accepted that PCP is a confounding factor and must be adjusted for in the analysis if PCP is (a) an independent risk (i.e., prognostic) factor for the outcome and (b) an independent risk factor for (predictor of) future treatment. By “independent” risk factor in (a) and (b) above, we mean a variable that is a predictor conditional upon all other measured variables occurring earlier than the event being predicted. Hence, to check condition (a), we must adjust for $A_0$ and $A_1$; to check condition (b), we must adjust for $A_0$.

Reading from Figure 10, we find that conditions (a) and (b) are both true:

$$0.5 = P[Y = 1 | L_1 = 1, A_0 = 1, A_1 = 1]$$

(3.3) $\neq P[Y = 1 | L_1 = 0, A_0 = 1, A_1 = 1]$

= 0.75

and

$$1 = P[A_1 = 1 | L_1 = 1, A_0 = 1]$$

(3.4) $\neq P[A_1 = 1 | L_1 = 0, A_0 = 1] = 0.5$.

The standard approach to the estimation of the direct effect of AZT controlling for AP in the presence of a confounding factor (PCP) is to compare survival rates among groups with common AP and confounder history (e.g., $L_1 = 1, A_1 = 1$) but who differ in AZT treatment. Reading from Figure 10, we obtain

$$P[Y = 1 | A_0 = 1, L_1 = 1, A_1 = 1] - P[Y = 1 | A_0 = 0, L_1 = 1, A_1 = 1]$$

= $4,000/8,000 - 10,000/16,000$

= $-1/8$.

Hence the analysis adjusted for PCP also suggests an adverse direct effect of AZT on survival controlling for AP.

However, the analysis adjusted for PCP is also problematic, because, as discussed in Section 2 and in Rosenbaum (1984) and Robins (1986, 1987), it is inappropriate to adjust (by stratification) for an extraneous risk factor for the outcome that is itself affected by treatment. Reading from Figure 10, we observe that PCP is affected by previous treatment, that is,

$$0.5 = P[L_1 = 1 | A_0 = 1]$$

(3.6) $\neq P[L_1 = 1 | A_0 = 0] = 1$. 

Fig. 10. Date from a hypothetical study: (+) survivors ($Y = 1$) at $t_2$ (Deaths ($Y = 0$) at $t_2$ in parentheses); (†) $A_k$ measured just after time $t_k$, $k = 0, 1, 2$; (λ) $L_1$ measured at time $t_1$. 

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