Woodhead (1930) noted that in light infections of *B. elegans* in *Eurynia iris* the gonad continued to produce eggs over 75% of its area, but in heavy infections the gonad was entirely replaced. He also considered that *E. iris* could maintain the infection for at least two winters.

Roughley (1933) found either no sexual products or only a few degenerating eggs in five *O. commercialis* and one *O. angasi* infected by a bucephalid which he did not describe.

Ozaki and Ishibashi (1934) described the cercaria *B. margaritae* liberated from *Pinctada martensi,* the Japanese pearl oyster. They stated of the sporocyst (p. 440) that " . . . this parasite is a terrible parasite of the pearl oyster and was having serious effects on the formation of the pearl." The parasite first affected gonad and liver, then spread to mantle, gills, palps and adductor muscle.

Andreu (1949) reported *Bucephalopsis haimeana* in the ovary of *Tapes aureus* and noted the destruction of the gonads. He stated the parasite occurred in May, but not in March or June.

Kniskern (1952) found the gonad of *Lampsilis siliquoidea* completely invaded and destroyed by the sporocysts of *Rhipidocotyle septpapillata* in " old and heavy infections ", but he stated (p. 322) that " In light or moderately heavy infection areas of normal gonadial tissue remained to actively produce ova and sperm."

Menzel and Hopkins (1955) gave details of the growth of one specimen of *Crassostrea virginica* (under natural conditions) parasitised by *B. cuculus.* They found that the oyster increased in weight from 59.0 to 227.0 grams over the 16 month experimental period, but failed to state whether the oyster became infected before or after the experiment commenced. The oyster was less than two years old when it became infected. The authors stated (p. 341) " The effects of *Bucephalus cuculus* infection are not so well known. From personal observations on many bucephalus-infected oysters we know that the sporocysts are confined to the gonad in the early stages of the infection. The gonad tissues are eventually destroyed so that oysters with well-developed infections never produce eggs or sperm." They point out that, initially, bucephalid infections may cause increased growth, but after completely replacing the gonad tissues, the sporocysts spread to mantle, gills, digestive glands and the adductor muscle and impair normal functions.

Millar (1963) carried out mortality experiments on a sample of *O. lutaria* from New Zealand. He found that of the oysters that died, 67.3% were infected with sporocysts of *B. longicornutus,* and only 15% of those that remained alive were infected.

Cheng and Burton (1965) found that the initial site of infection of young sporocysts of " *Bucephalus* sp." in *Crassostrea virginica* from Rhode Island (USA) was the pyloric caeca (digestive gland) rather than the gonads. This contrasted with the condition reported in *C. virginica* parasitised by *B. cuculus* further south along the Atlantic coast, and was the authors' reason for maintaining that the species they were dealing with was distinct from *B. cuculus.* (It should be noted that use of the generic name *Bucephalus* for these species may not be correct, *vide* Howell, 1966.) They also found that there is no increase in the number of amoebocytes in parasitised gonads or pyloric caeca.

## DESCRIPTION OF THE EFFECTS OF *Bucephalus longicornutus*

Oysters from Foveaux Strait were obtained between June and October 1963, and March and July 1964. Wherever possible infected oysters were compared with uninfected oysters of the same size and from the same locality.