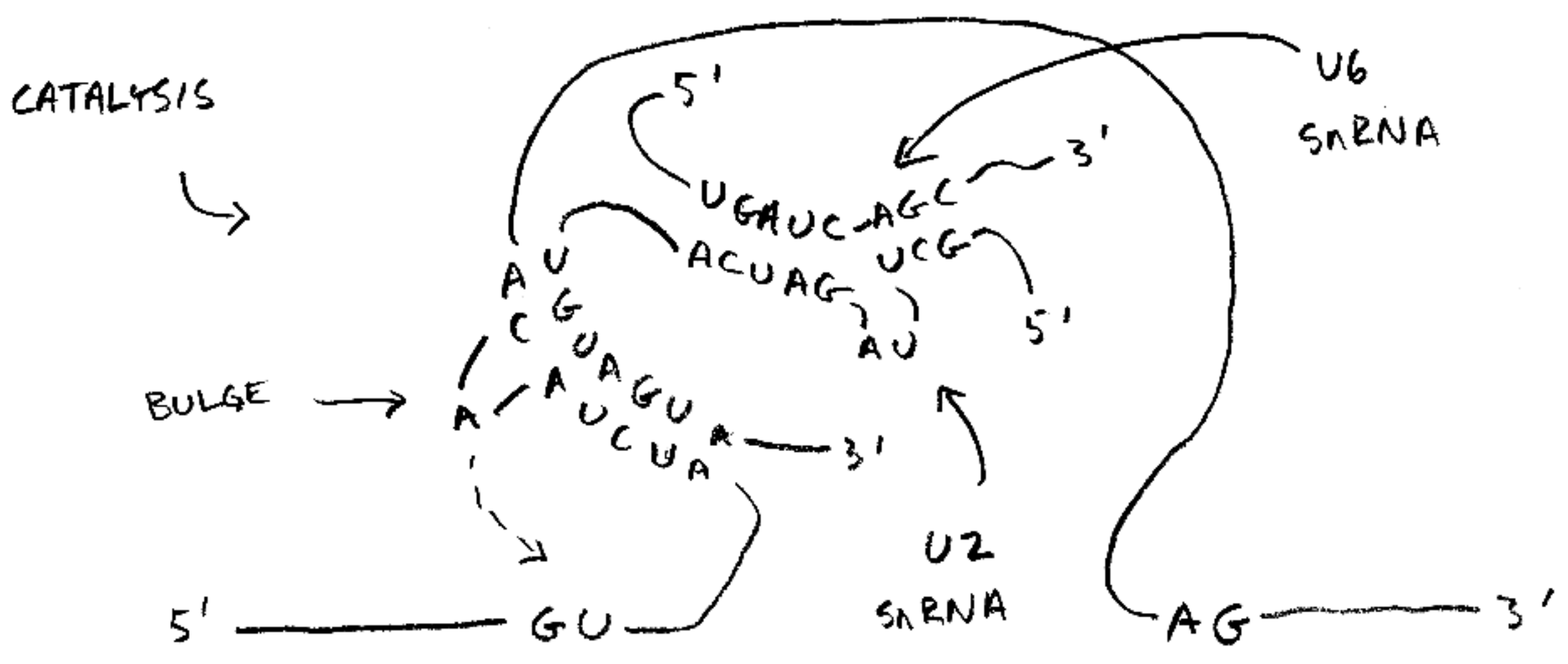
THE PROTEIN-RNA COMPLEXES INVOLVED IN SPLICING:

SMALL NUCLEAR RIBONUCLEO PROTEINS

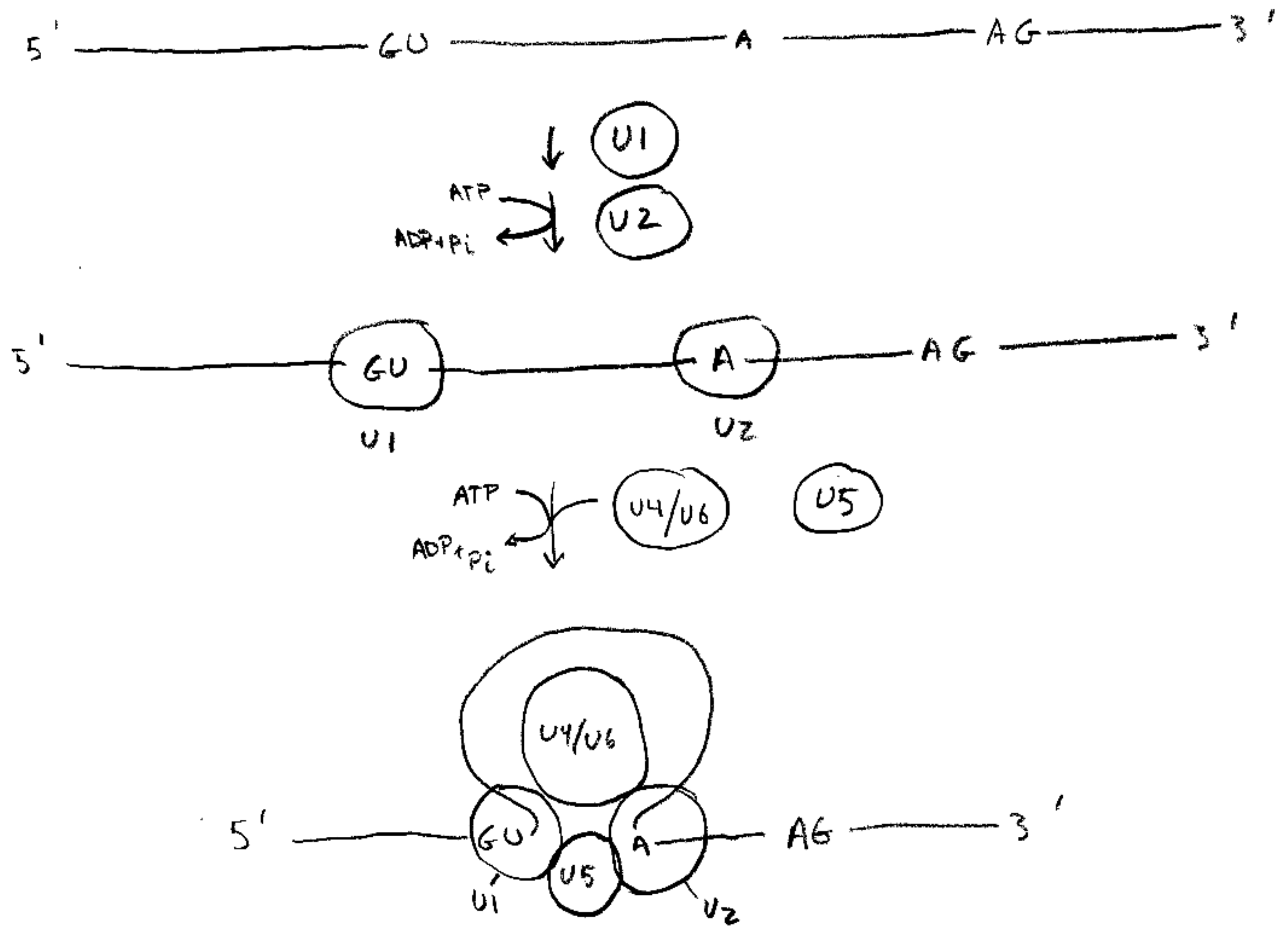
SNRNP	snRNA SIZE	FUNCTION
U1	165	BINDS 5' & 3' SPICE SITES
U2	185	BINDS BRANCH SITE
U5	116	BINDS 5' SPICE SITE
U4	145	PREVENTS PREMATURE CATALYSIS BY U6
U6	106	CATALYZES SPLICING

THE snRNA PORTION OF SNRNP'S FUNCTION TO RECOGNIZE SPECIFIC SEQUENCES & TO PARTICIPATE IN CATALYSIS

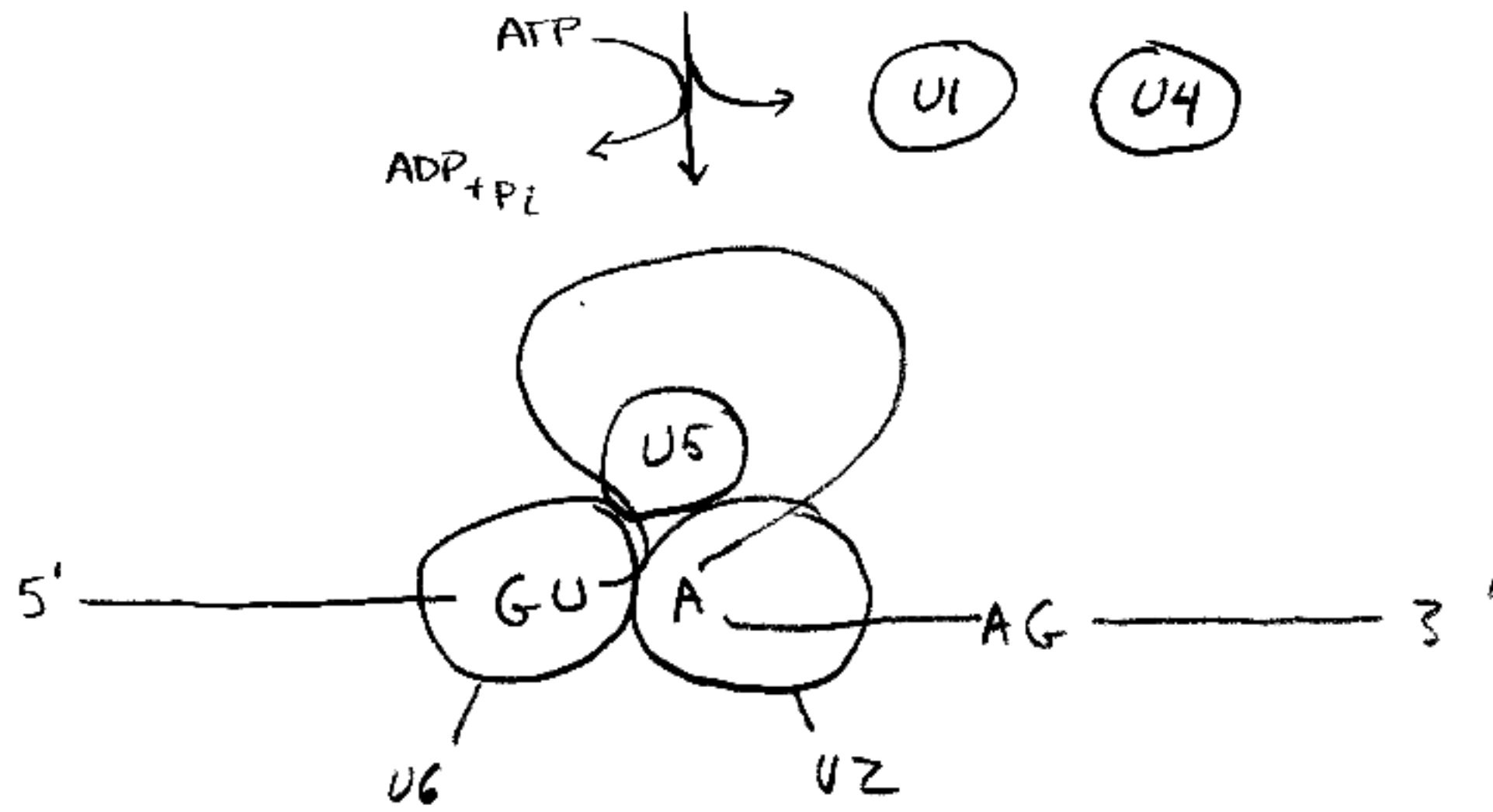




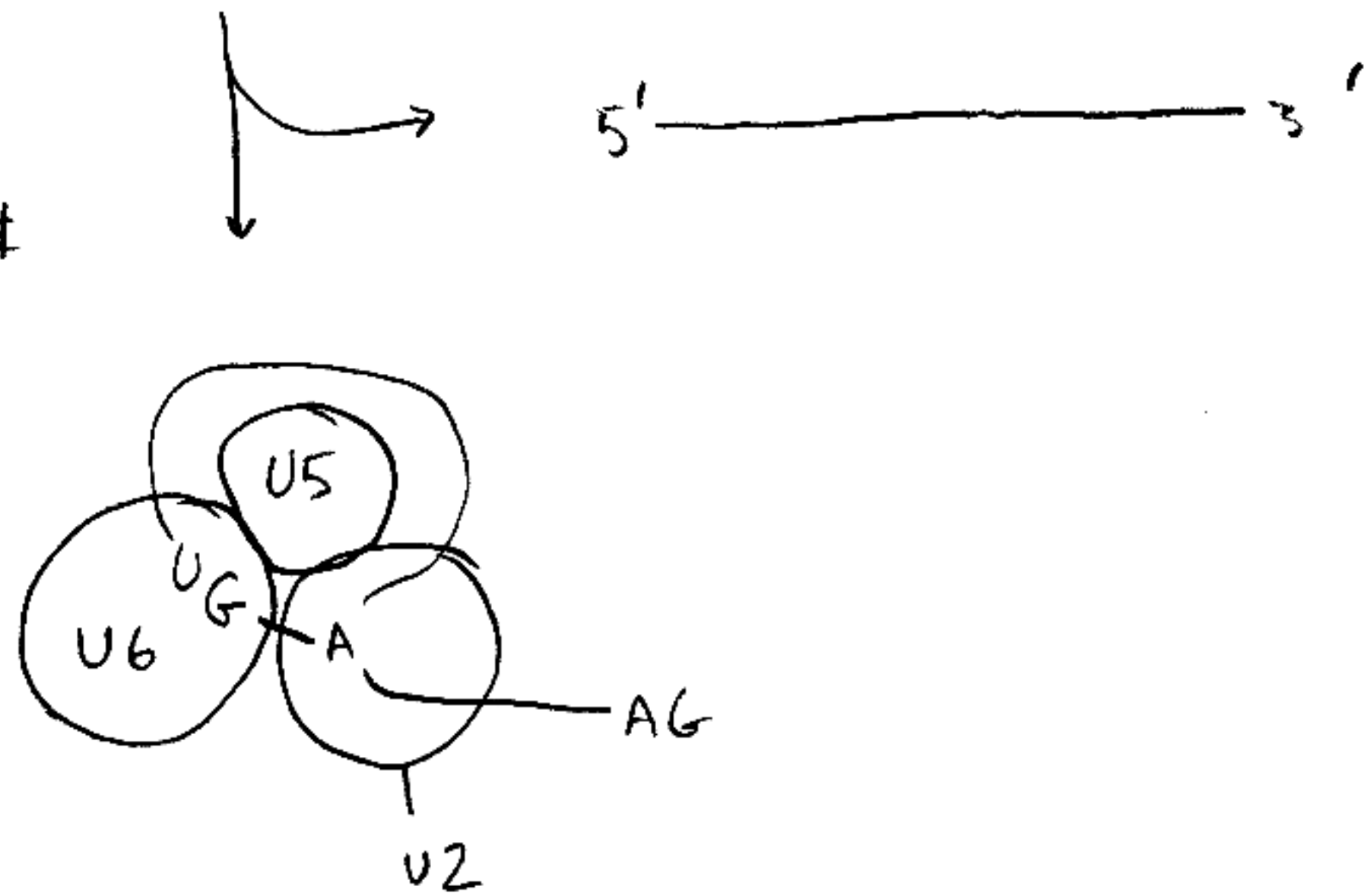
OVERALL PROCESS:



IN THIS INACTIVE SPLICEOSOME, U4 IS PREVENTING U6 FROM PREMATURELY CATALYZING THE SPLICING



- ① FORMATION OF LARIAT
- ② JOINING EXONS & RELEASE OF LARIAT INTRON



THE OTHER SPLICEOSOMAL INTRONS (AU-AC) ARE A LITTLE DIFFERENT.

U1 & U2 → U11, U12
 U4 & U6 → U4atac-U6atac

U5 HOWEVER, IS THE SAME