tion alternate with horizons containing an autochthonous and monospecific sporophyte vegetation (*11*). The sporophytes—the sporophytic nature is demonstrated by sporangia—show naked, roundish, and rare dichotomously branched axes which are more than 25 cm long. Many botanists, such as Bierhorst (*12*), Bold, Alexopoulos, and Delevoryas (*13*), and Taylor (*14*), regard sporophytes like *R. major*, *R. gwynne-vaughani*, *H. lignieri*, and *N. aphylla* as vascular plants and place them at the base of vascular plant evolution. Other botanists, such as Hébant (*15*) and Watson (*16*), mention bryophytic characteristics of *R. gwynne-vaughani* and *H. lignieri*.

The hydroids of *R. major* or *N. aphylla*, the creeping rhizoid-bearing "rhizomes" of *R. major*, *R. gwynne-vaughani*, and *H. lignieri*, or the columella in *H. lignieri* sporangia might be bryophyte-like characteristics. Our bryophyte-like gametophytes from the Rhynie Chert seem to revalorize these features. Moreover, the axes of, for instance, *R. major*, the most simply constructed sporophyte of the Rhynie Chert, show generally the same organization as the gametangiophore stalks of our Rhynie Chert gametophytes. Conducting tissue (hydroids and phloem-like cells) is surrounded by a simply constructed parenchymatous cortex, which is enclosed by an epidermis with stomata (without accessory cells) and a cuticle. Satterthwait and Schopf (*17*) described a phloem-like tissue in the axes of *R. major* with pores in the lateral cell walls. The gametangiophore head of every specimen of the new gametophyte contained similar tissue above the hydrom. *Lyonophyton rhyniensis* has stomata without accessory cells and double-layered antheridial walls. The stomata are comparable with those of *R. major* or *R. gwynne-vaughani*, and the double-layered antheridial walls might be compared with the many-layered sporangial walls of Rhynie Chert sporophytes (*12*).

On the basis of the phylogeny of single characteristics, some sporophytes, foremost *R. major*, and the gametophytes of the Rhynie Chert seem to exhibit a similar level of development. We might even suspect some taxonomical relationships, but there is no concrete evidence for such.

The bryophytic characteristics of the Rhynie Chert sporophytes do not have to be based on direct taxonomical relationships to genuine bryophytes. These characteristics, and the sporophyte-like characteristics of our gametophytes, might be remnants of a plant group which preceded both bryophytes and pteridophytes (Fig. 4). The mere fact of its geologic age puts the Rhynie flora close to the evolutionary roots of land plants. Our investigations suggest that consideration of sporophytes and their evolution only may lead to misinterpretations of phylogenetic relationships. The investigation of Devonian gametophytes puts our understanding of the phylogenetical development of the land plants into a new perspective.

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References and Notes

1. W. Remy and R. Remy, *Science* **208**, 295 (1980).
2. ———, *Argumenta Palaeobot.* **6**, 37 (1980).
3. For a detailed description and illustration of the new gametophyte, see W. Remy, *ibid.*, in press.
4. W. Remy, R. Remy, H. Hass, S. Schultka, F. Franzmeyer, *ibid.* **6**, 73 (1980).
5. R. Kidston and W. H. Lang, *Trans. R. Soc. Edinburgh* **52**, 603 (1920).
6. ———, *ibid.* **51**, 761 (1917).
7. ———, *ibid.* **52**, 643 (1920).
8. A. G. Lyon, *Nature (London)* **203**, 1082 (1964).
9. W. El-Saadawy and W. S. Lacey, *Rev. Palaeobot. Palynol.* **27**, 119 (1979).
10. O. A. Høeg, in *Traité de Paléobotanique*, E. Boureau, Ed. (Masson, Paris, 1967).
11. W. Remy, S. Schultka, H. Hass, F. Franzmeyer, *Argumenta Palaeobot.* **6**, 95 (1980).
12. D. W. Bierhorst, *Morphology of Vascular Plants* (Macmillan, New York, 1971), pp. 71–78.
13. G. C. Bold, C. J. Alexopoulos, T. Delevoryas, *Morphology of Plants and Fungi* (Harper & Row, New York, ed. 4, 1980), pp. 432–436.
14. T. N. Taylor, *Paleobotany, An Introduction to Fossil Plant Biology* (McGraw Hill, New York, 1981), pp. 96–104.
15. C. Hébant, *The Conducting Tissues of Bryophytes* (Cramer, Vaduz, Switzerland, 1977), p. 114, plate 80.
16. E. V. Watson, *The Structure and Life of Bryophytes* (Hutchinson, London, ed. 3, 1978), pp. 176–180.
17. D. F. Satterthwait and J. W. Schopf, *Am. J. Bot.* **59**, 373 (1972).
18. I thank W. G. Chaloner, London, who carefully read the manuscript and improved it, R. Blaschke, Münster, for technical help, H. Hass, Münster, for the careful microscopic analysis of this difficult material, for the microphotographs, and for discussions, and the Gesellschaft zur Förderung der Westfälischen Wilhelms-Universität for the use of instruments. Supported by Deutsche Forschungsgemeinschaft grants Re 200/10 and 200/11-4.

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Control of Vascular Contractility by the Cardiac Pacemaker

Abstract. *Rhythmic contractile activity, synchronized with pulsatile pressure changes, was recorded from rabbit aorta in vivo. The contractions were locked in frequency to the pulsatile activity of the heart even when the heart was electrically paced to rates as high as 600 cycles per minute; termination of cardiac contractility resulted in their elimination. When the atria and ventricles contracted at different rates, the pulse-synchronized contractions were locked to the atrial rate. Excision of the right atrium, but not the left, resulted in the abolition of pulse-synchronized contractions. It is concluded that a common pacemaker controls cardiac and vascular contractility, coordinating events in the two tissues.*

Although contractions can be elicited from the smooth muscle component of the large arteries by various stimuli (for instance, neural transmitters, hormones, and membrane depolarization) (*1*), the fundamental question of whether the vessels undergo rhythmic activation during the cardiac cycle was, until recently, unresolved. Classically, the large arteries were described as passive elastic tubes whose viscoelastic elements were periodically distended by pulsatile pressure changes (*2*). However, several studies indicated that the vessels may produce rhythmic tension changes during the cardiac cycle. Hysteresis in aortic pressure-circumference (*3*) or pressure-diameter (*4*) relations suggests that the aorta imparts energy to the blood. A possible explanation for this phenomenon is that active contraction of the smooth muscle wall occurs during each systole. Heyman and co-workers (*5*) measured the phase relation between the pulse wave as recorded simultaneously extra- and intra-arterially. A neurally dependent leading by the extra-arterial event was observed. This phase shift was attributed to rhythmic activation of the smooth muscle component of the arterial wall.

Recently, we bypassed the blood flow through sections of large arteries in rabbits in vivo. When mechanical activity was measured under these conditions, we found that the aorta and the carotid, femoral, and coronary arteries show rhythmic contractions which oscillate at the same frequency as the pulsatile activity of the heart (*6–8*). The observed phase-locking between the tension and pulse pressure changes, with an increase in tension being temporally correlated with the upstroke of the corresponding pulse wave (*8*), suggests the term pulse-synchronized contractions (PSC's). The PSC's were shown to be produced by neurogenic activation of the smooth muscle component of the arteries, with the neural signal responsible for activation conducted caudally, away from the heart (*6*). The PSC's remained frequency-locked to the heart rate, even after drug-induced perturbation from the rest-

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ing rate (*6*). In this report evidence is presented which indicates that the pacemaker region for PSC generation is in the right atrium. It is suggested that a common pacemaker may control myocardial and vascular contractility, affording better coordination between the two tissues.

Adult rabbits were anesthetized by an intravenous injection of sodium pentobarbital (35 mg/kg) through the ear vein. Tracheotomies were performed and the animals were then placed on a respirator pump. A longitudinal cut was made along the abdominal midline of the rabbits and a 5-cm segment of the abdominal aorta exposed. Mechanical activity was recorded from the aorta in vivo by two procedures. (i) The blood flow was temporarily occluded from the exposed aortic segment with standard arterial clamps. Small slits were then made in the occluded segment and a polyethylene tube (bypass tube) inserted and secured with ligatures (Fig. 1). In the center of the bypass tube a catheter was mounted and connected to a blood pressure transducer (Statham P23 Db). The clamps were then removed and the blood flowed freely through the bypass tube. In the center of the exposed aortic segment (bypassed segment) a small hook was inserted. The hook was attached to a tension transducer (Grass FTO 3C), which was mounted on a base attached to the surgical table. (ii) Alternatively, after the blood flow was occluded from the exposed aortic segment, a small slit was made in the caudal side of the occluded segment. A balloon-tipped catheter was then inserted through the slit and the open end of the catheter attached to a blood pressure transducer (Statham P23 Db) (Fig. 1). Muscular contraction of the vessel compressed the balloon, producing an increase in the signal. A catheter to record pulse pressure was inserted cranial to the upper border of the occluded segment. Blood was washed from all the exposed segments with physiological saline, and saline was applied to the exposed portion of the abdominal aorta to maintain viability.

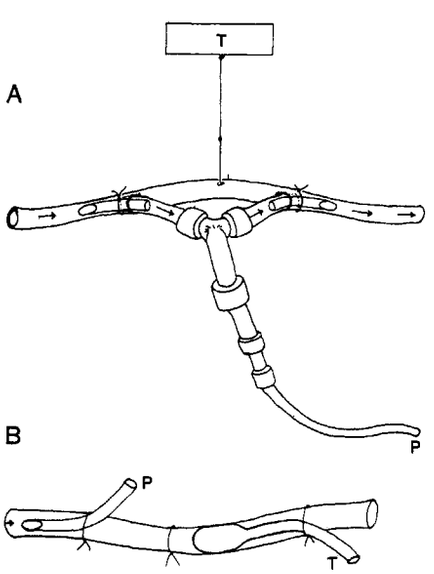

Fig. 1. Techniques used for measurement of mechanical activity in vivo. (A) The blood flow through a muscle segment was bypassed and contractile activity recorded with the tension transducer system. (B) The blood flow to a muscle segment was occluded and wall tension recorded with the balloon-tipped catheter system. *P*, recording of pulse pressure changes; *T*, recording of changes in tension. Arrows indicate the direction of blood flow.

Simultaneous recordings of changes in aortic pulse pressure and muscle wall tension, monitored with the balloon catheter system, are shown in Fig. 2A. An increase in wall tension is associated with the upstroke of the pulse wave. As reported previously (*8*), the balloon catheter and tension transducer systems give signals of opposite polarity. A possible explanation for the inversion of one signal is that the tension transducer records longitudinally directed movements in addition to circularly directed contractions. However, the balloon catheter system is less sensitive to longitudinal movements, and its recordings would therefore represent the correct signal polarity.

When animals were bled by lancing the thoracic aorta, PSC's continued for up to 10 minutes in the absence of either pulsatile flow or a pressure gradient in the aorta (N = 15). During this time cardiac contractility persisted. Cessation of cardiac activity always resulted in PSC termination. When PSC's were recorded from bled animals with either the balloon catheter or the tension transducer system, positive-going wave forms were observed.

The dependence of PSC activity on cardiac contractility, but not on pulsatile flow, suggests that the pacemaker region for PSC's may lie in the heart. If the heart was electrically driven by pulsatile stimulation of the right atrium, abdominal aortic PSC's remained locked to the pulsatile activity of the heart. The PSC's remained locked in frequency to cardiac activity even at stimulation rates as high as 600 cycles per minute. In some preparations ventricular conduction block developed during atrial pacing, and consequently ventricular muscle contractions occurred at a lower frequency than atrial ones. In these cases, aortic PSC's were locked to the atrial, not ventricular, frequency (Fig. 2B).

After excision of the left atrium, from either bled or intact animals, PSC activity persisted; however, if the right atrium was removed, PSC's stopped (N = 6) (Fig. 2C). When a significant reduction in ventricular activity occurred after excision of the atrium, the ventricles were electrically stimulated to return activity to the control level. This procedure did not cause the resumption of PSC's, ruling out a change in ventricular activity as a possible explanation for PSC termination.

In conclusion, evidence has been presented which indicates that the locus for

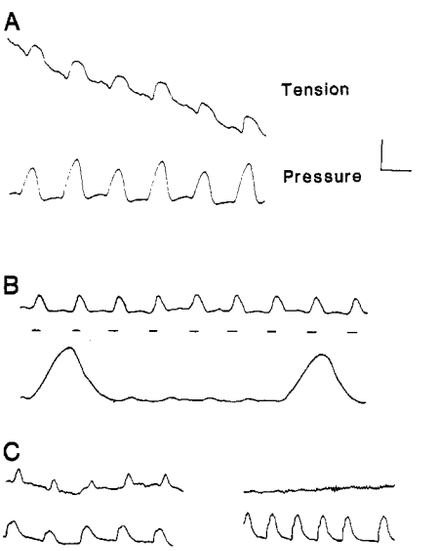

Fig. 2. (A) Simultaneous recordings of wall tension and pulse pressure changes from the aorta in vivo. Tension was monitored with the balloon catheter system. Vertical calibration bar: 3 mmHg (balloon catheter system), 25 mmHg (pulse pressure); horizontal bar: 250 msec. Diastolic blood pressure was 80 mmHg. Initial basal pressure in the balloon was 85 mmHg. (B) Recording of aortic PSC's (upper trace) and ventricular muscle contractions (lower trace) during right atrial pacing. PSC's were recorded with the tension transducer system from bled animals. In this preparation ventricular conduction block occurred. The dashed line beneath the upper record represents the rate of atrial stimulation. In this and other preparations tested there was a one-to-one correspondence between atrial stimulation rate and atrial contractions (not shown). Vertical bar: 0.35 g (upper trace), 3 g (lower trace); horizontal bar: 250 msec. Ventricular and atrial muscle contractility was monitored with the tension transducer system as described in procedure (i) for measuring PSC activity. (C) The record at the left represents aortic PSC activity (upper trace) and ventricular muscle contractions (lower trace) following excision of the left atrium. The record at the right represents the corresponding activity after excision of the right atrium. Recordings were made with the tension transducer system from a bled animal. Ventricles were electrically stimulated after removal of the right atrium to ensure that ventricular contractile activity was at least at the control level. Vertical bar: 0.4 g (upper trace), 3 g (lower trace); horizontal bar: 250 msec.

PSC generation is in the heart. (i) Elimination of myocardial contractility causes elimination of PSC's. (ii) In bled animals PSC's continue, ruling out the possibility that distension of the aorta triggers the tension changes. (iii) When the atrium and ventricles contract at different rates, PSC's are locked in frequency to the right atrial frequency. (iv) Excision of the right atrium, but not the left, causes elimination of aortic PSC's. Observations (iii) and (iv) suggest that the pacemaker region is in the right atrium, the site of the sinoatrial node.

A possible sequence of events can be summarized as follows. As the hydrostatic pressure in the vessel increases, nervous impulses originating from the heart initiate a contraction of the arterial smooth muscle. Since the pressure increase is very large, the muscle becomes overloaded and expands during the contraction phase of the PSC. The absence of active shortening in the intact vessel during the cardiac cycle probably explains the PSC having gone unnoticed for so long.

Because of the above lack of muscle shortening during activation, there is no incompatibility between the slow rate of smooth muscle shortening and the relatively rapid active tension oscillations. Under the conditions described here, with the muscle allowed to shorten in the absence of a hydrostatic pressure load, only a few percent of the maximal force development occurs (*9*), probably because the brief period of activation did not allow for substantial shortening. However, under conditions of increasing length the same degree of activation may represent a considerable resistance to stretch. This would prevent excessive dilatation of the arterial wall, which would otherwise, according to Laplace's law, amplify the wall stress caused by the pulse pressure wave. Possible initiation of both cardiac and arterial activity from a common pacemaker in the right atrium may ensure a high degree of coordination between these two events.

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References and Notes

1. R. F. Furchgott and S. J. Bhadrakom, *J. Pharmacol. Exp. Ther.* **108**, 129 (1953); R. F. Furchgott, S. J. Elrreich, E. Greenblatt, *J. Gen. Physiol.* **44**, 499 (1961); R. Deth and C. van Breemen, *J. Membr. Biol.* **30**, 363 (1977); C. van Breemen, P. Aaronson, R. Loutzenhiser, *Pharm. Rev.* **30**, 167 (1979).
2. O. Frank, Z. *Biol. (Munich)* **37**, 483 (1899); L. H. Peterson, R. E. Jensen, J. Purnell, *Circ. Res.* **8**, 622 (1969).
3. R. F. Rushmer, *Am. J. Physiol.* **183**, 545 (1955).
4. H. P. Pieper and L. T. Paul, *ibid.* **217**, 154 (1969); G. R. Nicolos and H. P. Pieper, *ibid.* **221**, 1209 (1971); *ibid.* **228**, 518 (1975).
5. F. Heyman, *Acta Med. Scand.* **157**, 503 (1957); *ibid.* **163**, 473 (1959); _____ and K. Stenberg, *ibid.* **171**, 9 (1962); F. Heyman, *Nature (London)* **216**, 402 (1967).
6. A. Mangel, M. Fahim, C. van Breemen, *Nature (London)* **289**, 692 (1981).
7. _____, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **40**, 409 (1981).
8. _____, R. Loutzenhiser, in *Eighth International Symposium on Vascular Neuroeffector Mechanisms*, J. A. Bevan, Ed. (Raven, New York, in press).
9. B. Gow, in *Handbook of Physiology*, vol. 2, *Vascular Smooth Muscle*, D. F. Bohr, A. P. Somlyo, H. V. Sparks, Eds. (American Physiological Society, Washington, D.C., 1980), p. 353.
10. Support from University of Miami biomedical grant HR 7198 and American Heart Association/Florida Affiliate grants 7/81 AG 610 and RF 2/81 is acknowledged.

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Cyanobacterial Blooms: Carbon and Nitrogen Limitation Have Opposite Effects on the Buoyancy of *Oscillatoria*

Abstract. *Gas vacuolation in* Oscillatoria rubescens *decreased with increased nitrogen limitation and increased with transitions from nitrogen to inorganic carbon limitation. Gas vacuoles consist of protein vesicles that can accumulate in carbon-limited but not in unenriched nitrogen-limited cells. Nitrogen limitation is a factor in the formation of deep population maxima; carbon limitation can promote surface blooms.*

Blue-green algae (Cyanobacteria) can form dense surface and deepwater population maxima in lakes because their cells possess gas vacuoles. Gas vacuoles are aggregates of vesicles with gas-permeable, proteinaceous walls. In a stable water column the relative cellular volume occupied by these structures can determine whether an alga will rise to the surface or accumulate at some depth. Laboratory and field studies indicate that relative gas vacuolation (RGV) and buoyancy in blue-green algae depend on an interaction of light and limiting nutrients that affects relative rates of photosynthesis, growth, and gas-vesicle synthesis (*1*). Of particular interest are growth-limiting conditions that permit RGV to increase, as it does before or during surface blooms. Smith and Peat (*2*) reported that gas vacuolation increased in the later phases of growth by batch cultures of *Anabaena flos-aquae*. Reynolds (*3*) described similar increases in bloom-forming populations of *Anabaena* and *Microcystis* as their growth rates decreased. In these studies, one or more factors limited growth but permitted RGV to increase. Light can do this, at least in nutrient-sufficient algae. With a reduction in light intensity, RGV increased in batch cultures of *A. flos-aquae* (*4, 5*), but not in *Oscillatoria rubescens* taken from a N-limiting chemostat (*6*). As alternatives to light, Reynolds and Walsby (*7*) suggested the major growth-limiting nutrients: C, N, and P. We report that RGV in *O. rubescens* decreased with increases in the degree of N limitation and increased with transitions to inorganic carbon (C_i) limitation.

We worked with cyclostats designed specifically for use with *O. rubescens* (*8, 9*), and we measured RGV nephelometrically with a modified version of Walsby's technique (*10*). After a reduction in the dilution rate of a N-limiting cyclostat, RGV in *O. rubescens* declined along with cell N content, and then stabilized as N content continued to decline (Fig. 1A). A companion culture was supplied by the same peristaltic pump but deprived of CO_2 by a trap installed in the air line after the reduction in dilution rate (Fig. 1B). With the removal of CO_2, RGV remained stable at first and then increased as the culture became increasingly limited by C_i. (Presumably, the smaller decrease in the cell N of this culture reflected the loss of some nitrogenous compounds such as RNA that vary with growth rate.)

The effect of CO_2 deprivation was also observed in two initially N-limiting cyclostats with constant dilution rates (Fig. 2). On day 3, we installed a CO_2 trap in the air line of culture A (Fig. 2), and by day 4, RGV had increased from its previous steady-state level. On day 6, we transferred the CO_2 trap to culture B (Fig. 2), and as RGV decreased in culture A, it increased in B. Thus, the relative gas vacuole content of the cells of *O. rubescens* decreased with increased N limitation and increased with transitions to C_i limitation. The negative effect of N limitation on RGV in the axenic cultures of *O. rubescens* used in this study confirmed observations reported earlier by A.R.K. on xenic cultures of the same alga grown in a chemostat (*6*). Moreover, increases in the availability of C_i and N can also have opposite effects on blue-green algal buoyancy. Increases in ammonium-ni-

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