INTRODUCTION

How does the pattern of the rudiments of the major blood vessels establish itself in the developing embryo? To understand the cellular biology of these events we must know how the angioblasts, the precursors of endothelial cells, segregate from the mesoderm, migrate, and cohere to one another to form the cords and tubes which are the earliest embryonic blood vessels. We have been using a monoclonal antibody (QH-1) and microsurgery to determine where angioblasts originate and how they assemble into vessel rudiments (Coffin and Poole, 1988; Poole and Coffin, 1989; 1991). The extent and type of directed angioblast migration define three distinct modes of vessel morphogenesis (Poole and Coffin, 1991; Poole, 1993). Vessel rudiments may organize in place, a process termed vasculogenesis, either from angioblasts originating at the rudiment's location (vasculogenesis type I) or from angioblasts which migrate as individual cells or small groups to that site from different locations (vasculogenesis type II). The dorsal aortae form by the first type of vasculogenesis (Coffin and Poole, 1988; DeRuiter et al., 1993; Pardanaud et al., 1987; Poole and Coffin, 1988; 1989; 1991). The endocardium, ventral aortae and posterior cardinal veins form by the second type (Coffin and Poole, 1991; DeRuiter et al., 1993; Drake and Jacobson, 1988; Poole and Coffin, 1991). New vessels may also form by sprouting from preexisting vessels, a process called angiogenesis. The intersomitic and vertebral arteries are the first vessels to form by angiogenesis, sprouting off the rudiments of the dorsal aortae (Coffin and Poole, 1988; Poole and Coffin, 1988; 1989; 1991). Figure 1 illustrates the different roles of endothelial cells in vasculogenesis and angiogenesis.
The construction of quail/chick chimeras in the 1980s has begun to delineate the details of endothelial cell lineage and its close relationship to the hematopoietic lineages. These studies have been reviewed in detail elsewhere (Noden, 1989; Pardanaud et al., 1989; Poole and Coffin, 1991; Poole, 1993). An interesting aspect which emerges from recent work is the variation in angioblast differentiation from quail mesoderm grafted to different sites in chick embryos. Blocks of tissues the size of a single somite grafted beneath the otic placode resulted in angioblast differentiation from all intraembryonic mesoderm except the prechordal plate and notochord (Noden, 1988; 1989). Similar blocks of tissues grafted to older chick embryos in the limb bud or coelom demonstrated a striking difference between splanchnopleural mesoderm (the portion of mesoderm adjacent to embryonic endoderm) and somatopleural mesoderm (the mesoderm adjacent to ectoderm). Splanchnopleural mesoderm gives rise to abundant angioblasts in these grafting experiments; whereas, somatopleural mesoderm produces no angioblasts or only a few (Pardanaud and Dieterlen-Lievre, 1993). We have transplanted a single quail somite or a piece of lateral mesoderm the size of a somite into the head or trunk of chick hosts at the same stage of development (10 somite stage) and found that a somite graft to the head produces many angioblasts, a somite graft to the trunk produces very few, and lateral mesoderm grafts (containing both splanchnopleural and somatopleural mesoderm) produce many angioblasts in both locations (Poole, 1991 and in preparation). Figure 2 shows an example of a somite graft result in each location. These differences between somite and lateral mesoderm are also seen in vitro. Quail mesodermal cells, dissociated with trypsin, cultured for 20 hours result in 5% QH-1 labelled cells of somite origin and 25% QH-1 labelled cells from the lateral mesoderm. The addition of basic fibroblast growth factor (bFGF) to these cultures at 25 ng/ml produced a ten-fold increase in angioblasts from somite mesoderm (50% QH-1 positive), but did not significantly affect lateral mesoderm cultures (25% QH-1 positive).