DIPLOPIA & MULTIPLE SCLEROSIS (Slide CC11-3)
CASE 12: Answers:
1. a. b. c.

2. a. R CN VI--R lateral rectus. L CN III--L medial rectus.

b. L CN VI--L lateral rectus. R CN III--R medial rectus.

c. R CN III--R medial rectus. L CN III--L medial rectus.

3. The medial longitudinal fasciculus (MLF) conveys information between CN nuclei III, IV, and VI and the vestibular nuclei. A number of different "centers" in the cortex, cerebellum and brain stem are involved in activating the final common pathway CN III, IV and VI to control eye movements (see Principles of Neural Science Chapter 43). The important point here is that horizontal eye movements are generated by activating the *ipsilateral abducens nucleus*--CN VI (see Principles of Neural Science p. 722, Fig. 46-8). The abducens nucleus, in turn, sends fibers both to adduct the ipsilateral eye, *and* fibers via the MLF to the contralateral oculomotor nucleus to adduct the contralateral eye. Therefore, for example, a lesion in the abducens nucleus does not simply produce weak abduction of the ipsilateral eye. Instead, it produces a lateral gaze palsy affecting horizontal movement of *both* eyes in the direction of the lesion.

In a lesion of the MLF, the fibers conveying information from the abducens to the oculomotor nucleus are interrupted (see Principles of Neural Science p. 722, Fig. 46-8). Thus, in a lesion of the left MLF, the right eye can abduct but the left eye cannot adduct past the midline, as was seen in our patient. This is called an *internuclear ophthalmoplegia* or INO. Lesions of the MLF producing INO is most commonly due to multiple sclerosis, but can also be seen in brainstem infarcts and tumors. The nystagmus which is seen in the abducting eye is not well understood but may be due to interruption of feedback pathways in the MLF from the oculomotor to the abducens nucleus.

MRI / Clinical Course:

These images vividly demonstrate a bright area in the floor of the fourth ventricle representing an MS plaque involving the L MLF. Her symptoms gradually progressed to the point where in addition to the above INO she also could not abduct the L eye or adduct the R eye (draw this). This is called 1 and 1/2 syndrome i.e., a lateral gaze palsy plus a unilateral INO. How could expansion of her lesion produce these deficits? HINT: she also developed transient mild L facial weakness. Her eye movement abnormalities gradually improved and she was discharged for followup as an outpatient.