Susceptibility of the Ovariectomized Hen to Cholesterol-Induced Coronary Atherogenesis

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A marked sex-difference in susceptibility to cholesterol-induced coronary atherosclerosis exists in mature, gonadally active birds—hens being protected against coronary involvement. This report concerns whether this protection is lost when hens are castrated.

Previous studies from this department demonstrated that exogenous estrogens protected cockerels against cholesterol-induced coronary atherogenesis.1-3 Further, mature, egg-producing hens, intact or oviduct-ligated, were also markedly resistant to the development of diet-induced coronary lesions.4 It was postulated that this protection was a result of the endogenous, physiologic estrogen secretion of the hen’s ovary.

The present experiment was undertaken to test this concept, by determining the effect of castration on coronary atherogenesis in cholesterol-fed hens.

Methods

The methods were in accordance with the long-established procedures of the department’s atherosclerosis research group.1-5 The main problem in this experiment was the achievement of complete ovariectomy. As is known, the hen has a single ovary on the left side, with a rudimentary contralateral organ. The latter proliferates after removal of the left ovary, and an estrogen-secreting ovotestis develops.6 After several unsuccessful attempts with various techniques, this problem was circumvented and complete suppression of estrogen secretion achieved by surgical removal of the left ovary at 5 weeks of age, followed by continuous administration of testosterone propionate*: 1.0 mg./bird/day parenterally until onset of the experiment at 33 weeks of age.

Results and Discussion

Consistent with previous observations,4 coronary lesions were minimal in mature, estrogen-secreting, egg-producing hens (intact or oviduct-ligated), despite diet-induced hypercholesterolemia and aorta atherogenesis (table 1). These hens also exhibited the usual estrogen-induced enhancement of hyperphospholipemia, with consequent lowering of total cholesterol: phospholipids (C/P) ratios toward normal levels.

In contrast, the ovariectomized hens had elevated C/P ratios and severe coronary lesions (table 1).*

Thus, castration effectively negated the resistance of hens to diet-induced coronary atherogenesis. These findings support the conclusion that endogenous, physiologic estrogen secretion is responsible for the resistance of sexually mature hens to development of coronary atherosclerosis.

The parallelism between man and chick in this regard is striking.2-3 Thus, pathologic studies demonstrate that ovariectomy in young women—as in hens—markedly sup-

* Completeness of ovariectomy was confirmed in 6 of 7 birds by masculinization of combs and tail feathers, by lack of estrogenic effect on plasma phospholipids, and by absence of ovarian-ovotesticular tissue at postmortem. One bird was exceptional in all of the foregoing, and also had no coronary lesions. The finding in these birds was a result of castration per se unrelated to the pre-experiment administration of androgen, since previous groups with incomplete ovariectomy + testosterone retained resistance to coronary lesions. Further, earlier studies failed to demonstrate any significant effect of testosterone.5-7

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presses their usual resistance to coronary atherogenesis.12

These observations constitute additional evidence that the remarkable immunity of premenopausal women to coronary atherosclerosis and coronary heart disease is in a major way a resultant of ovarian estrogenic secretion.1°

SUMMARY

In association with loss of endogenous estrogen secretion, ovariectomized hens lose their resistance to cholesterol-induced coronary atherogenesis.

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REFERENCES


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